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NEW YORK STATE JOURNAL OF MEDICINE

JANUARY 1990 • Volume 90, Number 1

MEDICAL SOCIETY OF THE STATE OF NEW YORK
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Contents

COMMENTARIES

The *New York State Journal of Medicine* enters its 90th year 1
PASCAL JAMES IMPERATO, MD

Topical low-dose thrombolytic therapy 2
LEWIS J. RUBIN, MD

RESEARCH PAPERS

Evaluation of methods for identification of *Campylobacter pyloris* infection 4
PETER J. SANTOGADE, MD; VICTOR D. BOKKENHEUSER, MD; MOHAMMED A. FAISAL, MD; DONALD P. KOTLER, MD; JOHN V. SCHOLES, MD; PETER R. HOLT, MD

Cervical angina 8
BERNARD JACOBS, MD

REVIEW ARTICLE

A critical appraisal of contact lens disinfection 12
MICHAEL B. STARR, MD

SPECIAL ARTICLES

Melville's "Bartleby the Scrivener": A case study 17
FRED A. WHITEHEAD, PhD; BRUCE S. LIESE, PhD; MICHAEL L. O'DELL, MD

History of medicine archives in New York State 23
KATHLEEN S. ANDERSEN, DANIEL M. FOX

CASE REPORTS

Local low-dose infusion of streptokinase for massive pulmonary embolism when systemic thrombolysis is contraindicated 32
MICHAEL A. NAKAO, MD

The use of a Swan-Ganz catheter and streptokinase in the management of massive hemoptysis 33
ROBERT D. BRANDSTETTER, MD

Trabecular (neuroendocrine) carcinoma of the skin: Report of four cases and review of the literature 35
ROBERT X. MURPHY, JR, MD; JOHN K. LI, MD; FLORA K. MINCER, MD; BERISH STRAUCH, MD

LETTERS TO THE EDITOR

Low-dose subcutaneous heparin therapy 39
J. GEORGE SHARNOFF, MD

Cocaine 39
LARISA KAUKONEN, MD; ROBERT S. HOFFMAN, MD

The new health code regulations concerning postgraduate medical education in New York State 39
PETER SLOCUM

Alcohol and public health: Implications for New York State 41
MARGUERITE T. SAUNDERS

LEADS FROM EPIDEMIOLOGY NOTES 42
NEWS BRIEFS 46
BOOKS RECEIVED 48
GUIDELINES FOR AUTHORS 50
MEDICAL MEETINGS AND LECTURES 6A

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MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

FEBRUARY 1990

AROUND THE STATE

MANHATTAN

Feb 1. **Calcium and the Cardiovascular System.** 2 Cat I Credits. Helmsley Hotel. Contact: New York Cardiological Society, Inc, 681 Lexington Ave, Suite 310, New York, NY 10022. Tel: (212) 888-5595.

Feb 10. **Cornea and Astigmatism Course.** Manhattan Eye, Ear & Throat Hospital. Contact: Francine Leinhardt, Course Coordinator, Manhattan Eye, Ear & Throat Hospital, 210 E 64th St, New York, NY. Tel: (212) 838-9200, ext 2776.

Feb 26–28. **Temporal Control of Drug Delivery.** Sheraton Centre. Contact: Conference Director, The New York Academy of Sciences, 2 E 63rd St, New York, NY 10021. Tel: (212) 838-0230.

AROUND THE NATION

ARIZONA

Feb 19–21. **Third Annual Congress of the North American Society of Phlebology.** The Pointe at Squaw Peak Resort, Phoenix. Contact: Helane Fronek, MD, Division of Thoracic and Vascular Surgery—SW 208, 10666 N Torrey Pines Rd, La Jolla, CA 92037.

CALIFORNIA

Feb 1–3. **Duplex Imaging.** 16 Cat I Credits. Le Meridian Hotel, Coronado. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

Feb 3–4. **The Third Bay Area Vascular**

(*continued on p 10A*)

NEW YORK STATE
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January 1990 • Volume 90, Number 1

COMMENTARIES

The *New York State Journal of Medicine* enters its 90th year

With this issue, the *Journal* enters its 90th year of publication. As such it ranks among the oldest continuously published medical journals in the country. During its nine decades of existence, the *Journal* has been guided by 12 editors and several publications committees and has undergone numerous changes in both design and format.[1]

While design and format have changed, the commitment to excellence has been constant since the first *Journal* issue appeared in January 1901. This commitment has earned the *Journal* a current place among the 30 leading medical journals in the world.[2] The significance of this ranking is underscored by the fact that some 5,000 medical journals are published in the world today, of which 2,400 are listed in *Index Medicus*. Among the latter, the leading 112 are indexed by *Index Medicus* in its rapid abridged listing. The *Journal* is among this select 112. The *Journal's* standing with readers remains very high. A readership survey conducted for the *Journal* in 1987 by Health Research Industries demonstrated high levels of reader satisfaction.[3]

The *Journal*, which has been published by the Medical Society of the State of New York (MSSNY) since 1906, actually began publication five years earlier, in January 1901, under the auspices of the New York State Medical Association (NYSMA). The latter organization was founded by disaffected MSSNY members in 1884. The impetus for the schism was the adoption in 1882 by the MSSNY of a code of ethics which was at variance with that of the American Medical Association (AMA). The MSSNY's code stated that "members of the Medical Society of the State of New York, and of the medial societies in affiliation herewith; may meet in consultation legally qualified practitioners of medicine."[4] This position was a very liberal one compared to that of the AMA, which stated that ". . . no one can be considered as a regular or fit associate in consultation, whose practice is based on exclusive dogma . . ."[4] The NYSMA drew to itself a large following, organized itself into district branches at the local level, and affiliated itself with the AMA. Kelly has written an insightful summary of the formation of the NYSMA and of the slightly more than two-decade period in which it simultaneously existed with MSSNY.[4]

During the first year of its founding, NYSMA published an annual volume known as the *Transactions of the New York State Medical Association.* This was patterned on the *Transactions of the Medical Society of the State of New York* which was first published in 1807. The council of the NYSMA voted on October 18, 1900, to suspend publication of the *Transactions* and to instead publish a monthly journal to be called the *New York State Journal of Medicine.*[1] The first issue appeared in January 1901 under the editorship of a publications committee headed by James Hawley Burtenshaw of New York City. An unsigned editorial in this first issue set out the aims of the *Journal*, which included the presentation of ". . . scientific papers . . . news items, letters of correspondence, notices of removals, marriages and deaths, and an occasional short sermon, perhaps on the needs of the Association or of the profession at large throughout the State." The editorial went on to say, "The *Journal* will not lend itself to unnecessary controversy."[5] Redway made a cogent observation when in 1951 he wrote that ". . . it may be remarked that the *Journal* today follows the objectives of fifty years ago . . ."[1] These objectives have of course been modified over time. Yet the commitment to excellence has always remained.

In 1905 the MSSNY and the NYSMA voted to join together and were then unified under the name "Medical Society of the State of New York." The MSSNY discontinued publication of its *Transactions* with the 1905 issue and adopted the *New York State Journal of Medicine* as its official publication. The first issue of the *Journal* published under MSSNY auspices appeared in January 1906. The aims of the *Journal* were presented in detail in an editorial:

> The *New York State Journal of Medicine* will continue as the official organ of the Society. Every endeavor will be made to make it attractive, interesting and worthy of the profession . . . It will publish news items . . . book reviews, and valuable original articles. Its aim will be to promote the best interests of the profession, to advance the science of medicine, to elevate the standard of medical advertising and to assist in every way in upholding the honor and dignity of the Medical Society of the State of New York.[6]

These objectives, while broader than those put forth in 1901, were essentially similar and also strove to achieve excellence.

Prior to 1906, the *Journal* was edited by a publication committee. However, in early 1906, James Peter Warbasse, MD, a prominent Brooklyn surgeon and distinguished medical writer, was appointed editor. He held this position until December 8, 1908, when he resigned. Warbasse moved the *Journal* forward in its pursuit of excellence. In a lead editorial published in March 1906 he wrote:

> With this number, the *New York State Journal of Medicine* enters the field of medical journalism under new conditions and with new aims . . . This journal will be devoted to what we regard as the best interests of medicine. It will present original articles which are the products of original investigation and thought. It will cull and digest, for the benefit of readers, the important things from the current medical literature of the world . . . Its editorial columns will be devoted to the fostering of the high ideals which we know to be so dear to the heart of our profession.[7]

This exposition of editorial objectives elaborated and refined those previously laid out by the *Journal's* publication committee. In large measure they were achieved by Warbasse and those editors who followed him.

The *Journal's* pages for the past 90 years document most of the medical progress of the 20th century. In addition, they chronicle the multiplicity of medical care issues which have confronted and which still confront the medical profession. The scope and magnitude of these issues have greatly increased in the past decade and constitute the larger canvas against which medical progress has taken place. These issues are a major focus of concern of virtually all physicians and as such have been extensively covered in the *Journal* in recent years. Some major medical and professional issues have been the subject of special thematic issues of the *Journal* or thematic groupings of papers. In recent years subjects that have received this in-depth coverage include the acquired immunodeficiency syndrome, cigarette smoking, geriatrics, human abuse, medical malpractice, minorities in medicine, the offshore medical schools, and sports medicine. In March of this year, the *Journal* will publish its fourth thematic issue on the acquired immunodeficiency syndrome and later in the year a special issue on current perspectives on women in medicine. The *Journal* enters its ninth decade on a firm footing, carrying forward its past traditions of excellence while prepared to meet the inevitable scientific and professional challenges of the next century.

PASCAL JAMES IMPERATO, MD
Editor

1. Redway LD: Fifty years of journal publication in retrospect. *NY State J Med* 1951; 51:55-70.
2. Garfield E: Which medical journals have the greatest impact? *Ann Int Med* 1987; 105:313-320.
3. Imperato PJ: Readership survey of the *New York State Journal of Medicine*. *NY State J Med* 1987; 87:606-612.
4. Kelly EC: History of the Medical Society of the State of New York. The years between 1882-1906. *NY State J Med* 1957; 57:471-475.
5. Association Notes. *NY State J Med* 1901; 1:1-2.
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7. The New York State Journal of Medicine. *NY State J Med* 1906; 6: 130.

Topical low-dose thrombolytic therapy

Over the past decade, thrombolytic therapy has become the primary nonsurgical approach to the treatment of patients with life threatening intravascular thrombosis or embolism. By activating the systemic fibrinolytic system, the functional integrity of the circulation can be improved or restored. However, the incidence of significant hemorrhagic complications with systemic thrombolytic therapy remains high, necessitating an alternative approach in patients who experience a massive thrombosis in the presence of concomitant active bleeding. In this issue of the *Journal*, Drs Nakao and Brandstetter report similar approaches in two patients with life threatening clotting and coexistent hemorrhage, who were treated with a local infusion of streptokinase.[1,2]

The patient reported by Nakao experienced a massive pulmonary embolism with hemodynamic compromise shortly after completing radiation treatments for cervical carcinoma, complicated by vaginal bleeding. The therapeutic options for such patients are limited: systemic thrombolytic therapy, which produces both clot lysis and pulmonary hemodynamic improvement, but carries a substantial risk of exacerbating active hemorrhage,[3] or pulmonary embolectomy. Even in experienced hands, embolectomy is associated with a substantial mortality.[4] Nakao treated his patient with low doses (10,000 units) of streptokinase infused directly into the region of the thrombus. Although coagulation parameters remained unchanged, clot lysis was evidenced by hemodynamic improvement and angiographic resolution. This approach has proved useful in other cases of arterial or venous thromboembolism, although the dosing and duration of streptokinase therapy have varied. Katzen and Van Breda[5] and Ambrose et al[6] used 5,000 U/hr, approximately 5% of the usual dose, to treat arterial and pulmonary emboli, respectively. The duration of treatment was based on angiographic or clinical improvement.

The patient reported by Brandstetter depicts a clinical course which is familiar to most pulmonary specialists: exsanguinating hemoptysis due to bronchogenic carcinoma. Mortality from massive hemoptysis remains high, up to 80%,[7] despite the development of a variety of medical and surgical approaches. In this patient with inoperable lung cancer, surgical resection was not considered appropriate by the patient or his physicians, and local maneuvers were unsuccessful. Other techniques which may be useful under these circumstances include vessel embolization under angiographic guidance, topical administration of vasoconstrictor agents, and selective airway intubation to protect the intact lung from alveolar flooding with blood.[8] The conservative and creative approach taken by Brandstetter was to insert a Swan-Ganz catheter under direct bronchoscopic visualization effectively to tamponade the site of bleeding. Unfortunately, an intrabronchial clot developed which produced airway compromise. Analogously to the reasoning used by Nakao in the approach to his patient, Brandstetter administered streptokinase into the airway, but delivered a dose higher than that used by Nakao (100,000 units). Although this appeared to lyse the airway clot successfully and to effect improved ventilation, massive hemoptysis recurred and was ultimately fatal. Although no coagulation parameters were measured, blood in the ventilator tubing remained unclotted for several hours, consistent with the development of a systemic "lytic state." Lower doses of streptokinase (30,000-60,000 units) have been used in similar cases without hemorrhagic complications.[9,10]

Several clinical trials are already underway or have been proposed to clarify the optimal dosing and timing of thrombolytic therapy in patients with acute myocardial infarction, pulmonary embolism, and deep venous thrombosis. Appropriately, most trials exclude from entry patients who are at risk for major bleeding complications, yet these patients may benefit from the findings of these studies nevertheless. That some patients may benefit from topical, low-dose thrombolytic therapy seems clear from these and other reports. We should now address the important issues of selecting appropriate patients, choosing the optimal agent and dose, and developing guidelines for monitoring and criteria for terminating this form of therapy.

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1. Nakao MA: Local low-dose infusion of streptokinase for massive pulmonary embolism when systemic thrombolysis is contraindicated. *NY State J Med* 1990; 90:32-33.
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5. Katzen BT, van Breda A: Low dose streptokinase, the treatment of arterial occlusions. *AJR* 1981; 136:1171-1178.
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Evaluation of methods for identification of *Campylobacter pyloris* infection

PETER J. SANTOGADE, MD; VICTOR D. BOKKENHEUSER, MD; MOHAMMED A. FAISAL, MD;
DONALD P. KOTLER, MD; JOHN V. SCHOLES, MD; PETER R. HOLT, MD

ABSTRACT. Fifty unselected patients undergoing routine esophagogastroduodenoscopy were evaluated for infection with *Campylobacter pyloris* (CP). Antral specimens were cultured, and biopsies from the antrum and the body of the stomach were examined histologically. Specimens of antral brushings were analyzed with Gram stain, and urease testing was performed on gastric aspirates, antral brushings, and antral biopsy homogenates.

Twenty-seven (54%) patients were CP-positive by silver stain and/or culture of mucosal biopsies. The simplest and fastest diagnostic method was Gram stain of antral brushing, which was 93% sensitive and 100% specific. CP-negative patients were more likely to have normal histology in antrum and body tissues, while CP-positive patients usually exhibited superficial or chronic gastritis ($p < 0.01$). Using ELISA technique, 67% of all patients and 89% of CP-positive patients had serum antibodies against sonicated CP organisms. We conclude that evidence of gastric CP infection is common, is associated with inflammatory changes of the gastric mucosa, is suggested by finding antibodies to CP in serum, and can be accurately and rapidly diagnosed by staining of endoscopically derived cytology and biopsy specimens.

(NY State J Med 1990; 90:4–7)

In 1983, Marshall[1] and Warren[2] reported an association between the cultivation of spiral bacteria from the gastric epithelial surface and evidence of active chronic gastritis. Spirochete-like organisms and gastritis have been intermittently associated in the literature since the late 1800s.[3,4] In the past five years, many studies have linked the organism, presently classified as *Campylobacter pyloris* (CP), with a spectrum of gastroduodenal diseases including epidemic acute gastritis,[5,6] dyspepsia,[7] peptic ulcer disease,[8] gastroesophageal reflux,[9] and gastric atrophy. The most striking association has been with peptic ulcer disease, and several reports suggest that more than 75% of patients with duodenal ulcer and about 70% of gastric ulcer patients have CP infection.[8] The majority of these patients show simultaneous antral gastritis.

The suggested infectious etiology of peptic disease thus makes *C pyloris* an organism of potential importance. Its fastidious growth requirements and the difficulty with which it is identified by usual histologic stains have complicated its identification in routine specimens. Since CP produces a powerful urease enzyme, urea hydrolysis of urea-containing media in which gastric biopsies are incubated has been used to identify the presence of CP.[10] However, the specificity and sensitivity of these techniques have not been established.[11,12]

Infection with CP results in both secretory and serum antibody responses.[7,13,14] It has been stated that antibodies to CP are uncommon in patients with normal antral histology, in asymptomatic blood donors, or in antenatal females,[15] but are found in 90–94% of patients with gastritis.

The aims of the present study were to determine the prevalence of CP infection in patients referred for upper gastrointestinal endoscopy for routine indications at this institution, to assess the reliability of several of the currently established methods for identifying CP infection, and to compare endoscopic findings and histologic changes in CP-positive and CP-negative patients.

PATIENTS AND METHODS

The study group consisted of 50 consecutive unselected patients referred for esophagogastroduodenoscopy, who agreed to participate in this study. Indications for endoscopy included evaluation of gastrointestinal bleeding, epigastric pain, or dyspepsia, and were not different from those indications for patients not selected for study. Patients were excluded if they were actively hemorrhaging or were at risk for hemorrhaging from biopsy sites, as evidenced by platelet counts of less than 50,000/cm or prothrombin times more than 4 seconds above control. All patients signed informed consent forms, and the study was approved by St Luke's/Roosevelt Hospital Institutional Review Board on December 2, 1986.

Immediately prior to endoscopy, a blood sample was obtained, and serum was separated and frozen at −90°C for later serologic assays for CP antibodies. The specimens that were collected at the time of the endoscopy and the tests performed are summarized in Table I. A gastric aspirate was obtained through an endoscopic channel at the beginning of the procedure. A 0.1-mL aliquot was inoculated into a standard nonbacteriostatic urea agar slant (BBL Microbiology Systems, Cockeysville, Md) containing phenol red as indicator. After the duodenum and stomach were examined visually by the endoscopist, a sheathed cytology brush was passed and the antrum was circumferentially brushed. The brush was withdrawn and inoculated in urea agar. After rebrushing of the antrum, dry cytology slides were pre-

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From the Gastrointestinal Division of the Department of Medicine (Drs Santogade, Faisal, Kotler, and Holt) and the Department of Pathology (Drs Bokkenheuser and Scholes), St Luke's/Roosevelt Hospital Center, and the College of Physicians and Surgeons, Columbia University, New York, NY.

Address correspondence to Dr Holt, Chief, Division of Gastroenterology, St Luke's/Roosevelt Hospital Center, Amsterdam Ave at 114th St, New York, NY 10025.

TABLE I. Specimens Obtained and Tests Performed

Specimens	Tests
Gastric aspirate	Urease activity
Antral brushing	Urease activity; modified Gram stain
Endoscopic pinch biopsies	
Antral (homogenized in saline)	Urease activity; modified Gram stain; microaerophilic culture
Antral and body (fixed in formalin)	Histology (by hematoxylin-eosin stain) Warthin-Starry silver stain
Serum	Antibody by ELISA

pared. Subsequently, an endoscopic pinch biopsy forceps was passed, and two pairs of antral biopsy specimens and one pair of biopsy specimens from the lesser curvature of the gastric body proximal to the angulus were obtained.

Warthin-Starry silver stains were read independently by two observers. Histologic observations in hematoxylin and eosin stained specimens were evaluated according to the criteria established by Whitehead,[16] which grades the chronicity and depth of the inflammation and the presence of metaplastic changes. Cytology specimens were stained by a modified Gram stain using 1.0% carbol fuchsin instead of safranin as counterstain to enhance bacterial color.

One antral biopsy pair was immediately homogenized in 0.5 cc nonbacteriostatic saline at a dilution of approximately 1:100 in a Teflon-glass tissue homogenizer, inoculated in the endoscopy suite onto Skirrow's medium, and cultured microaerophilically using CampyPak Plus (BBL). The cultures were then kept at 37°C, and growth was evaluated daily on days three to nine. Cultures which grew small, translucent colonies on Skirrow's media were Gram stained and tested for urease activity. Urease-positive colonies consisting of typical gram-negative curved rods were subcultured on chocolate agar (Choc II, BBL). These subcultures were again examined for morphologic abnormalities and tested for oxidase, catalase, and urease activity, and for inability to ferment dextrose.[17]

Urea slants were incubated at 37°C, and urease activity was read at 24 hr. A positive test was indicated by a color shift of the medium from cream to dark pink. Serum IgG antibodies to CP were sought by an enzyme linked immunosorbent assay (ELISA), using a method adapted from Whalen (J. W. Whalen, personal communication, 1987). A sonicated mixture of seven CP cultures was used as antigen. The patients' sera were diluted 1:200 and 1:800 and studied with positive and negative control sera. After incubation at 37°C for 90 min, a 1:50 dilution of goat antihuman IgG conjugated to alkaline phosphatase was added, incubated for 60 min, and the optical density (OD) recorded 15 to 30 min later using an automated spectrophotometer. The test serum was regarded as containing CP antibodies (CP-positive) if the positive control sera reached an OD of 1.0 to 1.3, the test serum OD was greater than 0.3, and the ratio of the OD of the antigen-containing well to the noncoated well was greater than 3.5.

Patient groups were compared by chi-squared analysis.

RESULTS

Twenty-seven (54%) of the 50 patients had evidence of CP infection as judged by the established criteria of typical organisms in silver stained specimens or isolated in culture on special media. The mean age of CP antigen-positive patients was 61 yr and CP-negative patients was 51 yr, with an age range of 26 to 81 yr; the majority of patients in both groups were female. A high percentage of CP-positive patients also were diagnosed by examining the modified Gram stain of antral brushings. The sensitivity of this method, silver stain of biopsies, and of culture all exceeded 85% with a 100% specificity (Table II).

TABLE II. Evaluation of the Accuracy of Biopsy, Brushing, and Urease Testing

	CP-positive (N = 27)	CP-negative (N = 23)	Sensitivity Percent	Specificity Percent
Positive tests				
Biopsy—silver stain	23	0	85	100
Biopsy—culture	24	0	89	100
Brushing—Gram stain	25	0	93	100
Aspirate urease	11	3	41	87
Biopsy homogenate urease	7	1	26	96
Brushing urease	17	2	63	91

In the 23 patients who demonstrated diagnostic silver stained organisms on histologic analysis, typical organisms were seen in both antral and body biopsies in 13, only in an antral biopsy in seven, and only in a biopsy from the body of the stomach in three. Modified Gram staining of antral brushings was positive in 25 of the 27 CP-positive patients (Fig 1), whereas Gram staining of biopsy homogenates was positive in only seven. Urease testing of gastric aspirates, homogenized biopsies, and antral brushings was less sensitive (Table II) than other methods. Of the specimens for urease tests, antral brushings proved more suitable than gastric aspirates or biopsy homogenates, with a 63% sensitivity in identifying infected patients (Table II). False positive urease tests, however, were observed with each method.

By endoscopic inspection, similar diagnoses were made in the two groups (Table III). However, ten chronic peptic ulcers, six gastric, and four duodenal ulcers were found in the 27 patients showing evidence of CP infection, compared to four ulcers in 23 patients without CP (not significantly different). Three of the four ulcers in CP-negative patients occurred during treatment with nonsteroidal anti-inflammatory medications, whereas only three of the ten CP-positive patients diagnosed as having peptic ulcers were using these drugs.

CP-negative patients were more likely to show normal histologic findings in the antrum and body of the stomach (Table IV). Conversely, CP-positive patients were more likely to demonstrate superficial body gastritis than were patients without evidence of this infection ($p < 0.001$). The presence of either superficial or chronic gastritis in antrum or body was significantly associated with CP infection ($p < 0.01$). Significant differences were retained when all of the data were analyzed together to reject the null hypothesis ($p < 0.02$).

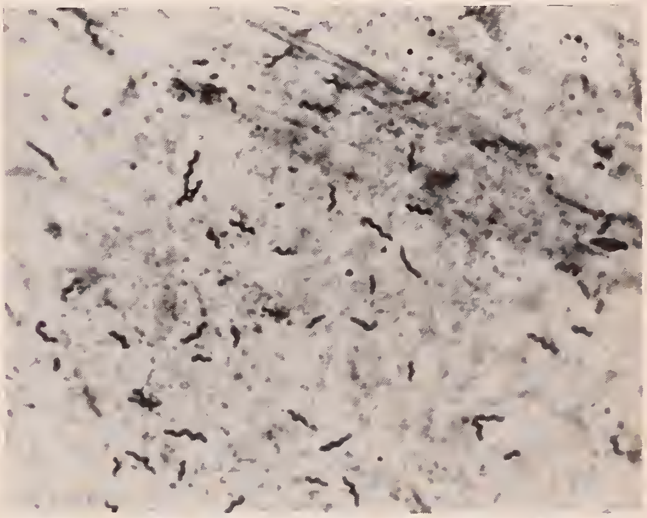

FIGURE 1. Modified Gram stain of antral brush cytology. Specimen from a patient with CP infection. Note abundant spiral bacilli adjacent to amorphous mucin and cellular debris (magnification × 2000).

TABLE III. Range of Diagnoses by Endoscopy*

Diagnosis	CP-positive (N = 27)	CP-negative (N = 23)
Normal	7	7
Esophagitis	1	4
Gastritis		
Body	7	4
Antrum	14	11
Duodenitis	5	7
Peptic ulcers		
Gastric body	2	1
Gastric antrum	4	2
Duodenum	4	1

* More than one diagnosis was made in 30 patients. The prevalence of these diagnoses between CP-positive and CP-negative groups was not significantly different by chi-squared testing.

Serologic evidence of current or prior CP infection was evaluated by ELISA. Sixty-seven percent of the total group of study patients had serologic evidence of CP infection (Table V). In patients with histologic or culture evidence of CP, 24 of 27 (89%) also showed a positive CP serology. Of these 24, 19 had titers of 1:800 or greater, and five (19%) had titers between 1:200 and 1:800. Three patients who were positive for CP infection by all three methods of silver stain, culture, and Gram stain of cytology specimens had no detectable serum antibody. Sera were available from 21 of the 23 patients with no evidence of CP infection; 62% (13 of 21) were antibody-negative, and eight had a positive antibody titre (five at 1:800 dilution). Five of these eight patients had received antibiotics for an unrelated condition in the four weeks prior to endoscopy. The sensitivity of the ELISA assay for anti-CP was 89% and the specificity was 62%.

TABLE V. Evaluation of the Accuracy of Detecting Serum CP Antibodies*

CP Antibody	CP-positive (N = 27)	CP-negative (N = 21)	Sensitivity	Specificity
			(percent)	
Present	24	8	89	62*
Absent	3	13	—	—

* Chi-squared significance $p < 0.005$.

DISCUSSION

The present study confirms that *C pyloris* infection is common in the population of patients referred for upper gastrointestinal endoscopic evaluation in New York. In this study, we found an association between the presence of CP and gastritis or peptic ulcer disease. Recent studies have used histologic scales other than Whitehead's[16] to grade gastritis associated with CP infection. The term "active chronic gastritis" has recently been used to replace Whitehead's categories of superficial or chronic gastritis, denoting a mixed inflammatory infiltrate in which polymorphonuclear cells accompany lymphocytes and plasma cells.[18] The majority of the patients presented here with "chronic gastritis" would fall into this category.

TABLE IV. Range of Histologic Diagnoses in Study Patients*

Histology	CP-positive (27)	CP-negative (23)
Antrum		
Normal	1	12†
Superficial gastritis	9	2
Chronic gastritis	14	6
Atrophic gastritis	3	3
Body		
Normal	2	14‡
Superficial gastritis	14	1‡
Chronic gastritis	7	5
Atrophic gastritis	4	3

* Histologic diagnostic criteria according to Whitehead.[16] Differences between CP-positive and CP-negative patients determined by chi-squared testing and considered significant if $p < 0.05$.

† $p < 0.01$.

‡ $p < 0.001$.

If CP causes inflammatory changes in gastric epithelia (and does not simply colonize previously inflamed mucosa as some maintain),[19] how does it effect its damage? Most likely the rapid hydrolysis of urea to ammonia results in a high ammonia concentration at the surface of the gastric epithelium, particularly at the intercellular junctions where these bacteria preferentially reside. Consequently, normal H+ transfer across the gastric mucosa may be disrupted, paracellular and transmucosal permeability increased (thus enhancing H+ back diffusion), or the protective function of the mucus layer altered.[20] Enzymes produced by CP have recently been shown to alter several properties of gastric mucus.[20]

The techniques that presently are used to culture CP involve selective media and microaerophilic culture conditions which are cumbersome and time consuming, requiring four to nine days for final identification. Silver staining of gastric biopsy material, an accepted standard method for identifying CP, has been used in the majority of studies presented to date, but this technique is costly, difficult to standardize, and requires special technical expertise. In our hands, a modification of the standard Gram staining techniques, using antral brushings, was found to be a simple, fast, and inexpensive method of diagnosing this infection. The substitution of 1.0% carbol fuchsin for safranin greatly enhanced color contrast and facilitated ready diagnosis of the organism. Our data indicate that the levels of sensitivity and specificity for this method make it comparable to either culture or silver staining. The test can be performed in the endoscopy suite within minutes of the procedure, or the air-dried, heat-fixed slides can be stored for staining at a future date.

Since CP produces the most powerful urease enzyme identified in bacteria to date, it rapidly generates the hydrogen acceptor ammonia from urea. This capacity of rapid urea hydrolysis is the basis for several approaches to identifying the presence of CP by measuring urease activity. In such studies, gastric biopsies have been incubated in a urea-containing medium and, if CP is present, ammonia is produced, resulting in a rise in pH and a readily visible indicator color change. In general, the speed and depth of color change are approximately proportional to the number of organisms present in the biopsy. In some studies, urease testing was reported to be 88–100% sensitive and 90–100% specific.[11,21] Several investigators have suggested that urease testing techniques should be used in large studies that test the relationship of CP to peptic disease because the method is reliable, inexpensive, and readily available to the endoscopist.[22,23] However, in the present study, using the standard technique available, urease testing was not found to be specific or sensitive when incubating gastric washings, antral brushings, or homogenized biopsies. Recently, a modification of the urease test, in which antral biopsies are immersed in urea agar, has been reported to be more specific, sensitive, and rapid.[23] Further experience with this technique is needed.

CP antibody assay in serum was the only noninvasive method evaluated in this study. Eighty-nine percent of patients with evidence of the presence of CP organisms in the stomach had positive serologic findings whereas only 38% of CP-negative patients were antibody positive. In at least some of these CP-negative patients showing antibody reactivity, recent antibiotic treatment may have temporarily abolished CP from the stomach. Thus, determination of the presence of high-titer antibodies to CP in serum may allow for initial screening to select subjects for more invasive techniques that lead to specific testing.

It also has been suggested that lower relapse rates in patients with duodenal ulcers treated with bismuth subcitrate (an agent used recently in other countries for the treatment of peptic disease), compared to H-2 blockers, is due to eradication of CP by the bismuth salts.[24] Bismuth causes the bacteria to swell, detach from the mucosa, and lyse. However, complete bacterial eradication is difficult, even with a four-week course of therapy with bismuth and an antibiotic, such as amoxicillin, and relapses occur in up to 30% of patients so treated. A clearer picture of the importance of *Campylobacter pyloris* in peptic ulcer disease awaits the development of an animal model and controlled therapeutic trials with agents that effectively eradicate the organism from the stomach.

Acknowledgments. The authors thank A. Tierney for assistance with the statistical data. This work was supported in part by NIH grants AG00124 and AI21414 and the Overseas Maritime Corporation.

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Cervical angina

BERNARD JACOBS, MD

ABSTRACT. **Cervical angina, resembling true angina pectoris, but resulting from cervical spondylosis and nerve root compression, is also known as pseudoangina. This report describes 164 patients treated over a 22-year period. Patients included 103 men and 61 women, with ages ranging from 45 to 68 years and averaging 54 years of age. The duration of symptoms prior to definitive diagnosis averaged ten months and ranged from ten to 18 months. Most patients had consulted at least two cardiologists prior to diagnosis. The results of stress testing were abnormal in ten patients, but none underwent angiography. Symptoms common to all patients, in varying severity, included neck pain and stiffness, occipital headache, arm pain with sensory symptoms. Neurologic deficit was found in only three instances. The majority of patients responded satisfactorily to a standard nonsurgical regimen, employed for at least three months, involving the use of a hard collar, intermittent traction, isometric exercise, and a combination of anti-inflammatory and muscle relaxant medications. In cases where disability persisted, myelography was usually employed and when confirming nerve root compression, anterior disc excision and spine fusion were performed. Such treatment was required in only 38 cases and resulted in complete relief of complaints in all but five instances in which fusion failure required re-operation with ultimate success. Fusion usually was completed in three months, during which time the patient was required to wear a hard collar.**

(NY State J Med 1990; 90:8–11)

Cervical angina, resembling angina pectoris, but secondary to arthritic changes and cervical radiculopathy, is also known as pseudoangina. The first comprehensive report dealing with this syndrome was by Oille[1] who, in 1937, reported 197 cases of chest pain of nerve root origin. In 1957, Davis[2] reported his experience with 100 cases of radicular chest pain, observing that commonly present parasternal tenderness was a useful clue in the differentiation from true angina. Jackson[3] observed that unusual heart symptoms without electrocardiographic changes occurred after trauma to the neck and in patients with degenerative changes of the cervical spine. Jackson also noted that in some patients tachycardia or palpitations may develop when recumbent. In 1964, Masters[4] reported on the cause of anginal and noncardiac chest pain and affirmed the association of pseudoangina secondary to cervical arthritis. Masters also observed that in some instances pseudoangina pain could be relieved by the use of nitroglycerin.

METHODS

The case histories of 164 patients diagnosed as having pseudoangina (cervical angina) over a 22-year period from 1963 to 1985 were retrospectively examined (Table I).

RESULTS

The average age of these patients was 54 years and ranged from 45 to 68 years. There were 103 men and 61 women. The average duration of symptoms prior to definitive diagnosis was ten months, and ranged from ten to 18 months. Most patients had seen more than one cardiologist before the diagnosis of cervical angina was made. Thirty-six had undergone stress testing, the results of which were abnormal in ten instances (Table II). No patient had undergone angiography.

Symptoms common to all patients included neck and arm pain, occipital headache and sensory symptoms in the upper extremity of varying intensity and type (Table III). Three patients recalled experiencing a crushing type of chest pain that had been relieved by the use of nitroglycerin. However, 12 other patients without this type of pain had also gained relief with nitroglycerin (Table IV).

Neurologic examination revealed an absent biceps reflex in eight patients. Seven patients demonstrated weakened hand grip, and in 49 cases there was localized parasternal tenderness, usually at the 5–7 costochondral junction. Other clinical findings common to all patients included restricted neck motion and chest or arm pain. Such pain may be increased by downward pressure on the head accompanying active motion in any direction, especially rotation and lateral bending.

Sixty-two patients had experienced chest pain while at rest, while 35 had experienced pain with specific physical activity, such as racket sports or volleyball. A history of prior neck injury was obtained in 155 patients, ten caused by work injury and 145 involving motor vehicle collisions. Plain neck radiographs demonstrated disc space narrowing at one or more levels associated with osteophyte formation and neural foramen encroachment. A presumptive diagnosis of cervical angina was made based upon these radiographic findings together with the clinical manifestations.

Treatment Approaches. All patients received a standardized regimen of nonsurgical treatment, comprising neck care precautions and avoidance of unnecessary neck stress, such as repetitive neck rotation and extension, and prolonged flexion. Patients were advised to use a plastic neck collar with adjustable height for those activities known by the patient to produce pain, such as housework and gardening. Patients were advised to avoid racket sports, volleyball, basketball, and swimming. Neck traction, either at home or in a physical therapy clinic, was prescribed, not to exceed 20 pounds.

Approximately 35% of patients reported immediate relief of chest or arm pain shortly after traction use. In 10% of cases, traction appeared to aggravate pain. Reports of either relief of pain or even increased pain in traction serve to confirm a diagnosis of cervical or radicular angina. One hundred twenty-six patients were treated solely by nonsurgical means (Table V); 76 obtained complete relief of chest or arm pain and only occasionally required the use of analgesic medication. Except for 25 patients, all were able to return to prior occupations, each of the exceptions being involved in medicolegal actions resulting from injury. Medications used included nonnarcotic analgesics, muscle relaxants, and anti-inflammatory and

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anti-anxiety medications.

Surgical treatment was required in 38 instances in which nonsurgical treatment, employed for at least three months, had failed. Each patient underwent pre-operative myelography (Fig 1). Discography was employed in ten instances in which the myelographic findings were deemed to be equivocal (Fig 2). Discography involves injecting contrast media into the disc under local anesthesia. This increases interdiscal pressure, usually reproducing symptoms and defining the extent of disc disruption. In each case the discography reproduced chest pain and confirmed that the disc was herniated. In all other instances, myelography demonstrated nerve root compression. In each case, there was reproduction of either the arm or chest pain in the course of the discogram or shortly thereafter.

The surgical treatment employed consisted of disc excision with osteophyte removal via the anterior approach, combined with interbody fusion. After removal of the diseased disc and osteophytes, the disc space is trephined and a dowel graft obtained from the iliac crest is inserted. This procedure and its rationale were previously reported by the author[5] and, except for minor modifications, the technique was based on the procedure first described by Cloward.[6] In ten instances, one disc level was dealt with; in 20 cases, two disc levels were operated on simultaneously; and in eight instances, three disc levels were dealt with simultaneously (Table VI). The most common site of discography was C 5-6, involved in 39 cases, closely followed by the C 6-7 disc, which was involved in 27 instances.

The postoperative regimen required the use of a firm neck collar until radiographs demonstrated the presence of bony fusion, which was achieved in all but five instances within two to three months of surgery (Fig 3). All patients were asymptomatic when the fusion was completed. Four of the five cases with failed fusions experienced sufficient chest or arm pain to warrant a second operation, which

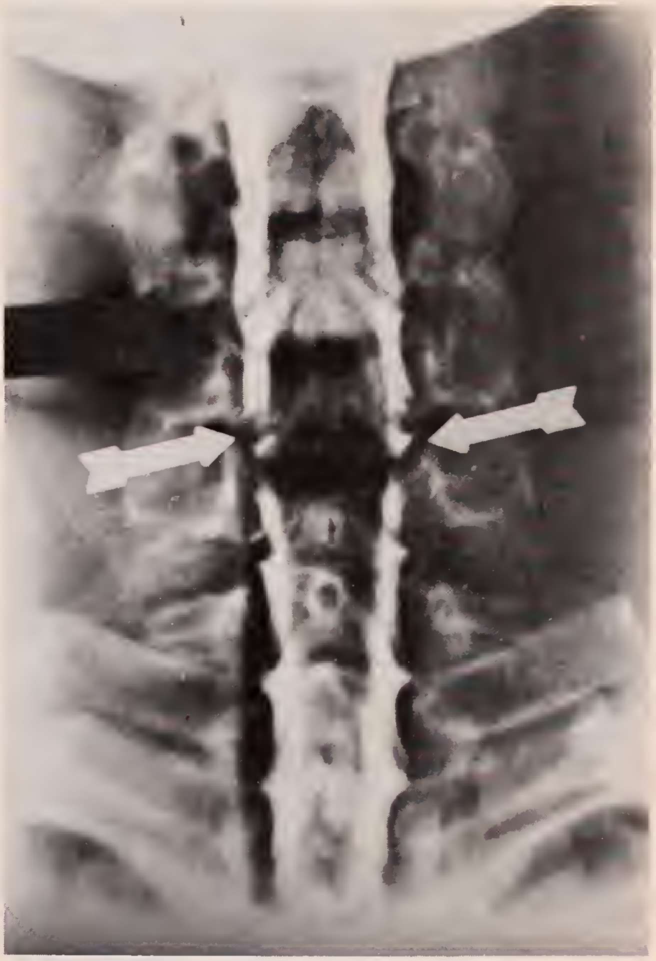

FIGURE 1. Typical preoperative myelogram showing multiple root defects.

TABLE I. Patient Characteristics

Number of Patients	164
Sex	
Male	103
Female	61
Age range	45–68 yr
Average age	54 yr
Prior trauma	155
Vehicular "whiplash"	145
Work injury	10
Duration of symptoms	10–18 mo
Average duration of symptoms	13 mo

TABLE II. Preoperative Evaluation

Diagnostic Test	Number of Patients
Stress testing	36
Repeated electrocardiographs	164
Myelography	28
Discography	10

TABLE III. Common Manifestations Associated with Cervical Angina

Neck and arm pain
Upper extremity numbness, paresthesias
Restricted neck motion
Parasternal tenderness
Occipital headache
Arm fatigue
Pain often relieved by neck traction

TABLE IV. Less Common Manifestations Associated with Cervical Angina

Reflex loss
Muscle atrophy
Symptoms aggravated by neck traction
Chest pain relieved by nitroglycerin

TABLE V. Treatment Approach and Results

Approach	Number of Patients
Nonsurgical	126
Complete relief	76
Mild residual symptoms	50
Surgical: disc excision and fusion	38
Complete relief	33
No relief due to failed fusion	5
Relief after re-fusion	164

TABLE VI. Site and Extent of Disc Surgery

Level	Number
C 4–5	5
C 5–6	39
C 6–7	27
C 7–T1	3
Single disc surgery	10
Two disc surgery	20
Three disc surgery	8

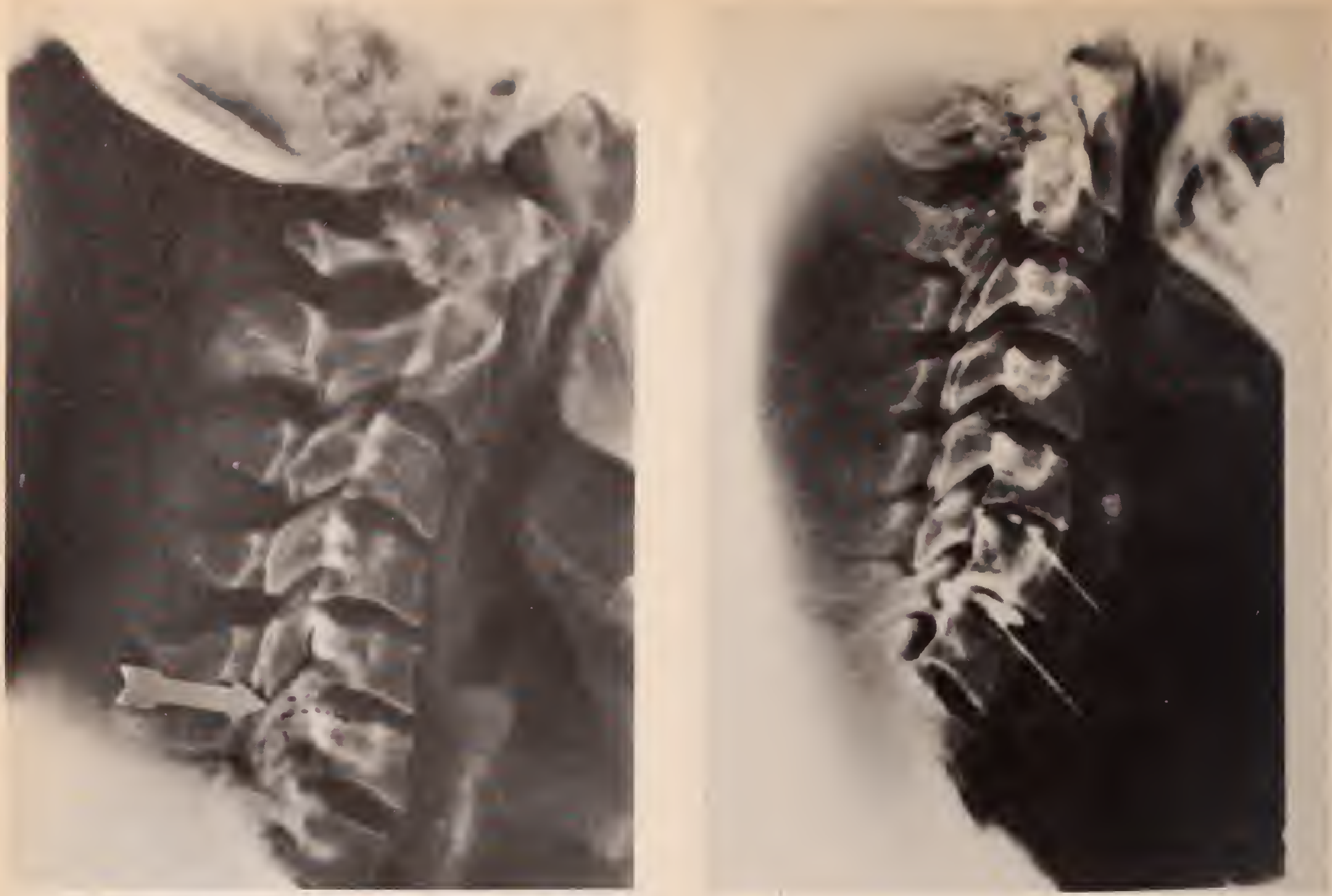

FIGURE 2. Left, Lateral view demonstrating posterior osteophyte formation. Right, Positive discogram at two levels.

succeeded in obtaining a fusion in all cases. The remaining patients with a failed fusion had experienced only mild and episodic discomfort. In follow-up studies performed at ten and 15 years after the initial surgery, only 8% of patients subsequently experienced symptomatic disc degeneration and arthritis adjacent to the fusion level.

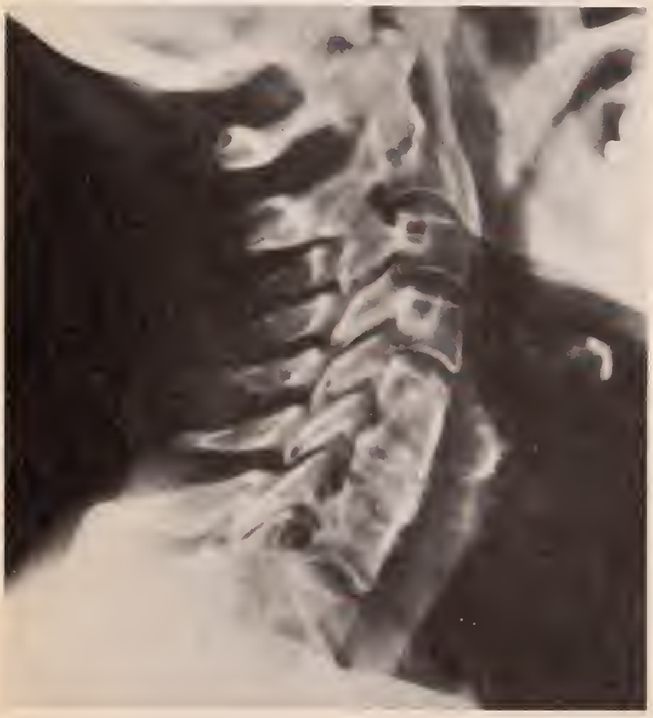

FIGURE 3. Postoperative two-level fusion at three months after surgery.

DISCUSSION

Brodsky's[7] comprehensive report dealing with cervical angina is of considerable interest. He accumulated 439 cases over a 19-year period, noting antecedent vehicular injury in 36 patients, abnormal serial electrocardiograms in 18 cases, positive stress testing in three instances, and abnormal angiography in 25 cases. Eighty-seven patients needed surgery similar to that described here. However, in contrast to the experience reported here myelographic studies were deemed normal in 19 cases. Brodsky reported excellent results in 78% of cases, with failure of fusion in 17 patients, of whom 14 required additional surgery. Brodsky attributed chest pain to compression of C 4–8 nerve roots. In my experience, the sixth and seventh nerve roots were most frequently involved, and I believe that chest pain is mediated via the medial and lateral pectoral nerves. Jackson[3] ascribed chest pain to irritation of the cardiac sympathetic nerve supply by cervical osteophytes.

Cervical angina or pseudoangina is a well-proven clinical entity caused by cervical arthritis and nerve root compression. Prompt and accurate diagnosis requires a strong sense of suspicion in patients with inadequately explained chest pain. Useful clues to diagnosis include parasternal tenderness, abnormal radiographic findings, chest or arm pain induced by active neck motion combined with downwards head pressure, and the ameliorative effect of neck traction. In the vast majority of cases, satisfactory relief of symptoms is accomplished by nonsurgical means. Surgical intervention is also capable of producing excellent results in most instances. In this author's opinion, all patients require preoperative myelography, or discography when the myelogram is not sufficiently positive. The need for angiography is always the option of the referring or consulting cardiologist.

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MEDICAL COMMUNICATIONS DAY

The Practicing Physician: Transmitter and Receiver of Medical Information

The Medical Society's Concern with the Problem. To extend medical knowledge and advance the science and art of medicine, to promote the betterment of public health, these are among the purposes of the Medical Society of the State of New York. Similar worthy aims certainly motivate all serious medical organizations.

Even to begin to fulfill these purposes each society is at once involved, consciously or not, in a pattern of medical intercommunication. In the smaller units this may be nothing more than a simple periodic get together for informal exchanges of experiences, case presentations, or general thoughts. Or there may be a great convention, and a vast complex of bureaus, committees, councils, and publications, as is true of the AMA and some of the larger state medical societies.

However simply or complexly expressed, the basic purposes remain paramount, and a society can best measure its success in terms of how consistently its motivations find expression in the orientation and day-to-day practice of its members. This cannot be left entirely to chance.

No doubt it is true that becoming and remaining well informed in the science and art of medicine, in its broadest sense, is the individual responsibility of the physician. Nevertheless, it is also a legitimate concern of medical societies to facilitate this, to stimulate it, and to give aid in every possible way. The society can help to create and maintain the medically receptive mind, scientific curiosity in practice, and balanced judgment of scientific works. It can expose and reject the inept, the unworthy, the superstitious, and the unscientific. Further, whatever it does directly and individually can be augmented by the leadership and standards it sets for or exacts from organizations or institutions dependent on its approval.

In the Medical Society of the State of New York we have long recognized our responsibilities to our members in this area. Over the years there has been a constant striving to improve our methods and technics. . . .

We sponsor and promote programs of postgraduate education, scientific meetings, and a scientific journal, among a host of other diverse efforts. . . .

For an established medical society . . . a journal acts as a twofold means of communication. It contains information concerning the society itself, its official actions, its plans, and its program. It is also a scientific publication. It preserves in written form the best of the discussions and presentations made at the society's scientific meeting. It publishes worthy scientific contributions of its members.

The important thing to remember, however, is that if the medical society publishes a journal, that journal must be of high quality. Only thus can members who receive it, usually as a benefit of dues, be informed intelligently on scientific developments and procedures.

In New York State, we have for the past sixty years published the NEW YORK STATE JOURNAL OF MEDICINE. Over the years this has developed from a means of recording official meetings and activities to its present philosophy. The Publication Committee of our State Medical Society and the editors join in putting into actuality a plan to make our JOURNAL a truly outstanding scientific publication. Indeed, we have been charged by the House of Delegates and the Council of our Society to exert every effort to improve our JOURNAL's appearance, to enhance the scientific importance of its content, and to widen its readership. We feel that only thus can we fulfill our responsibilities to our membership and to others to provide the best in medical information. . . .

ALFRED P. INGEGNO, MD
(*NY State J Med* 1960; 60:347-3528)

A critical appraisal of contact lens disinfection

MICHAEL B. STARR, MD

Over the last several years, ophthalmologists have become increasingly concerned about contact lens-associated corneal infections. Although secondary infection has always been a potentially blinding complication of contact lens wear, the population at risk has become so large (18 million contact lens wearers in the United States, of whom approximately 13 million wear soft lenses and the remainder wear hard polymethylmethacrylate (PMMA) or rigid gas permeable (RGP) lenses),[1] that such infections appear to be more common.[2] Furthermore, these infections may be caused by particularly virulent organisms, such as *Pseudomonas aeruginosa* and *Acanthameba* species, which occur more commonly in association with contact lens use, and especially with soft contact lenses.[3]

These events have occurred in spite of the US Food and Drug Administration (FDA) efficacy requirements for commercially marketed lens disinfection regimens. These infections have been attributed in large part to less than optimal compliance with such regimens among contact lens wearers. To a lesser extent, inherent failures in such regimens are also possible.[4] Both the Centers for Disease Control (CDC)[5] and professional organizations such as the Contact Lens Association of Ophthalmologists (CLAO)[6] have published guidelines for contact lens care, including recommended cleaning and disinfection procedures, aimed at increasing awareness among the lay and professional communities of this problem and disseminating information on the current state of knowledge in preventing such infections. Since most of this disseminated information has been published in the ophthalmologic and optometric literature, the non-ophthalmic practitioner may not be aware of the problem or currently recommended guidelines. Although improper compliance with lens care systems currently appears to be of paramount importance in the pathogenesis of such infections, a discussion of compliance is beyond the scope of this review, which is intended to focus on the efficacy of different lens care regimens as directed by the manufacturers.

CONTACT LENS CARE REGIMENS

Soft contact lens care regimens comprise several sequential steps. On removal of the lens from the eye, a surfactant cleaner is rubbed over the lens surface to remove lipid and mucin deposits and, to a lesser extent, protein, which are predominantly derived from the tear film. Surfactant cleaner, which is irritating on contact with the ocular surface, and loosened deposits are then rinsed from the lens with sterile saline. Lenses are then disinfected by immersion in a disinfectant solution or saline which is heated by an electrically powered thermal disinfection unit. Another rinse with sterile saline may be used to remove chemicals and dead microorganisms before reinsertion of the lens onto the ocular surface. Generally, weekly enzymatic cleaning with a proteolytic enzyme serves to enhance removal of tightly bound proteins from the lens surface, as an additional step in lens care.

This sequence of lens care, including cleaning, disinfection, and rinsing, has been developed for use in its entirety, without deletion of any step, to insure safe lens wear. To cut costs or save time, however, some lens wearers deviate from the recommended lens care regimen and thereby substantially increase the risks of lens contamination and ocular infection. Since contact lens deposits provide a foothold for microorganisms, daily and weekly enzyme cleaning are important steps in reducing the microbial burden, in addition to enhancing lens comfort and longevity. Such cleaning, however, does not accomplish lens disinfection nor obviate the need for a separate disinfection step. Similarly, soaking lenses in saline containing preservatives at concentrations intended to discourage microbial growth in the solution also does not disinfect the lenses. Use of nonpreserved saline or homemade saline solution constituted from distilled water and salt tablets or capsules is extremely dangerous since the distilled water is a source of abundant microbial contamination, including the particularly virulent *Acanthamebae*. Use of such homemade solutions should be strongly discouraged, especially when methods other than thermal disinfection are used, and at points in the lens care sequence that do not precede exposure to thermal disinfection. Since all contact lens care solutions are commercially formulated for one-time use, they all should be discarded after any of the care steps. Preserved solutions that are not fully used by three months after purchase should be discarded and replaced to minimize microbial contamination that can occur in spite of preservatives.

Continuous wear of soft contact lenses for periods of up to one month before removal and initiation of the lens care sequence is considered extended wear. Concern that extended wear may increase the risks of corneal infection has prompted recommendations for shorter durations of

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uninterrupted lens wear and more frequent lens cleaning and disinfection. The optimal frequency for lens removal is not known, nor is the relative risk of extended versus daily wear. Reliable statistics for the incidence of infection among both types of lens wearers have been elusive, since the size of the lens-wearing population at risk has been unknown in the retrospective studies conducted thus far. In spite of this qualification, a strong impression persists among many eye care professionals that extended wear represents an increased risk of corneal infection over daily wear, and that more frequent lens removal, cleaning, and disinfection would help reduce such risks.

Hard and rigid gas permeable contact lenses are also subjected to daily surfactant cleaning, and are stored dry or placed in chemical disinfectants, respectively, since both lens types are deformed by thermal disinfection. Such lenses do not have the water content of soft lenses, and their matrix is thereby far less likely to be contaminated by microorganisms. Disinfection procedures are therefore less stringent than for soft lenses, but remain necessary to prevent surface contamination. In fact, clinical experience has shown PMMA and RGP lens-associated infections to be less frequent but hardly nonexistent.

EVALUATION OF DISINFECTION EFFICACY

The efficacy of contact lens disinfection systems is evaluated by several FDA mandated tests. In the modified United States Pharmacopeia XX Preservative Effectiveness test, disinfectant solutions are incubated with 10^6 colony forming units (CFU)/mL of five test organisms: *Staphylococcus epidermidis*, *Pseudomonas aeruginosa*, *Serratia marcescens*, *Candida albicans*, and *Aspergillus fumigatus*. Bacteria must be reduced by a factor of 10^3 CFU and fungi held at initial inoculum levels for two weeks. The same test solutions are then reinoculated with 10^5 CFU of the same organisms, and residual antimicrobial activity must be sufficient to again reduce bacterial levels by a factor of 10^3 and inhibit fungal growth for another two weeks.[7] This rechallenge test addresses a problem with user compliance, whereby disinfectant solutions may not be discarded after each use as recommended, but may be reused on one or more occasions. This evaluation, however, is conducted in a system free of contact lenses or lens cases, which have been shown to add a burden to the antimicrobial activity of a disinfectant.

Disinfectant efficacy is also evaluated by calculating the time required to reduce microbial populations by 10%, or 1 log unit. This time, expressed in minutes, is calculated for the five organisms noted above, as well as Herpes simplex virus, and is known as the "D value." Although no specific D value is required by the FDA, the duration of any chemical disinfection regimen must be nine times the largest D value obtained among the six test organisms (sufficient to reduce an inoculum of 10^6 organisms to 10^0 plus an additional safety factor of 3 D values).

In the Contribution of Elements test, each component in the contact lens care system, including cleaning and rinsing, as well as the incubation with disinfection solution, is evaluated separately with respect to its ability to reduce microbial populations on the contact lens. This test is important in its demonstration of the efficacy of surfactant cleaners and sterile saline rinses on reducing lens surface contamination, often effective in reducing CFU by a factor of 10^3 to 10^4.[8] Contact lens deposits may be composed of many substances including proteins, lipids, mucin, and inorganic materials, which accumulate to a variable extent on all contact lenses. In addition to compromising lens surface integrity, leading to problems with lens longevity and comfort, such deposits may be responsible for hypersensitivity reactions, and provide a foothold for microorganisms. Poor lens care compliance may include less than recommended use of surfactant and enzymatic lens cleaners, and significantly add to the risk of contact lens-associated infection.

In the "20 item test," 20 contact lenses are inoculated with 10^6 CFU of each of the six microorganisms noted above. The contaminants are inoculated in an "organic soil" containing both dissolved and particulate proteins, simulating the environment of a tear film-coated contact lens mixed with contaminating microorganisms. After a period of incubation, the lenses are treated with the entire disinfection regimen, from cleaner through disinfection and any final rinses, as recommended by the manufacturer, and all 20 contact lenses must be culture negative for two weeks. In a modification of this test used to evaluate thermal disinfection systems, only *Streptococcus faecalis* is used as the test organism. Since current technology does not permit destruction of bacterial spores without also destroying soft contact lenses, disinfection systems do not sterilize the lenses, and growth of spore forming bacteria, such as *Clostridium* and *Bacillus* species, is permitted in the 20 item test. Corneal infection due to these spore-forming bacteria in association with contact lens wear, although rare, has occurred.[9]

Criticisms of the evaluation techniques just described have included insufficient standardization of testing procedures permitting variation in culture results, the inability to accurately calculate "D values" for microorganism and disinfectant combinations that do not follow linear semilogarithmic killing curves,[8,18] and lack of FDA standardized evaluation of disinfection of *Acanthameba* organisms. Disinfection systems that are FDA approved, therefore, meet certain minimum and theoretically equivalent standards. However, different types of disinfection systems are not thereby equivalent in efficacy. In fact, differences in these systems are the basis for CDC and CLAO guidelines, and should be strongly considered when choosing a disinfection regimen.

INFECTIOUS KERATITIS

A panoply of microorganisms cause contact lens-associated ocular infections, including bacteria, fungi, and *Acanthamebae*.[2,3,11,14] Although sporadic or epidemic cases of ocular viral infection due to virally contaminated contact lenses have rarely been reported, the possibility of transmission by this route is of great concern.[12] Human immunodeficiency virus (HIV) and hepatitis B virus (HBV) are both found in the tears of infected individuals, as are adenovirus and herpes simplex virus, to name some of the more prominent viruses of concern; furthermore, these may be present in individuals asymptomatic for ocular infection.[11] Since sharing of contact lenses among individuals is unusual, risks of contact lens-transmitted viral infections have been more relevant to the contact lens fitting sets of eye care professionals who may not be able to identify a carrier based on clinical evidence of ocular infection.

Among bacteria, gram-negative rods have been capable of fearfully rapid corneal destruction, and recent epidemiologic data suggest that such organisms may frequently cause contact lens-associated corneal infection. In one study, 78% of culture-positive cases of keratitis were due to *Pseudomonas aeruginosa*.[13] Much less common, but perhaps most destructive of all potential pathogens, and most resistant to currently available forms of treatment, are *Acanthameba* species.[14] This great variety of potential pathogens places differing demands on lens disinfection regimens. In general, lens disinfection regimens kill bacteria most effectively, with variable effectiveness in disinfection of viruses and fungi, and, except for heat disinfection, generally unsatisfactory activity in killing *Acanthamebae*.

THERMAL DISINFECTION

The various classes of contact lens disinfection systems have advantages and drawbacks peculiar to them, but in the context of their primary purpose, disinfection, the thermal method unequivocally remains the best. Current thermal systems employ temperatures of 78°C to 90°C for 20 to 60 minutes, and have D values for all potentially contaminating microorganisms including bacteria, fungi, viruses, and *Acanthamebae*, and excluding only bacterial spores, when used according to manufacturers' guidelines. *Acanthameba* cysts are particularly resistant to destruction, and thermal disinfection is the only method among those available that is consistently effective.[6]

Thermal disinfection, however, is not ideal. Its major drawback is incompatibility with traditional hard, rigid gas-permeable, and high water content (>50%) soft contact lenses. An electrical power source is generally required as well, making use of heat units impossible among outdoor campers, or inconvenient during international travel, when electrical current adaptors may be necessary. Although soft lenses may be disinfected in these circumstances by exposure to boiling water surrounding the disinfection and storage case, repeated exposure to such temperatures denatures contact lens protein deposits, causing more rapid lens deterioration. Although thermal disinfection units employ significantly lower temperatures with less resultant lens deterioration, a negative effect of some units on lens longevity has been observed. In practical terms, useful lens life may be reduced from two years with nonthermal disinfection methods to one year with thermal methods. Since more frequent lens replacement may be important in reducing the degree of lens deposits and imperfections and thereby the degree of lens contamination and associated ocular infections, decreased lens longevity to periods of one year or even six months by thermal disinfection methods may not be a significant drawback. In either case, heat exposure remains the gold standard for lens disinfection and should be used whenever feasible.

CHEMICAL DISINFECTION

Each of the chemical disinfection techniques has one or more weaknesses regarding disinfection efficacy, but these can be offset by other advantageous characteristics. Each of the major chemical disinfectants, therefore, must be considered individually (Tables I, II, III).

Of the available chemical disinfectants, only hydrogen peroxide has been shown effective against HIV, and its efficacy against bacteria is excellent.[15] Disinfection of some fungi and molds, such as *Aspergillus niger* and *Candida parapsilosis*, and *Acanthameba*, however, is poor for the exposure times recommended by manufacturers.[16] Prolonging exposure time to 3% hydrogen peroxide to six hours or more increases action against such relatively resistant organisms, although the adequacy of such enhanced levels of disinfection remains unproven. Hydrogen peroxide is toxic to the ocular surface and can produce severe corneal burns, so that neutralization is necessary before contact lens insertion. This is accomplished using platinum discs, catalase, or pyruvate neutralizing solutions, or incubation with sterile saline. Since this neutralization step may be time consuming and an inconvenience that could discourage compliance among contact lens wearers, some manufacturers have devised systems that automatically neutralize residual hydrogen peroxide. Although such systems provide a measure of protection against peroxide-induced ocular surface burns, they also preclude prolonged incubation times for better disinfection of resistant organisms.

The practitioner who prefers the advantages of hydrogen peroxide disinfection but desires better disinfection of resistant organisms should be certain to choose a system that allows patient control of the timing of the neutraliza-

TABLE I. Three Percent Hydrogen Peroxide Systems for Contact Lens Disinfection*

Disinfection Solution†	Manufacturer	Neutralizer	Mfr Recommended Duration Soak H_2O_2/ Neutralizer (min)
Aosept	CIBA	Platinum disc	360‡
Lens Plus Oxysept	Allergan	Catalase	10/10
Lensept	CIBA	Catalase	55/5
Mirasept	CooperVision	Sodium pyruvate	10/10
Murine Puresept	Ross Labs	Saline	20/10
Quik-sept	Bausch & Lomb	Saline	20/10
Soft Mate Consept	SOLA-Barnes Hind	Sodium thiosulfate	10/10

* Sources: *PDR for Ophthalmology*, Oradell, NJ, Medical Economics Co, Inc, 1989; *Contact Lenses*, vol 20, no. 6, May 1987; *International Eye Care*, 1986 Reference for Contact Lens Practice, vol 2, no 7, July 1986.

† The list of products is intended to be representative, and not inclusive of all available products.

‡ A single six-hour soak without separate neutralization step.

TABLE II. Chemical Disinfection Systems—Soft Contact Lenses*

Disinfection Solution†	Manufacturer	Active Disinfectants	(%)
Allergan Hydrocare	Allergan	Tris (2-hydroxyethyl) tallow ammonium chloride	0.013
		Thimerosal	0.002
Flex-Care	Alcon	Chlorhexidine gluconate	0.005
		Edetate sodium	0.1
Opti-Free	Alcon	Polyquaternium-1	0.001
		Edetate sodium	0.05
ReNu	Bausch & Lomb	Polyaminopropylbiguanide	0.00005
Soft Mate	Barnes-Hind	Chlorhexidine	0.005
		Edetate sodium	0.1
		Povidone	

* Sources: *PDR for Ophthalmology*, Oradell, NJ, Medical Economics Co, Inc, 1989; *Contact Lenses*, vol 20, no. 6, May 1987; *International Eye Care*, 1986 Reference for Contact Lens Practice, vol 2, no 7, July 1986.

† The list of products is intended to be representative, and not inclusive of all available products.

tion process. Hydrogen peroxide offers significant advantages over all other disinfection techniques, including heat, in its compatibility with all contact lenses. It also exerts a cleaning action on contact lens deposits which may help in prophylaxis of infection. Due to its proven efficacy against HIV, it remains the only alternative to heat disinfection of trial contact lens fitting sets among lens dispensers.

Mercurial disinfectants, primarily represented by thimerosal, and the biguanides, represented by chlorhexidine and polyaminopropylbiguanide, are the other commonly employed chemical disinfectants, particularly for soft contact lenses. None of these agents have been shown to be effective against HIV, HBV, or *Acanthamebae*, and their action against bacteria and fungi is variable. Thimerosal disrupts sulfhydryl groups on bacterial enzymes and thus requires both cell penetration and actively growing organisms. This may explain its slower onset of action, limiting its usefulness in disinfection systems that strive to be as fast acting as possible. Thimerosal, however, also has a prolonged duration of action that is advantageous when lenses are stored in disinfecting solutions for periods longer than overnight. By contrast, chlorhexidine disrupts cell walls and its onset of action is faster but of shorter duration than thimerosal. Since these two agents are complementary in this regard, they are sometimes combined into one disinfection solution.[17] This combination is also preferred, since biguanides alone may have suboptimal action against some bacteria, particularly *Serratia marcescens* and some fungi.[18]

A major drawback of thimerosal is the frequent occurrence of sensitization and hypersensitivity reactions of the ocular surface resulting in keratoconjunctivitis. Although such reactions rarely threaten one's vision, they may occur in up to 20% or more of exposed individuals and necessitate permanent withdrawal of the offending agent.[18] Biguanides such as chlorhexidine less commonly provoke sensitization, but may cause toxic reactions of the conjunctiva and cornea. Polyaminopropylbiguanide, a recently marketed biguanide, is claimed to be less sensitizing than other biguanides and at least equally effective, but clinical experience is thus far limited with this agent.

At first glance, the problem of hypersensitivity reactions and direct toxic effects on the ocular surface might seem to limit the applicability of mercurials and biguanides; the simplicity of the disinfection method, however, requiring only incubation with the disinfection solution for several hours or longer, makes this the most convenient of all disinfection methods, as it eliminates the need for a power source or a neutralization step. To the extent that such simplicity encourages compliance, this method may be preferable to others for some contact lens wearers.

Quaternary ammonium compounds, such as benzalkonium chloride (BAK), Polyquaternium I (Polyquad),

TABLE III. Disinfection, Soaking Solutions, Hard and Gas Permeable (RGP) Contact Lenses*

Disinfection Solution†	Manufacturer	Active Disinfectants	(%)	Lens Compatibility
Gas permeable wetting and soaking solution	Barnes-Hind	Chlorhexidine	0.003	Hard and many RGP
		Thimerosal	0.002	
		EDTA	0.02	
The Boston conditioning solution	Polymer Technology	Chlorhexidine	0.006	Many RGP
		EDTA	0.05	
ONE solution	Allergan	Benzalkonium chloride	0.01	Hard
		EDTA	0.1	
Soquette	Barnes-Hind	Benzalkonium chloride	0.01	Hard
		EDTA	0.2	
Wet-N-Soak	Allergan	Benzalkonium chloride	0.004	Hard and some RGP

* Sources: *PDR for Ophthalmology*, Oradell, NJ, Medical Economics Co, Inc, 1989; *Contact Lenses*, vol 20, no 6, May 1987; *International Eye Care*, 1986 Reference for Contact Lens Practice, vol 2, no 7, July 1986.

† The list of products is intended to be representative, and not inclusive of all available products.

and alkyltriethanolammonium chloride (TEAC), are limited in their effectiveness against microorganisms at concentrations compatible with ocular surface use. They are often combined with divalent cation chelators, such as edetic acid (EDTA), which enhance their antimicrobial effect, but they remain suboptimal in their effect against gram-negative bacteria and some fungi and yeasts, in particular. In addition, their antiviral and anti-*Acanthameba* effectiveness is uncertain.[10,16,19] Since these chemicals also bind to a variable extent to soft contact lenses during disinfection, and then are slowly released from the lens on reinsertion onto the eye, they may exhibit intolerable ocular toxicity, especially with soft contact lenses of high (more than 50%) water content. Their main usefulness is for disinfection of hard and gas permeable contact lenses that do not bind these agents.

CONCLUSIONS

In summary, contact lens-associated corneal infection is a potentially blinding affliction of increasing incidence among the 18 million contact lens wearers in this country. Infection prophylaxis is possible with contact lens cleaning and disinfection procedures. However, even the most effective procedures have not been designed to kill potential pathogens when used incorrectly or not at all. Patient education can go far in promoting proper compliance, but human nature still permits lapses in lens care techniques among the most intelligent and carefully instructed lens wearers. Such lapses are often identified among patients with contact lens-associated ocular infections. The in vitro efficacy of any lens care regimen, therefore, must be considered, as well as the patient's individual life style and needs, in choosing the lens care system best able to prevent infection for that patient.

When compatible with the lenses worn, thermal disinfection is the most effective method against all potential pathogens, but may lead to some decrease in lens longevity, and requires a power source. The patient who must at least occasionally wear his contact lenses when a power source is unavailable, and for whom lens disinfection is thereby not possible, may be less at risk for ocular infection with cold chemical disinfection. Although the latter may be less effective than thermal disinfection, it is more effective than no disinfection at all. Similar considerations should prevail with other patients and circumstances, such as the less compliant patient, and office disinfection of contact lens fitting sets.

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Melville's "Bartleby the Scrivener": A case study

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Herman Melville's "Bartleby the Scrivener," first published in 1853,[1] has become one of the most celebrated and widely anthologized pieces of short fiction in American literature. In addition, because of certain enigmatic aspects to the story, there has been an enormous outpouring of scholarship and criticism on it, amounting to hundreds of articles.[2,3] In brief, the plot deals with a copyist, or scrivener, who obtains employment in a Wall Street law firm in the middle of the 19th century. This quiet and withdrawn man, whom we know only by his first name of Bartleby, is, at the beginning, assiduous in his labor of copying, but after three days he refuses to undertake the correction of his papers. The narrator of the story, an attorney and Bartleby's employer, is shocked, but due to the cool mildness of Bartleby's demeanor, refrains from dismissing him on the spot. The scrivener's interaction with other employees is negligible. Although precise in his work, he gradually refuses more and more tasks. Verbal responses usually consist only of the phrase "I would prefer not to." His work output steadily slows and becomes negligible, while his appearance becomes more and more forlorn. He reveals no pleasure in any task or event, and his appetite wanes during the course of the narrative. From a medical standpoint, the story is one of progressive mental deterioration followed by physical deterioration. In order to maintain his professional reputation, the attorney moves his office away from the vicinity of the recalcitrant scrivener, who is then ordered to prison as a vagrant by the new tenant. There Bartleby refuses food and soon dies.

Part of the "problem" of the story is that we learn everything about Bartleby through the eyes of this attorney-narrator, who is himself sorely vexed and troubled by what has transpired. Bartleby refuses to make any explanation whatsoever about his actions, and, indeed, we are given only one item of information about his past, which comes at the very end of the story. Therefore the attorney and his employee devolve into a situation where their fates are inextricably intertwined and fused. From their predicament, the reader is likewise drawn into mazes of perplexity.

HERMAN MELVILLE'S BACKGROUND

With the first biographies of Melville in the 1920s, it was recognized that he had poured much of his personal experience into the story. Its period of incubation was a "miserable year," as he faced unanimous critical condemnation for the novel *Pierre* and disastrously poor sales.[4] In addition, he had direct knowledge of the legal profession through his uncle Peter Gansevoort, a Wall Street attorney, and he was financially dependent to some extent on his father-in-law, Lemuel Shaw, Chief Justice of the Massachusetts Supreme Court. Furthermore, Melville went through several evidently severe periods of depression during the course of his life.[5]

Two of Melville's close friends suffered mental illnesses similar to Bartleby's. A school chum, Eli James Murdock Fly, worked as a clerk for Peter Gansevoort, and later found a situation with a Mr Edwards "where he has incessant writing from morning to evening." Several years at such tasks, during which he complained of his poverty, rendered him a "confirmed invalid," and Melville acted as his agent "in some small things." George J. Adler, a scholar of German metaphysics whom Melville met on a voy-

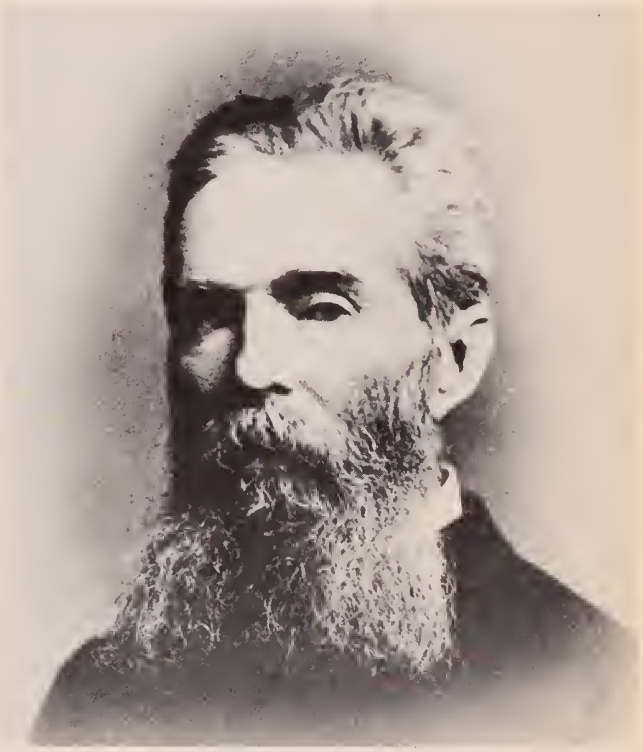

FIGURE 1. Herman Melville (from the Gansevoort-Lansing Collection, Rare Books and Manuscripts Division, The New York Public Library, Astor, Lenox and Tilden Foundations).

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age to England, developed severe agoraphobia, for which he was confined to Bloomingdale Asylum in 1853.[6] It was out of such a matrix of personal experience and close relationships that Melville developed his philosophical and symbolic masterpiece. However, this essay concentrates on the dynamics of the story itself, rather than on Melville's biographical situation.

A summary of the story should provide some instructive lessons concerning the development of diagnostic criteria for mental disease, with practical insights for clinicians.

SUMMARY OF "BARTLEBY THE SCRIVENER"

"I am a rather elderly man," commences the narrator, and he establishes at once that he is "one of those unambitious lawyers who never address a jury, or in any way draw down public applause; but, in the cool tranquillity of a snug retreat, do a snug business among rich men's bonds, and mortgages, and title-deeds."[1] We learn that his offices are on Wall Street, that "All who know me, consider me an eminently *safe* man," through which character he was "not unemployed . . . by the late John Jacob Astor." In short, we are introduced to a professional man: calm, judicious, and respectable in the eyes of his peers. He is at pains to stress that descriptions of himself, his employees, and his office are all indispensable to the story. According to the attorney, his chambers:

> looked upon the white wall of the interior of a spacious sky-light shaft, penetrating the building from top to bottom. This view might have been considered rather tame than otherwise, deficient in what landscape painters call "life." But if so, the view from the other end of my chambers offered, at least, a contrast, if nothing more. In that direction, my windows commanded an unobstructed view of a lofty brick wall, black by age and everlasting shade; which wall required no spy-glass to bring out its lurking beauties, but, for the benefit of all nearsighted spectators, was pushed up to within ten feet of my windowpanes. Owing to the great height of the surrounding buildings, and my chambers being on the second floor, the interval between this wall and mine not a little resembled a huge square cistern.

The attorney's bland tone cannot conceal a locale of extreme bleakness, with the walls becoming a symbol of psychological alienation and claustrophobia.[7,8] Indeed, the motif of burial becomes vital in the rest of the story, and continues as it were to a logical conclusion. It is within this atmosphere of Dickensian gloom that most of the story takes place.

The attorney then proceeds to introduce his employees: Turkey, Nippers, and Ginger Nut and Bartleby. Ginger Nut is the office boy, little more than a beginning apprentice in the trade, and not of great importance to the plot. Turkey is "a short, pursy Englishman" of about 60, whose "face was of a fine, florid hue." We soon learn that his complexion is due to his fondness for beer and ale; that he is, in short, an alcoholic. Nippers is "a whiskered, sallow, and upon the whole, rather piratical-looking young man, of about five-and-twenty." He is ambitious beyond his station in life, and aspires not only to mere copying but "the original drawing up of legal documents." He also suffers from indigestion "betokened in an occasional nervous testiness and grinning irritability, causing the teeth to audibly grind together over mistakes committed in copying." He claims that his writing desk "stopped the circulation in his arms," and is in a recurrent battle to adjust it. Further, the remarkable thing about these two clerks is that Turkey usually has a productive morning, but his afternoon labors are marred by carelessness and errors, while Nippers has an inefficient morning, but improves after lunch. They are a pair of employees who take turns being unproductive and inefficient. Otherwise their work is for each half-day accurate enough to secure their continuing employment. It has convincingly been argued that they are both "types" of melancholics derived from the Burtonian tradition of humoristic psychology.[9] In any event, both clerks are impaired for approximately half of their working day.

FIGURE 2. Wall Street, looking west from Pearl Street, to Broadway, photographed between 1872 and 1880 (from United States History, Local History and Genealogy Division, The New York Public Library, Astor, Lenox and Tilden Foundations).

Due to an increase in business, it becomes necessary for the attorney to hire an additional copyist: "In answer to my advertisement, a motionless young man one morning stood upon my office threshold, the door being open, for it was summer. I can see that figure now—pallidly neat, pitiably respectable, incurably forlorn! It was Bartleby." He is hired at once because his "singularly sedate aspect might operate beneficially upon the flighty temper of Turkey, and the fiery one of Nippers." That is, the attorney calculates that the new scrivener might improve the efficiency of his other employees. Bartleby is assigned to a corner "close up to a small side-window in that part of the room, a window which originally had afforded a lateral view of certain grimy backyards and bricks, but which, owing to subsequent erections, commanded at present no view at all, though it gave some light." Furthermore, Bartleby is placed behind "a high folding screen," which "entirely isolates" him from the sight of the attorney, though not from his voice. With "no view at all" it cannot be surprising that Bartleby and the other scriveners find their prospects as human beings terribly limited; they are enclosed from the world and from one another except for the purposes of command.

For the first three days of his employment, Bartleby does "an extraordinary quantity of writing," but on the third day, the attorney asks him to examine a small paper for accuracy, only to receive the reply: "I would prefer not to." Nearly flying into a rage at this impertinence, but restraining himself, the narrator advances to the screen: "I looked at him steadfastly. His face was leanly composed; his gray eyes dimly calm." Had the scrivener been himself upset, he would have instantly been dismissed from the premises, but Bartleby quietly continues to work at copying. As the lawyer is in a hurry, he summons Nippers to assist him.

From this point on in the story, Bartleby refuses to undertake any work of examining documents, though for some time he labors steadily at his own task of copying. On the next occasion he is asked to examine copy, in the company of the other scriveners this time, the narrator asks him: "*Why* do you refuse?" and the reply is the same: "I would prefer not to." The attorney is once again upset but comments: ". . . there was something about Bartleby that not only strangely disarmed me, but, in a wonderful manner, touched and disconcerted me. I began to reason with him." All his attempts to fathom the reason are similarly repudiated by the scrivener, but it is this very process of reasoning which is typical of the responsible, professional approach to the whole matter. Though the narrator is an attorney rather than a physician, his concentration on clinical details is impressive: "It seemed to me that, while I had been addressing him, he carefully revolved every statement that I made; fully comprehending the meaning." As is often the case, Bartleby's fellow scriveners have thoroughly internalized the business ethos, so they are more ruthless than their employer, urging his immediate dismissal. Ginger Nut, however, suggests, "I think, sir, he's a little *luny*," and it is at this point that the suggestion of insanity enters the story.

The attorney reflects that Bartleby may have a poor diet: he "never eats a dinner, properly speaking; he must be a vegetarian, then, but no; he never eats even vegetables, he eats nothing but ginger nuts." However, he rules out the effects of ginger, since the other men in the office partake of them, with no adverse results.

Soon Bartleby also refuses to undertake even the simplest tasks, such as going to the post office, which leaves the attorney "suffering much from perplexity and distress of mind." Though the scrivener is still content to work at copying, he becomes given to periods of "standing revery behind his screen." The attorney further discovers that Bartleby is actually living in the office, when he happens to stop by on a Sunday morning. Within Bartleby's desk he discovers a few household items, with "crumbs of ginger-nuts and a morsel of cheese." He begins to reflect on the scrivener's severe isolation from the rest of mankind:

> For the first time in my life a feeling of overpowering stinging melancholy seized me... These sad fancyings—chimeras, doubtless, of a sick and silly brain—led on to other and more special thoughts . . . Presentiments of strange discoveries hovered around me. The scrivener's pale form appeared to me laid out, among uncaring strangers, in its shivering winding sheet.

The phrase "strange discoveries" indicates that the entire basis of the attorney's secure and confident world is being called into question, that there is something hollow and deathly at its core.

This theme of social "inferiors" who provoke a re-thinking of fundamental moral issues is common in 19th century literature. In Dickens' "Christmas Carol" Scrooge is given a vision of "Christmas future" in which he foresees his own death, as the uncaring denizens of the "lower depths" pick through his effects, discussing his character. Similarly, in Chekhov's "Ward Six" the patient Gromov, a paranoiac and also a former copyist, rudely challenges the comfortable world view of Dr Ragin, with fatal consequences for the doctor.

Melville's narrator carefully reviews the "case" almost in the manner of a physician: the scrivener "never spoke but to answer," is given to silence, never reads, never visits any other place, never socializes with others, has never spoken about his family; he surmises that Bartleby is "the victim of an innate and incurable disorder." The next morning Bartleby "prefers not" to give any details about his origins, and has poor eye contact: "He did not look at me while I spoke, but kept his glance fixed upon my bust of Cicero." Then there is an amusing episode when the other scriveners enter the scene, and it transpires that they have all started "involuntarily using this word 'prefer' upon all sorts of not exactly suitable occasions." In short they are succumbing to a form of echolalia, and the attorney apprehensively asks himself, ". . . what further and deeper aberration might it not yet produce?"

It is at this point that Bartleby declares that he had decided upon doing no more writing. Asked the reason, he replies, "Do you not see the reason for yourself?" Once again the attorney recalls that he "looked steadfastly at him, and perceived that his eyes looked dull and glazed." Surmising that his employee has impaired his vision "in copying by his dim window for the first few weeks of his stay," he once again decides to leave Bartleby alone. After "added days" go by, the attorney imagines that Bartleby's eyes have improved, but the scrivener still refuses to do any work, at which point the attorney says that Bartleby must leave the office within six days; at the expiration of that time, he "prefers not" to leave. Meditating on his predicament while walking in the street, the attorney suddenly imagines that perfect strangers are talking about him. He thus begins to show signs of psychopathology, in this instance what in modern terms is known as an "idea of reference."

Finally, the narrator is drawn into "a state of nervous resentment," because he both wishes to help Bartleby yet is repelled by him. Consequently he has feelings of guilt about the scrivener and his situation comparable to a physician's feelings of mixed compassion and irritation in dealing with a depressed patient. More and more the scrivener is given to "profoundest dead-wall reveries," which in Melville's time would have been called a "brown study": "A state of mental abstraction or musing . . . esp. an idle or purposeless reverie."[10] Today this would be considered as a kind of imploded fugue, in which the "immediately precipitating factor" is usually "a desire for escape from some intolerable situation."[11] Instead of physically traveling to some new location, Bartleby remains resolutely and helplessly rooted in the same place. In any event, this is clearly what we would today call psychomotor retardation and loss of energy.

The narrator's attorney acquaintances begin to marvel at this person who silently inhabits his office, which determines him to actually move to another location, abandoning Bartleby, who, however, remains in the hallway to the consternation of the new tenants. The narrator offers him money, but "it dropped upon the floor" when slipped into the scrivener's hand. Even in his new office, "for a day or two I kept the door locked, and started at every footfall in the passage," suggesting the further development of guilt in the narrator. He is summoned back to his old office at the request of the new tenants, but Bartleby refuses his offer of some new employment, or of traveling: "I like to be stationary. But I am not particular." He refuses even to go home with the attorney until something may be arranged: ". . . at present I would prefer not to make any change at all," whereupon the attorney flees: "effectually dodging every one by the suddenness and rapidity of my flight, rushed from the building, ran up Wall Street towards Broadway, and, jumping into the first omnibus, was soon removed from pursuit." The odd thing is that in all probability, no one was chasing him—an instance, then, of panic flight arising from fear and guilt.

The attorney himself takes a vacation for a few days, and learns upon returning that Bartleby has been removed to the city jail "as a vagrant." His first response—"At first I was indignant, but, at last, almost approved"—is succeeded by a desire to visit Bartleby in jail, where he finds him standing and staring at a wall. Upon hearing his name called Bartleby replies: 'I know you,' he said, without looking round—; 'and I want nothing to say to you.' " The attorney tries to distract him by pointing to the trees and the grass growing in the yard of the prison, but Bartleby responds similarly: "I know where I am." Though the attorney arranges for Bartleby to have better food in jail, the scrivener refuses to eat, which continues the pattern of poor appetite, and now significant weight loss. At last he is found thus:

> Strangely huddled at the base of the wall, his knees drawn up, and lying on his side, his head touching the cold stones, I saw the wasted Bartleby. But nothing stirred. I paused; then went close up to him; stooped over, and saw that his dim eyes were open; otherwise he seemed profoundly asleep. Something prompted me to touch him. I felt his hand, when a tingling shiver ran up my arm and down my spine to my feet.

Bartleby had committed suicide by starving himself to death. Curled up in a fetal position, still within the walls he could never escape and which were his final resting place, his death becomes known only through a last human touch, which is also the first he experiences in the story, so complete is his isolation.

Still puzzled months later about the case, the attorney describes hearing "a vague report" that "Bartleby had been a subordinate clerk in the Dead Letter Office in Washington, from which he had been suddenly removed by a change in the administration. When I think over this rumor, hardly can I express the emotions which seize me. Dead letters! does it sound like dead men?" The attorney, who commences his narrative with an almost priggish self-satisfaction, concludes with a moving cry: "Ah, Bartleby! Ah, humanity!" Though he cannot even yet comprehend his clerk's sad plight, he recognizes that it is common to mankind in general. He has perceived Bartleby to be "a bit of wreck in the mid-Atlantic," but there are many in the great modern metropolis whose fate is identical.

PAST LITERARY VIEWS OF "BARTLEBY"

The mystery of the story has largely prevailed until today, and has provoked several scholarly controversies. The crux is, as Bluestone[12] put it: "Melville never tells us the cause of Bartleby's malaise." Many authors have strongly urged the importance of the story's stark and gloomy environment,[7,8] while others have emphasized Bartleby's innate character, usually as a kind of Gandhian pacifist.[13] Some have viewed the story as a parable of the deadening aspects of commercial society.[14] The environment versus character approaches parallel the reactive versus endogenous concepts of etiology. Indeed, this story is an excellent illustration of how the two are intimately intertwined. The attorney believes that Bartleby has an "organic" illness, but he remains unaware of any possible origin for this in the office environment, not only in Bartleby, but in his other two clerks who effectively function in their jobs only a half-day each. While the attorney closely observes Bartleby and tries to assist him according to his lights, including the hope that the whole problem may be merely some sort of "eye-strain," he remains mystified. Marcus[15] has suggested that Bartleby is a psychological "double" for the attorney, partially representing a "subliminal death drive within him." In any event, it is clear that the relationship between the attorney and his clerk takes on overtones of a fateful, decisive encounter in which deep issues of human morality are explored.

In a letter to his friend Nathaniel Hawthorne, Melville proclaimed: "With no son of man do I stand upon any etiquette or ceremony, except the Christian ones of charity and honesty."[16] The attorney tries to hold to such a standard also: "Mere self-interest, then, if no better motive can be enlisted, should, especially with high-tempered men, prompt all beings to charity and philanthropy." The irony is that unlike his creator, the attorney can take this only so far, and no further. As Stein[17] has argued, the sense of charity as social love which we find in the New Testament has become a dead letter in modern society. Indeed, it is the very conventions of society itself which prevent kindness from prevailing. "Mere self-interest" cannot suffice to sustain life.

PAST PSYCHOLOGICAL PERSPECTIVES ON "BARTLEBY"

Starting in the late 1940s, psychologically minded critics began to use the term "schizophrenic" in trying to explain Bartleby's character.[18,19] For the last 30 years this diagnosis has prevailed.[20-22] A recent study by a psychiatrist linked Bartleby with the patients described in Kahlbaum's *Catatonia*.[23] The pertinence of Burton's *Anatomy of Melancholy* has also been suggested, because it was a classic literary discussion of depressive-type maladies, and we know that Melville had a copy in his library.[9] In addition, Bartleby has been viewed as agoraphobic,[24], autistic,[25,26] and as having some form of eating disorder.[27,28] Another study by Henry Murray,[29] a physician, reviewed the welter of emergent diagnoses and proposed a unique solution:

> Bartleby is unprecedented, an invention of Melville's creative spirit, the author's gift to psychology, a mythic figure who deserves a category in his own name. I see the scrivener as a composite of several very human dispositions . . . I will end by crediting Mr Melville with the discovery of the Bartleby complex.

However, short of naming an entirely new complex after him, it may be useful to reconsider the powerfully realistic details of this story, and a symbolic content so minutely elaborated as to resemble an actual "case history," to see if current diagnostic criteria can be of any help in comprehending the scrivener and his plight. Of course, it is important to bear in mind that even though Melville based his story on real people he knew well, this is a work of fiction, and thus may not easily fit preconceived categorizations.

From a psychological standpoint, the story presents a number of instructive dilemmas, especially concerning problems of diagnosis. These are closely connected to answering the persistent question: What is the cause of Bartleby's problem? For the past 35 years, Bartleby has been viewed by the critics as a schizophrenic. While Bartleby certainly demonstrates a number of symptoms in common with the patients Kahlbaum discussed in his classic work on catatonia, such a diagnosis would not be tenable according to our present diagnostic criteria. Most significant, he shows no evidence of delusions, hallucinations, or other severe thought impairments which are practically a *sine qua non* of schizophrenia today. Indeed, he repeatedly asserts his cognitive abilities: "I know where I am," "I know who you are," and so on. It was not only literary critics who were drawn into making a diagnosis of schizophrenia; physicians themselves during the same time frequently resorted to this diagnosis for their patients, and the great decrease in the incidence of such diagnoses is surely one of the most dramatic changes in psychiatry during the last ten to 15 years,[30] though a significant number of psychiatrists have continued to use the term in an unsystematic fashion.[31] In addition, we may note that the time frame of Melville's story is in the range of at most several weeks, instead of the long term trajectory of schizophrenia.

DSM-III-R DIAGNOSIS

The revised *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III-R)[32] is the most recent edition of the diagnostic "bible" of psychiatry. This text provides rigorous criteria for psychiatric diagnoses of acute and chronic psychopathology. As in the previous edition, psychiatric problems can be categorized on five axes (ie, I–V. Axis I is used for clinical syndromes and "V Codes," which are "conditions not attributable to a mental disorder that are a focus of attention on treatment" (eg, academic and marital problems). Axis II is used for developmental and personality disorders. Physical disorders and conditions are listed on Axis III, while Axis IV is available for listing the severity of psychosocial stressors, ranging from one (none) to six (catastrophic). The Global Assessment of Functioning Scale (GAF) is included on Axis V. On this axis, the clinician has the option of rating patients' functioning from one (persistent danger of hurting self) to 90 (absent or minimal symptoms). No previous writers have applied DSM-III-R criteria to diagnose Bartleby's condition.

Using DSM-III-R criteria, it can be concluded that Bartleby's Axis I diagnosis was major depression. While retaining his orientation to time, place, and person and other cognitive abilities, he gradually slips into moods of silence, poor appetite, psychomotor retardation, loss of energy, loss of interest and pleasure in usual activities, slowed thinking, decreased appetite, and ultimately suicide by starvation at the end. Melville provides no evidence as to Bartleby's sleep patterns, nor does Bartleby overtly express feelings of self-reproach or worthlessness, but otherwise he presents all the symptoms of major depression.[32] Agoraphobia is difficult to accept also, since Bartleby has no evident fear of leaving the office; he only "prefers not to," an assertion of will power repeated time and time again.

There is some possibility that Bartleby had an Axis II Schizoid Personality Disorder, as he had no apparent friends, did not socialize, did not demonstrate a variety of emotions, etc. But at the same time it must be acknowledged that we have no evidence of his life while an employee of the Dead Letter Office. That is, there is insufficient information regarding the onset or history of his psychiatric dysfunction. The main features of Bartleby's physical condition are pronounced bodily thinness and dullness of his eyes (Axis III). On Axis IV, psychological stressors in Bartleby's life might be rated a three (moderate), considering his previous loss of employment, overwork in his new position, homelessness, and social isolation. And finally, on Axis V, Bartleby's functioning early in the story might be rated high (ie, 70s or 80s), deteriorating to single digit ratings by the end of the story with his ultimate suicide.

Of course, we are dealing here with a fictional character, the creation of Melville's imagination, rather than with a real person; therefore any attempt to provide a conclusive diagnosis must be speculative. From a review of the critical literature, it is apparent that each generation brings its own preoccupations to the interpretation of this story, and that each individual reader will find it a considerable challenge no matter what conceptual tools he employs. That Melville has provided us with such a rich and problematic narrative is a tribute to the enduring power of his creative achievement.

CONCLUSIONS

The contemporary pertinence of this story is suggested by a recent newspaper account of a homeless vagrant in New York City who was once a promising law school graduate but who, upon failing his bar examination, gravitated toward a life on the street where he repeatedly refused treatment or assistance even from close friends.[33] Thus the social conditions and emotional problems which Herman Melville described in 1853 are very much a part of our current social and clinical situation.

From a current clinical perspective some lessons can be learned from Bartleby. The attorney's attempt to understand and aid his clerk parallels that of the physician who must treat a "difficult" patient, and many of the special problems of that therapeutic situation are illuminated in Melville's story. The attorney well illustrates the social role of the professional, who holds to "higher standards" than society in general, yet this same position places certain burdens on him which can be quite challenging to face, let alone solve. Certainly the story urges the importance of searching beyond surface "symptoms" to the subtle ways in which patients are connected to larger problems of society such as alienation and poverty. Furthermore, like the physician, the attorney wishes to serve a therapeutic purpose to Bartleby, who is an unwilling patient. Also, like the physician, in the end the attorney must accept that his patient has the final say in his own decision to live or die.

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History of medicine archives in New York State

KATHLEEN S. ANDERSEN, DANIEL M. FOX

Because so much history of medicine has been made in New York for three centuries, the state's institutions are rich repositories of primary sources; of, for example, letters, diaries, patient records, official documents, photographs, and equipment. These primary sources have been used by historians of medicine from all over the United States and from abroad as the basis for articles and monographs.

This description of archival resources in the history of medicine in New York State is a joint effort of the author-compilers and the medical archives members of the Archivists Round Table of Metropolitan New York. The work was undertaken as one of several medical activities in conjunction with New York Archives Week 1989.

This description of archival resources is intended both to guide and inspire. It should guide physicians and others interested in particular questions about the history of medicine to new evidence. Some of the evidence bears on important local events; for instance, what happened in the past in particular medical societies or hospitals. But the evidence in the state's archives can also help scholars to explore events of national and international significance. Such events include research findings that have led to advances in prevention and treatment, the founding and maturing of great institutions to provide education and care, and the participation of New Yorkers in important decisions about the course of medicine and science.

These archival resources should also inspire physicians and other interested persons to explore the history of medicine in New York State. The records of great events and splendid careers are close at hand. The archivists who manage these resources are eager to make them available to people who want to write history, to experience the past at first hand, or to reclaim a part of their lives or the collective life of an institution with which they are closely associated.

The descriptions that follow can be read in several ways. Some readers will skim them looking for records in which they are particularly interested. Others will scan them looking for names that spark particular interests. Still others will read them as one reads a catalog of a museum or an auction house—as an exercise to stimulate the imagination, a prelude to further action.

The materials in the archives described below are vital connections to our past. They are better, because truer, than memory. They are vital to the future of medicine in New York because they are the surviving records of the achievements, the failures, the joy and the anger of those who preceded us in making history.

The archives are described in alphabetical order, as follows:

1. Archives of Albany Medical Center
2. Bellevue Hospital Center Archives
3. Beth Israel Medical Center Archives
4. The Brooklyn Hospital–Caledonian Hospital Archive
5. Columbia University
   Augustus C. Long Health Sciences Library
6. Cornell University
   Department of Manuscripts and University Archives
   Olin Library
7. Archive of the Long Island Jewish Medical Center
8. Medical Society of the State of New York Archives
9. Memorial Sloan-Kettering Cancer Center Archives
10. Montefiore Medical Center Archives
11. The Archives of the Mount Sinai Medical Center
12. Special Collections
    New York Academy of Medicine
13. New York City Municipal Archives
14. Medical Archives, New York Hospital-Cornell University Medical College
15. New York Hospital-Cornell Psychiatry Department
    Archives of Psychiatry
16. New York Medical College Archive
17. Russell S. Hibbs Memorial Library
    New York Orthopedic Hospital
18. The New York Public Library
    Rare Books and Manuscripts Division
19. New York State Archives
20. New York State Library
21. New York University College of Dentistry Archives
22. New York University Medical Center Archives

From the Center for Assessing Health Services, State University of New York at Stony Brook, Stony Brook, NY.

Address correspondence to Ms Andersen, Associate Director, Center for Assessing Health Services, State University of New York Health Science Center at Stony Brook, Stony Brook, NY 11794-8400.

23. The Rockefeller Archive Center
24. The Medical Research Library
    Health Science Center
    State University of New York at Brooklyn
25. Robert L. Brown History of Medicine Collection
    Health Sciences Library
    State University of New York at Buffalo
26. Slide Archive of Historical Medical Photographs
    Health Sciences Center
    State University of New York at Stony Brook
27. State University of New York Health Science
    Center at Syracuse
    Library Archives
28. Museum and Library of the Suffolk Academy of
    Medicine
29. Special Collections
    Milbank Memorial Library
    Teachers College
    Columbia University
30. Edward G. Miner Library
    History of Medicine Section
    University of Rochester

1. ARCHIVES OF ALBANY MEDICAL CENTER

47 New Scotland Ave
Albany, NY 12208
(518) 445-5197
(518) 445-5810

Inquiries to	Rue Moore, Canon
Access	Open to anyone
Hours	Mon–Fri: 8:00 AM–11:30 AM Appointment preferred Telephone reference service is limited; letter preferred
Finding aids	No published finding aids
Photocopy	By staff, at cost

Papers of the faculty and institutional records of Albany Medical College, 1839–present. Records of Albany Medical Center Hospital 1849–present, including minutes of the Hospital Board of Governors. Records of the Albany Medical College also include scrapbooks, alumni files, and student theses, 1839 to 1891 (on microfiche). Photographic collection (1½ file drawers). A few oral histories, a few paintings, and a collection of old medical instruments and equipment.

2. BELLEVUE HOSPITAL CENTER ARCHIVES

467 1st Avenue (27th St)
Rooms C & D 740
New York, NY 10016
(212) 561-3920

Inquiries to	Lorinda Klein, Assistant Director, Public Affairs
Access	Open to anyone
Hours	Mon–Fri: 10:00 AM–6:00 PM By appointment only Telephone reference service available
Finding aids	No published finding aids or on-line systems
Photocopy	Staff makes copies, no charge.

Centerpiece of the archives is the Board of Managers collection, which includes documents and photographs from the Bellevue Nursing School (1872–1972), the first nursing school in this country based on Florence Nightingale's precepts. Medical board minutes (1848–present), board minutes of Bellevue and allied hospitals (1901–1929), old medical volumes, and pathological reports of the medical examiner's office (1904–present). Numerous photographs and some lantern slides of the buildings, scenes from surgery, and other hospital activities. A few instruments and pieces of medical equipment. Original documents date from 1848 to the present; photocopies of documents from as early as 1798. Most of the collection is from 1870 to 1935.

3. BETH ISRAEL MEDICAL CENTER ARCHIVES

First Avenue at 16th St
New York, NY 10003
(212) 598-6007

Inquiries to	Deborah Tadevich, Archivist
Access	Open to the public
Hours	Mon, Wed, Fri: 9:30 AM–3:30 PM Appointment required for researchers outside the institution Telephone reference service available
Finding aids	No catalog, but Ms Tadevich is available to assist in finding items.
Photocopy	Staff makes copies, no charge.

Institutional records and minutes of the Board of Directors from 1889 to the present. Minutes from 1889 to 1891 are in Yiddish. Records of the nursing school (1904–1920s). Reports of medical research, including physicians' papers (1950s–present). A few recent oral histories. Records of the Bernstein Institute for Alcohol and Drug Dependency, the largest substance abuse treatment center in the world (1964–present). Newspaper clippings from 1907 to 1915, which include descriptions of early surgical methods in New York City. Letters from physicians in the field in World War I to the hospital director. Extensive photographic and archival material documenting the institution's involvement in the implementation of the Medicare and Medicaid programs. Large collection of photographs (1900 to 1950s and 1960s).

4. THE BROOKLYN HOSPITAL-CALEDONIAN HOSPITAL ARCHIVE

121 DeKalb Ave
Brooklyn, NY 11201
(718) 403-6943 or 6944

Inquiries to	Roy Brayton, Archivist
Access	Open to hospital staff and qualified researchers
Hours	Mon–Fri: 9:00 AM–5:00 PM By appointment only Telephone reference service available
Finding aids	Some materials in RLIN; catalog available
Photocopy	Staff makes copies of some material.

Records of the Brooklyn Hospital-Caledonian Hospital and the institutions absorbed by it, including Board of Trustees minutes, correspondence, annual reports, and other items of historical and antiquarian interest (1845–present). Fairly complete record of The Brooklyn Hospital from 1845; most complete record from 1986 to the present. Collection includes records of the Brooklyn Thoracic Hospital (1881–1956), providing a good source of information about the history of a tuberculosis hospital. Photographs.

Size: 75 linear feet.

5. Augustus C. Long Health Sciences Library of Columbia University

701 West 168th Street
New York, NY 10032
(212) 305-7931

Inquiries to	Barbara Paulson, Librarian
Access	Open to qualified researchers
Hours	Mon–Fri: 9:00 AM–5:00 PM Appointment is strongly recommended Telephone reference service available
Finding aids	No published finding aids; some in-house aids
Photocopy	Yes, depending on condition of the materials to be copied; staff makes copies for a $5.00 service charge plus 50¢/page.

The archives are part of the Health Sciences Library and include materials on the history of medicine and the history of Columbia-Presbyterian Medical Center and affiliated institutions (1770–present, majority of the materials from 1920 to 1965). Collection includes institutional documents, manuscripts and private papers, photographs and films, instruments and equipment. The large rare book collection is especially useful for the study of the history of medicine in the 15th–19th centuries and for study of medical illustration.

Size: archival materials, 50 linear feet; manuscripts, 300 linear feet; rare books, 15,000 volumes.

6. Department of Manuscripts and University Archives Olin Library of Cornell University

101 Olin Library
Cornell University
Ithaca, NY 14853
(607) 255-3530

Inquiries to	Thomas Hickerson, Assistant Director
Access	Open to the public
Hours	Mon–Fri: 8:00 AM–5:00 PM; Sat: 9:00 AM–1:00 PM Telephone reference service available
Finding aids	In addition to other published finding aids, there are documentation newsletters published by the archive that describe the various collections, including those in medicine and health care. Collections are included in RLIN.
Photocopy	Staff makes copies. Charges vary depending on the type of document and number of copies requested.

Included in the archives are 100 collections related to the history of medicine, hospitals, health planning, and nursing. The archives include the private papers of physicians in New York State and the papers of a number of county medical societies and the Medical Society of the State of New York.

7. Archive of the Long Island Jewish Medical Center

Administration Building
400 Lakeville Rd
New Hyde Park, NY 11042
(718) 470-8659

Inquiries to	Silvia Bakst, Director of Archives
Access	Open to qualified researchers
Hours	Tues: 9:00 AM–5:00 PM By appointment only No telephone reference service
Finding aids	No finding aids available
Photocopy	Staff makes copies, charge varies.

Material documenting the planning, founding, and history of the Long Island Jewish Medical Center (founded 1954) and the Schneider Children's Hospital (1971). Records of Hillside Hospital (1932), LIJ's psychiatric division, including planning documents from the 1920s. Papers of Dr Gene Rosenfeld, first administrator. Institutional records and press clippings from 1950 to the present. A few manuscripts or private papers. Films documenting the construction of the facility and dedication ceremonies. Newspaper clippings and photographs of major news events, personalities, and activities from 1954 to the present. About 60 oral histories of members of the board of directors, chairs of the clinical departments, other physicians, and staff.

8. Medical Society of the State of New York

420 Lakeville Rd
Lake Success, NY 11042
(516) 488-6100

Inquiries to	Ella Abney, Librarian
Access	Open to qualified researchers approved by the President, Chairman of the Board of Trustees, or the Executive Vice-President of the Society
Hours	See above entry for the Department of Manuscripts and University Archives, Olin Library of Cornell University
Finding aids	"
Photocopy	"

The older archives of the Medical Society of the State of New York are on deposit at the Olin Library of Cornell University in Ithaca, New York. These archives include minutes of meetings and agendas of the Council (1934–1973), records of annual meetings of the House of Delegates (1931–1971), minutes of meetings of Trustees (1941–1965), President's files (correspondence of Norman S. Moore, MD, 1960–1961), membership records (1945–1959).

Publications include the *New York State Journal of Medicine*, 1910–1972, *Transactions of the Medical Society of the State of New York*, 1807–1903, *The Reflex*, 1966–1967, newsletters, journals, and other printed records, subject files, committee files, biographical sketches, and photographs. Post-1973 materials are on file at the Society's headquarters in Lake Success, New York. Complete holdings for the *Transactions of the Medical Society of the State of New York* and for the *New York State Journal of Medicine* are housed in the Society's Albion O. Bernstein Library at Lake Success.

9. Memorial Sloan-Kettering Cancer Center Archives

1275 York Ave
New York, NY 10021
(Entrance at 430 East 67th St)
(212) 639-8487

Inquiries to	Jeanne Becker, Director of Library Services
Access	Open to all qualified researchers; access to some records is restricted
Hours	Mon–Fri: 9:00 AM–5:00 PM

	By appointment only. Records are located at the Rockefeller Archive Center, North Tarrytown, NY. Researchers must contact Ms Becker for permission to access the Archives at the Rockefeller Archive Center. Telephone reference service available
Finding aids	No published finding aids or on-line systems
Photocopy	Available upon request. Library staff or Rockefeller Archive Center staff make copies at charge of 10¢ per copy (library) or 20¢ per copy (Rockefeller Archive Center).

The collection includes the records of Memorial Sloan-Kettering Cancer Center, Sloan-Kettering Institute, and Memorial Hospital for Cancer and Allied Diseases (1884–1989). Materials from the 1940s and 1950s predominate. In addition to institutional records, the collection includes manuscripts or private papers, photographs, rare books, oral history, medical instruments, and equipment.

Size: 455 cubic feet.

10. MONTEFIORE MEDICAL CENTER ARCHIVES

111 East 210th St
Bronx, NY 10467
(212) 920-6149

Inquiries to	May Manheimer, Archivist
Access	Open to qualified researchers
Hours	Mon–Tues: 9:00 AM–5:00 PM By appointment only Telephone reference service available
Finding aids	No published finding aids; catalog is being prepared
Photocopy	Researchers may make copies on library's photocopy machine at charge of 10¢ per page.

Institutional records of the Montefiore Medical Center and affiliated institutions. Most valuable period is 1884–1950. Patient records (1917–present), which include detailed information on the background and behavior of patients in the Home for Chronic Invalids. Minutes of the Board of Trustees and its Executive Committee (1884–present). Annual reports from 1884 to 1934; intermittent reports in subsequent years. Minutes for the medical board (1893–1918). Newsletter, *The Montefiore Echo*, edited by patients from 1915 to 1928. Autopsy reports from 1913. Professional papers published by staff (1917–present). Photographs from 1889 to present. Tape recorded interviews with physicians, administrators, nurses, patients, and social workers. Newspaper clippings.

Size: archival materials, 234 linear feet; photographs, 73 linear feet.

11. THE ARCHIVES OF THE MOUNT SINAI MEDICAL CENTER

Box 1013
Mount Sinai Medical Center
1 Gustave L. Levy Pl
New York, NY 10029-6574
(212) 241-7239

Inquiries to	Barbara Niss, Archivist
Access	Open to the public; restricted access to certain collections
Hours	Mon–Fri: 9:00 AM–5:00 PM By appointment only Telephone reference service available
Finding aids	No published or on-line aids; in-house catalog and guides available
Photocopy	Staff makes copies at charge of 10¢ per page.

Administrative and historical records of the Mount Sinai Hospital, Mount Sinai School of Medicine of the City University of New York, Mount Sinai School of Nursing, Neustadter Convalescent Center, and the Mount Sinai Medical Center from 1852 to the present (most materials from 1950 to present). Private papers: Charlotte Friend, PhD; Hans Popper, MD, PhD, and Horace Hodes, MD. Films and photographs. Instruments and equipment that were developed either at Mount Sinai or by Mount Sinai staff. Large oral history collection started in the 1960s containing information particularly rich for the study of the graduate medical education experience in the early decades of this century, the description of Crohn's disease, and the history of surgery at Mount Sinai.

Size: 450 linear feet.

12. SPECIAL COLLECTIONS, NEW YORK ACADEMY OF MEDICINE

2 East 103rd St
New York, NY 10029
(212) 876-8200

Inquiries to	Florie Berger, Head, Special Collections
Access	Open to qualified researchers
Hours	Mon–Fri, 9:00 AM–5:00 PM By appointment only Telephone reference service available
Finding aids	*Subject Catalog of the New York Academy of Medicine* (G. K. Hall: 1969, Supplement 1974); *Author Catalog of the New York Academy of Medicine* (G. K. Hall: 1969, Supplement 1974); *Illustration Catalog of the New York Academy of Medicine* (G. K. Hall: 1976).
Photocopy	Copy service is available. Hours and fees vary. Call for specific information.

Records of the New York Academy of Medicine and other institutions and societies (1847–present), including the Medical Society of the County of New York, the Health and Hospital Planning Council of Southern New York, the American Hungarian Medical Association, and the Rudolf Virchow Medical Society of the City of New York. Late 19th and early 20th century materials predominate. The collection also includes manuscripts or private papers of individuals, including the Michael M. Davis Collection, and also photographs.

Size: 2,388 linear feet.

13. NEW YORK CITY MUNICIPAL ARCHIVES

31 Chambers St
New York, NY 10007
(212) 566-5292

Inquiries to	Kenneth Cobb, Deputy Director
Access	Open to the public
Hours	Mon–Fri, 9:00 AM–4:30 PM Limited telephone reference service available
Finding aids	No published finding aids or on-line systems but finding aids are available.
Photocopy	Reader makes copies at charge of 15¢ per page.

Collection consists of municipal government records of New

York City. Those pertinent to the history of medicine and health care include the Almshouse Collection (mostly late 19th century) and the minutes of the Board of Health. The Almshouse Collection contains the records of institutions on Blackwell's Island (now Roosevelt Island)—detailed admission, discharge, census, and housekeeping records (beginning 1758) for the almshouse, city home, and hospitals located on Blackwell's Island. Health Department records include the minutes of the Board of Health from 1732 to 1960s. Mayors' papers contain all correspondence between mayor's office and other municipal agencies. Photographs from the Department of Public Charities/Department of Hospitals (c. 1900). Photographs from the Health Department have not yet been cataloged. Films from television station WNYC.

Size: 1,300 cubic feet.

14. MEDICAL ARCHIVES, NEW YORK HOSPITAL-CORNELL MEDICAL CENTER

1300 York Ave
New York, NY 10021
(212) 746-6072

Inquiries to	Adele A. Lerner, Archivist
Access	Open to qualified researchers; access to certain collections is restricted.
Hours	Mon–Fri, 9:00 AM–5:00 PM Prior phone call requested Telephone reference service available for short questions. Usually a written request is needed as follow-up to a telephone request.
Finding aids	*An Introduction to the Medical Archives, New York Hospital-Cornell Medical Center* (November 1976, Adele A. Lerner). The archives are also listed in the Historical Documents Inventory and many finding aids and databases, including RLIN.
Photocopy	Available on premises.

The Archives of the New York Hospital-Cornell Medical Center is the repository for the official records of the following institutions and associations: The New York Hospital-Cornell Medical Center, 1927–present; The Society of the New York Hospital, 1771–present; Cornell University Medical College, 1898–present; Cornell University Graduate School of Medical Sciences, 1952–present; Cornell University-New York Hospital School of Nursing, 1877–1979; The Society of the Lying-In Hospital of the City of New York, 1799–present; Manhattan Maternity and Dispensary, 1905–1939; Nursery for the Children of Poor Women and Nursery and Child's Hospital, 1854–1910; New York Infant Asylum, 1865–1910; New York Nursery and Child's Hospital, 1910–1947; New York Asylum for Lying-In Women, 1823–1899; The American Medical Women's Association, 1915–present; The Women's Medical Association of New York City, 1902–present.

In addition, the Archives collects the personal papers of people of note connected with the Medical Center, such as Drs George N. Papanicolaou; Vincent du Vigneaud, the 1955 Nobel Laureate in Chemistry; Connie M. Guion; George J. Heuer; and Walsh McDermott.

The approximately 4,600 linear feet of records and manuscript collections date back to 1771. The Archives also has a photograph and print collection with over 20,000 items, architectural plans, films and tapes, oral histories, and over 500 antique medical instruments, many from the Reichert Collection of Diagnostic Instruments. The records of the Archives include medical case histories from the New York Hospital, 1808–1932 (192 linear feet), and the Lying-in Hospital, 1891–1932 (103 linear feet), and other patient related records.

15. NEW YORK HOSPITAL-CORNELL PSYCHIATRY DEPARTMENT, ARCHIVES OF PSYCHIATRY, HISTORY OF PSYCHIATRY SECTION

525 East 68th St
New York, NY 10021
(212) 746-3726

Inquiries to	Dr Eric T. Carlson, Director
Access	Open to qualified researchers
Hours	By appointment only Telephone reference service available for brief inquiries
Finding aids	Not published but do have finding aids on site
Photocopy	Staff makes copies at cost.

Manuscripts relating to the history of American psychiatry, currently with the most extensive holdings in child psychiatry, the development of the mental health movement, and legal psychiatry. The Archives is the official depository for the American College of Psychiatrists, American Psychoanalytic Association, American Psychopathological Association, Group for the Advancement of Psychiatry, New York Psychiatric Society, and a number of other organizations, in addition to two major foundations based in New York City whose special interest is mental health: the Ittleson Foundation and the van Ameringen Foundation. Papers of notable psychiatrists include those of Marion E. Kenworthy, David M. Levy, Frankwood E. Williams, and the English child psychiatrist Donald W. Winnicott, in addition to audiotapes of seminars given by Erich Fromm and papers relating to Harry Stack Sullivan. The origin and growth of the mental health movement is documented through the papers of its founder, Clifford W. Beers, the National Committee for Mental Hygiene, and the World Federation for Mental Health: U.S. Committee, Inc. Prints and photographs portray numerous medical people who influenced the development of psychiatry and scenes illustrating various aspects of this broad field. There are also papers of a few 19th century English psychiatric patients and miscellaneous papers of the 18th and 19th centuries. The holdings date from 1708 to the present, with the vast majority devoted to the 20th century.

Size: 61 collections occupying nearly 400 cubic feet.

16. NEW YORK MEDICAL COLLEGE ARCHIVE

Medical Sciences Library
Basic Sciences Building
Valhalla, NY 10595
(914) 993-4209

Inquiries to	Judy Myers, Assistant Director
Access	Open to qualified researchers
Hours	Mon–Thurs: 8:30 AM–11:00 AM, Fri: 8:30 AM–8:00 PM, Sat: 9:00 AM–5:00 PM, Sun: 2:00 PM–10:00 PM (shorter hours during summer; open only until 8:00 PM, closed on Sundays) By appointment only No telephone reference service. (Callers are referred to the New York Academy of Medicine.)
Finding aids	No published finding aids; all materials are cataloged and accessible through the OCLC network.
Photocopy	Reader makes copies at charge of 10¢ per page.

Early history of the New York Medical College, established in 1860 as the first homeopathic medical college in New York City. Collection includes full runs of all institutional documents (yearbooks, catalogs, announcements), minutes and ledgers of the Board of Trustees, a few manuscripts, photographs, and a good collection of journals on homeopathic medicine. Materials date from the 1860s to the present. Original collection of the College's library, which contains early texts on homeopathic medicine.

17. RUSSELL A. HIBBS MEMORIAL LIBRARY OF THE NEW YORK ORTHOPEDIC HOSPITAL

Columbia-Presbyterian Medical Center
622 West 168th St
New York, NY 10032
(212) 305-3294

Inquiries to	Jack E. Termine, Medical Librarian-Archivist
Access	Open to staff and qualified researchers
Hours	Mon–Fri: 8:30 AM–7:00 PM By appointment only Telephone reference service available
Finding aids	No published finding aids
Photocopy	Staff makes copies, no charge.

Archives of the New York Orthopedic Hospital, founded in 1866 by Theodore Roosevelt, Sr. Most materials date from 1910 to the present. Papers of famous orthopedic surgeons: Russell Hibbs, Charles Fayette Taylor, and Newton M. Shaffer. Institutional records include minutes of committees and daily operating schedules. Photographs, lantern slides, and films. Rare books include anatomy, surgery, and orthopedic surgery, including incunabula. Instruments, equipment, and memorabilia.

Size: 50 linear feet.

18. THE NEW YORK PUBLIC LIBRARY

Astor, Lenox and Tilden Foundations
The Research Libraries
Rare Books and Manuscripts Division
Manuscripts and Archives Division
Fifth Ave and 42nd St, Room 324
New York, NY 10018-2788
(212) 930-0801

Inquiries to	Mary B. Bowling, Curator of Manuscripts
Access	Open to qualified and experienced researchers who need access to original materials
Hours	Mon–Wed, Fri–Sat: 10:00 AM–5:45 PM By appointment only. A minimum of 24 hours' notice is required because materials are stored off site. Telephone reference service available, but limited to confirming whether a particular item or collection is available. However, thorough responses will be provided to written requests for information.
Finding aids	*The New York Public Library, Research Libraries, Dictionary Catalog of the Manuscript Division* (Boston: G. K. Hall, 1967).
Photocopy	Paper photocopy is limited to 30 pages for requests by mail, 10 pages per day on site. For more pages, readers must place order for microfilm. Staff makes copies at charge of 30¢ per page. A postage and handling charge of $2.00 is added to mail orders.

The Lillian Wald papers (Henry Street Settlement, visiting nurses, social policy issues). Records of the U.S. Sanitary Commission, a Civil War era voluntary relief organization that supported the Union troops; collection includes many medical records. Collections in psychiatry and psychology, including papers of Erich Fromm, Robert J. Lifton, Gustav Scholer, and Max Wertheimer. Miscellaneous letters and manuscripts listed in catalog. Earliest item in the collection is an Arabic manuscript on medicine from 1375. Western materials date from 1758 to the present. Most materials pertinent to the study of medicine and health care are from 1850 to 1950 and are predominantly American. Photographs, instruments, and equipment, memorabilia of area physicians.

Size: ca. 850 linear feet.

19. NEW YORK STATE ARCHIVES

Room 11D 40
Cultural Education Center
Empire State Plaza
Albany, NY 12230
(518) 474-8955

Inquiries to	William Evans, Chief of Reference
Access	Open to anyone; restricted access only to records covered by personal privacy legislation
Hours	Mon–Fri: 9:00 AM–5:00 PM except for state holidays Telephone reference service available
Finding aids	*Guide to the Records of the New York State Archives* (new edition scheduled for 1990). On-line finding aids through the Collections Management System and RLIN for all series descriptions
Photocopy	Staff will xerox material that fits machine at charge of 25¢ per page. Will arrange to have photostatic copies at charge of $2.50 per photostat. Patrons may bring their own camera.

Records generated by state government. For example: records of the Department of Health; state hospital; Veterans Home at Oxford; scientists on state museum staff; New York State Investigation Commission; State Board of Charities; New York Insurance Department; and Attorney General. Materials date from 1640 to present; strong in colonial period and post-1911 except in the area of legislative records. No manuscripts in this collection, but they share facilities with Special Collections, Manuscript Division of the State Library. A few educational films. Photographs—glass plate photograph collection of the Division of Visual Instruction (in schools), State Board of Charities. Largest motion picture script collection in the world (54,000 scripts). Scripts date from 1921 to 1965: computer-generated microfiche index with 23 access points to the script collection. Strong in foreign language scripts (with translations); all include censors' notes.

Size: 49,000 cubic feet.

20. NEW YORK STATE LIBRARY, MANUSCRIPTS AND SPECIAL COLLECTIONS

Empire State Plaza
Albany, NY 12230
(518) 474-5963

Inquiries to	James Corsaro, Associate Librarian
Access	Open to state government employees and qualified researchers; restricted access to some collections

Hours	Mon–Fri: 9:00 AM–5:00 PM Telephone reference service available
Finding aids	A number of finding aids are available. Most collections are included in the National Inventory of Documentary Sources (microfiche) and on RLIN.
Photocopy	Photocopy, microfilm, and photostat available depending on condition of the material to be duplicated. Staff makes copies, at charge of 25¢/page for photocopy, $4.00/page for photostat. Fee for microfilm varies.

Private papers of individuals and documents of private organizations in New York State or related to the state, 1630 to the present, with most materials from the 19th century. Important examples of published medical literature, including a large collection of 19th century pamphlets from state hospitals. Notable collections include the following: Willowbrook Developmental Center Research Collection (41 cubic feet); Elber van der Veer Collection, bound volumes of surgical cases observed in Albany Hospital, private papers (19th century); Dutchess and Putnam Counties Medical Society: 1913–1919; John W. Francis papers: 1780–1866; Earl W. Stevens papers: 1795–1856.

21. New York University College of Dentistry Archives

Dental Library
345 East 24th St
New York, NY 10010
(212) 998-9792

Inquiries to	Erich Meyerhoff, Archivist
Access	Open to NYU students and staff, interested scholars
Hours	Thur: 2:00 PM–5:00 PM By appointment only Telephone reference service available
Finding aids	No published finding aids but catalog available there.
Photocopy	Staff makes copies if condition of material permits; $7.50 service charge and 25¢ per page.

History of the College of Dentistry: institutional documents, clippings, some personal papers of the faculty. Materials from 1865 to the present; most materials from 1880 to 1969. Some photographs and films. Rare book collection contains 1,600 volumes of dental and medical books, chiefly 19th century, some (300) earlier, including the first printed work devoted exclusively to dentistry and the first English language dental book. Oral history, instruments and equipment.

Size: 170 linear feet.

22. New York University Medical Center Archives

Ehrman Library
550 First Ave
New York, NY 10016
(212) 340-8280

Inquiries to	Michael Rissinger, Archivist
Access	Open to NYU students and staff, interested scholars
Hours	Mon–Fri: 9:00 AM–5:00 PM By appointment only Telephone reference service available
Finding aids	No published finding aids; catalog to collection available there
Photocopy	Staff makes copies if condition of material permits; $7.50 service charge and 25¢ per page. Copies of photographs are also available; $2–5.00 per exposure, by medical center staff only.

Material documenting the history of the Medical Center: institutional documents (occasional minutes, bulletins); photographs; newspaper clippings; no personal papers of faculty or administrators. Some materials from the NYU Veterinary Hospital, the Postgraduate Medical School and Hospital, and some Bellevue Hospital Medical College records. Materials date from the founding of the Medical Department of NYU in 1841 to the present; materials from 1880 to 1960 make up most of the collection. Films and photographs, rare books, instruments and equipment, memorabilia (chairs, hats, paintings).

23. The Rockefeller Archive Center

15 Dayton Ave
Pocantico Hills
North Tarrytown, NY 10591-1598
(914) 631-4505

Inquiries to	Darwin H. Stapleton, Director
Access	Open to serious scholars and researchers
Hours	Mon–Fri: 9:00 AM–4:45 PM Appointments not required but prior contact is preferred. Telephone reference service available
Finding aids	*A Guide to Archives and Manuscripts at the Rockefeller Archive Center* (1989). *Photograph Collections in the Rockefeller Archive Center* (1986). Published surveys of resources for the history of nursing, psychiatry and related areas, child studies, and labor and industrial relations. Unpublished registers and inventories are available at the Archive Center. The Rockefeller Archive Center participates in RLIN.
Photocopy	Researchers may request photocopies of documents and photographs; Archive Center staff copies and mails the material with invoice. Charges are 20¢ per page for the first 1,000 copies ordered during a six-month period; 30¢ per page for 1,001–5,000 copies; 50¢ per page after 5,000 pages.

The Archive Center was founded to house, preserve, and make available to researchers records related to the wide-ranging philanthropic activity of members of the Rockefeller family. Founding institutions whose archives are housed here include the Rockefeller family, the Rockefeller Foundation, Rockefeller Brothers Fund, and the Rockefeller University. Subsequent additions have brought in the papers and records of organizations and individuals associated with these institutions, as well as the records of the Commonwealth Fund and the Russell Sage Foundation. Collection also includes films and photographs. Materials begin as early as John D. Rockefeller's first ledger in 1855 and continue to the present.

Archives and records of organizations: Bureau of Social Hygiene; China Medical Board; Commonwealth Fund; Davison Fund, Inc; General Education Board; Health Research Fund, Inc; Laura Spelman Rockefeller Memorial; Memorial Sloan-Kettering Cancer Center; Population Council; Rockefeller Brothers Fund; Rockefeller Foundation; Rockefeller Sanitary Commission for the Eradication of Hookworm Disease; Rockefeller University. Papers and manuscripts of individuals: Oswald

T. Avery; Alexis Carrel; Alfred E. Cohn; Nelson C. Davis; Lindsley F. Kimball; John H. Knowles; Rebecca C. Lancefield; Phoebus A. T. Levene; Jacques Loeb; Harold H. Loucks; Alfred E. Mirsky; the Rockefeller Family; Hugh Smith; Benjamin Washburn.

Size: A total of about 23,000 cubic feet of material is housed in the RAC. Approximately 50% of the materials are related to medicine and health care.

24. The Medical Research Library of the Health Science Center State University of New York at Brooklyn

450 Clarkson Ave
Brooklyn, NY 11203
(718) 270-3780

Inquiries to	Gordon Mestler, Archivist Kathleen E. Powderly, Director, Division of Humanities in Medicine
Access	Open to anyone
Hours	Mon–Fri: 9:00 AM–5:00 PM By appointment only Telephone reference service available
Finding aids	No published finding aids; handwritten catalog to the collection available on site.
Photocopy	Staff makes copies if materials are not too fragile at charge of 10¢ per page.

Collection includes archives of the Long Island College of Medicine (est. 1860) and Long Island College Hospital; current archival materials of SUNY at Brooklyn; materials related to the history of medicine in Brooklyn (1860–present, predominantly from the Civil War to 1920). Dr William Schroeder's collection on the history of medicine in Brooklyn (1860–1900). Daybooks of some important Brooklyn physicians, including Alexander Skene, the author of a widely used 19th century text on gynecology and obstetrics, and various annual reports from Brooklyn's hospitals, charity associations, and dispensaries, many of which are now closed. The varied correspondence of Dr Joseph Raymond, secretary of the Long Island College Hospital during the 1890s and early 20th century. Also many of the materials formerly kept at the Kings County Medical Society, including copies of directories, reports, and newsletters sent to its membership. Photographs and rare books.

25. Robert L. Brown History of Medicine Collection State University of New York at Buffalo Health Sciences Library

Abbott Hall
SUNY at Buffalo
Buffalo, NY 14214
(716) 831-3024

Inquiries to	Lilli Sentz, Associate Librarian
Access	Open to anyone; restrictions on who can borrow materials, some materials are noncirculating.
Hours	Mon–Fri: 8:30 AM–5:00 PM or by appointment Telephone reference service available
Finding aids	*Pre-Nineteenth Century Catalog of the Robert L. Brown History of Medicine Collection*
Photocopy	Yes, depending on the condition of the materials; staff makes copies at charge of 10¢ per page.

Collection consists of more than 12,000 volumes of 19th century medical monographs with particular strength in the areas of surgery, obstetrics/gynecology, dentistry, psychiatry, and pharmacology in addition to the 439 titles included in the catalog of pre-19th century works (earliest holdings are from the 15th century). Includes the private collections of Roswell Park, James Platt White, George N. Burwell, and L. Maxwell Lockie (collection on gout and rheumatism), as well as the medical collection of the former Grosvenor Library. Small collection of medical instruments, the Edgar R. McGuire Medical Historical Instrument Collection.

26. Slide Archive of Historical Medical Photographs State University of New York at Stony Brook Health Sciences Center

PO Box 66
East Setauket, NY 11733
(516) 444-3100

Inquiries to	Jeanne Nook
Access	By mail
Hours	Not applicable No telephone reference service
Finding aids	*Illustrated Catalog of the Slide Archive of Historical Medical Photographs at Stony Brook* (Westport, Conn, Greenwood Press, 1984)
Photocopy	Not applicable

Almost 5,000 slide copies of historical medical photographs taken from the 1850s to the present. The collection has a representative sample of photographs in archives across the United States. It is designed to make slides accessible at cost for teaching and scientific publications. Prospective users should consult the published catalog, which is indexed and contains instructions for ordering slides. The catalog is indexed by personal name, institutional name, photographers, geographic location, medical conditions, chronology, and subject.

27. State University of New York Health Science Center at Syracuse Library Archives

766 Irving Ave
Syracuse, NY 13210
(315) 473-4580

Inquiries to	Rosemarie Bundy, Senior Staff Associate
Access	Open to the public; primarily used by staff and students of the Health Sciences Center
Hours	Mon–Fri: 10:00 AM–8:00 PM Telephone reference service available for brief inquiries
Finding aids	No; they are just beginning to catalog the collection.
Photocopy	Reader makes copies at charge of 10¢ per page or 8.3¢ with purchased copy card.

Records of the SUNY Health Science Center at Syracuse and its predecessor institutions, the Geneva Medical College and Syracuse University. Personal papers of presidents of Syracuse University and SUNY Health Science Center (late 19th century–present, most materials devoted to 1930–present). Some 19th and early 20th century records from area hospitals (Crouse Irving Memorial, Good Shepherd, Silverman). Personal papers, book collections, and memorabilia of area physicians connected with the institution. Large collection of instruments and equipment, including old medical supplies and orthopedic equipment. Large photographic collection. Separate rare book collection of 2,000 volumes: early American medicine; late 19th and early 20th century German medicine; material pertaining to Geneva Medical College (1834–1872). Includes materials from the 17th through 19th centuries, with the majority of materials devoted to the 19th century.

Size: archives, ca. 1,600 linear feet.

28. Museum and Library of the Suffolk Academy of Medicine

850 Veterans Memorial Highway
Happauge, NY 11788
(516) 724-7970

Inquiries to Joyce Bahr, Acting Librarian

Access Open to the public; access to rare books by application to librarian

Hours Mon–Fri, 9:00 AM–5:00 PM
Telephone reference service available

Finding aids No published finding aids, but a catalog to collection is available there.

Photocopy Staff makes copies at charge of 10¢ per page.

Museum collection of medical and surgical instrumentation, including complete sets of Civil War era surgical instruments, pharmaceutical instruments and equipment, early baby bottles, and nursing devices. A few archival materials: some letters, documents, minutes of the Academy (incomplete and uncataloged). Materials from the early 1800s to present; most are 19th century. The book collection includes all holdings of the Suffolk County Historical Society related to medicine.

Size: 15–20 display cases.

29. Special Collections, Milbank Memorial Library, Teachers College, Columbia University

New York, NY 10027
(212) 678-4104

Inquiries to David Ment, Head, Special Collections, or Lucinda Manning, Manuscripts Curator

Access Open to anyone

Hours Mon: 2:00 PM–6:00 PM, Tues, Thur, Fri: 10:00 AM–6:00 PM, Wed: 2:00 PM–9:00 PM (during fall and spring semesters); closed daily for lunch 1:00 PM to 2:00 PM. Appointments recommended but not required
Telephone reference service available

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Local low-dose infusion of streptokinase for massive pulmonary embolism when systemic thrombolysis is contraindicated

MICHAEL A. NAKAO, MD

Thrombolytic therapy is indicated for acute massive, life-threatening pulmonary embolism.[1] However, it is contraindicated in patients with active bleeding, and in patients who have experienced a cerebrovascular or ocular procedure (or disease process) within the previous two to three months. Many physicians would also not use such therapy in cases of surgery, trauma, invasive procedure, childbirth, cardiopulmonary resuscitation, bleeding within the past ten days, severe hypertension, pregnancy, significant bleeding tendency,[2] or in patients more than 75 years of age. The following report describes a 77-year-old patient with acute massive pulmonary embolism and active vaginal bleeding who was treated by an infusion of streptokinase at one tenth the usual rate directly onto the pulmonary thromboembolism.

CASE REPORT

A 77-year-old obese woman with metastatic endometrial carcinoma was treated for two days with an intrauterine cesium implant at bed rest. She had ongoing vaginal bleeding, using approximately six pads daily. Three hours after the implant was removed, the patient was walking when she experienced sudden onset of lightheadedness and dyspnea with central chest pain. She collapsed, was unconscious, and had no pulse. After cardiopulmonary and volume resuscitation, intubation, mechanical ventilation, and the initiation of dopamine therapy, she had a blood pressure of 86/47 mm Hg and sinus tachycardia of 160 beats/min. On 50% oxygen, with a minute ventilation of 24 liters, the arterial pH was 7.20; P_{CO_2}, 47 mm Hg, and P_{O_2}, 63 mm Hg.

The electrocardiogram showed acute right bundle branch block, with nonspecific ST-T wave abnormalities. The chest roentgenogram showed increased lucency over the right hemithorax. A right pulmonary artery catheterization detected the following pressures: right atrium (RA), 10–15 mm Hg; right ventricle, 45/10 mm Hg; pulmonary artery (PA), 45/30 mm Hg; and a cardiac index (CI) of 2.7 L/min. A satisfactory pulmonary artery wedge pressure (PAW) wave form was initially unobtainable. A perfusion lung scan showed complete loss of perfusion to the right upper lobe and all the basal segments of the right lower lobe. Right pulmonary angiography demonstrated a massive thrombus involving the right main pulmonary artery and its branches (Fig 1).

With the catheter in the right main pulmonary artery, streptokinase infusion was instituted through the catheter at 10,000 units per hour, without a loading dose, onto the thromboembolism. Five hours after the infusion was started, on 50% oxygen with a minute ventilation of 14 liters, arterial pH was 7.54, P_{CO_2} was 26 mm Hg, P_{O_2} was 169 mm Hg, the blood pressure normalized, and dopamine was discontinued. After 30 hours of therapy the infusion of streptokinase was stopped, and heparin therapy was instituted. A follow-up arteriogram (Fig 2) and perfusion lung scan showed right lung perfusion to be increased an estimated three times. On 40% oxygen by face mask, the arterial pH was 7.50; P_{CO_2}, 34 mm Hg, and P_{O_2}, 87 mm Hg. The pulse was 100 beats/min; blood pressure, 135/76 mm Hg; RA, 12 mm Hg; PA, 38/21 mm Hg; PAW, 14 mm Hg; and CI, 3.0 L/min. Throughout the streptokinase infusion, although the euglobinlysis time decreased to one hour, and the thrombin time was 31.3 sec with a control of 17.3 sec, the partial thromboplastin and prothrombin times did not change significantly, and the fibrinogen level remained within normal limits (four determinations were obtained during the infusion). The patient's vaginal bleeding remained unchanged. The hemoglobin decreased from 10.0 g/dL to 9.1 g/dL, and the hematocrit decreased by 3.2 points. The patient was subsequently discharged from the hospital.

DISCUSSION

Very low dose streptokinase infusion

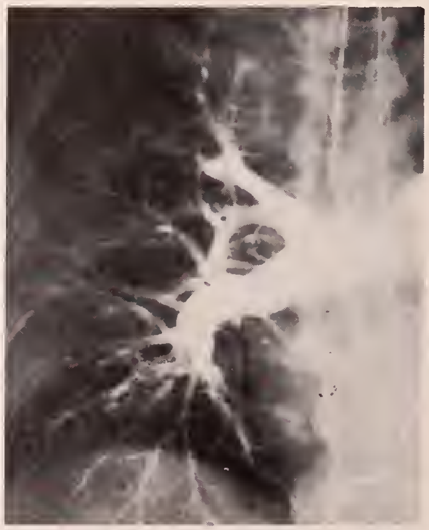

FIGURE 1. Pulmonary angiogram demonstrating large filling defect within the right main pulmonary artery.

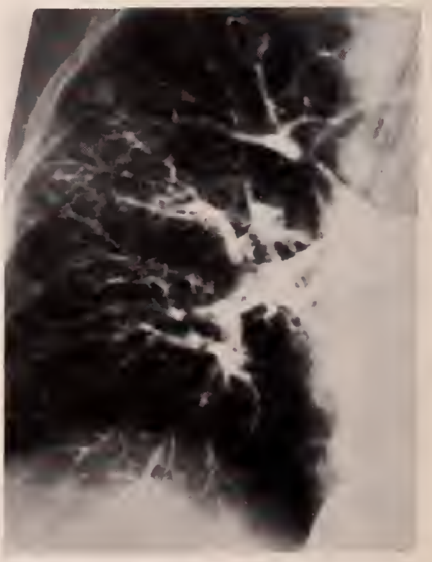

FIGURE 2. Marked dissolution of massive pulmonary embolus by direct pulmonary artery infusion with streptokinase.

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into a systemic artery thrombus has been shown to be effective therapy, achieving clot lysis without side effects or lowering fibrinogen levels below normal.[3] In this patient, infusion of streptokinase at one tenth the usual rate without a loading dose onto the principal site of pulmonary artery occlusion effected rapid clot lysis and significant hemodynamic and clinical improvement within five hours, without significant bleeding. Other authors have reported similar results.[4]

The pulmonary artery catheter failed at first to give a satisfactory PAW wave form, but with marked hemodynamic improvement, likely due to clot lysis, a satisfactory PAW wave form was obtained. This has been described in other cases of embolic pulmonary artery occlusion.[5] Acute massive, life-threatening pulmonary embolism in which systemic thrombolytic therapy is contraindicated has traditionally only been treated with pulmonary embolectomy. However, embolectomy is not always feasible. Infusion of a thrombolytic agent at one tenth the usual rate, or even lower rates, directly onto the major thromboembolus might reduce mortality while decreasing hemorrhagic complications. Such infusions would probably best be terminated as soon as an acceptable hemodynamic response is obtained, and should be combined with an inferior vena cava filter or, if not contraindicated, heparin infusion.

The potential role of this therapy depends on whether the systemic bleeding complications of standard thrombolytic therapy can be significantly reduced. Forty-two percent of patients in a urokinase-streptokinase pulmonary embolism trial, who had had no contraindications to thrombolysis, had decreased hematocrits by ≥5 points.[6] In this patient, whose hematocrit decreased only 3.2 points, local infusion may have effected rapid binding of the streptokinase-plasminogen complex, limiting exposure of the streptokinase to inactivating factors and antibodies and thus allowing a sufficiently high titer to effect local thrombosis. Streptokinase has a short half-life, and infusion at one tenth the usual dose might have allowed enough inactivation of the agent to protect distal thrombi. Tissue plasminogen activator is even shorter acting, and thus may have further theoretical advantages.

At this time, this therapy might best be considered for patients with acute life threatening pulmonary embolism, in which neither full-dose thrombolysis nor pulmonary embolectomy are possible. The effectiveness of local streptokinase infusion should be evaluated in a larger series of patients.

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The use of a Swan-Ganz catheter and streptokinase in the management of massive hemoptysis

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Exsanguinating hemoptysis (greater than 1,000 mL/24 hr or greater than 150 mL/hr) carries a 75% mortality rate.[1] Aggressive surgical therapy is acknowledged to improve survival.[2,3] Medical management for endobronchial control of hemorrhage has included pharmacologic intervention and sophisticated techniques requiring expertise. These procedures include pulmonary separation with the Carlin's and Robertshaw tubes,[4] selective bronchial intubation,[1] balloon tamponade,[5] ice cold saline lavage,[6] and bronchial artery angiography and embolization.[7] This report describes the use of a Swan-Ganz balloon flotation catheter for the temporary tamponade of massive endobronchial hemorrhage in a patient with bronchogenic carcinoma. In addition, prior to the patient's death, streptokinase was utilized to dissolve a blood clot, a complication of hemorrhage, which had totally occluded the endotracheal tube. Although the role of these interventions requires further clarification in massive hemoptysis, the simplicity of their use did enable immediate resolution of two difficult problems associated with exsanguinating hemorrhage.

CASE REPORT

A 46-year-old man with bronchogenic carcinoma was admitted to the intensive care unit (ICU) with massive hemoptysis (200 cc in 2 hr). The patient's pulmonary history had been significant for a smoking history of 100 pack years and evaluation of blood-streaked sputum six weeks prior to ICU admission. A chest roentgenogram disclosed a left hilar mass; subsequent computerized axial tomography demonstrated a left hilar mass with mediastinal and hilar adenopathy. Flexible fiberoptic bronchoscopy was nondiagnostic, with open lung biopsy revealing poorly differentiated squamous cell carcinoma. A course of chemotherapy was administered (cisplatinum/velban/5-fluorouracil), and thoracic irradiation was given (a total of 4,500 rads) with intermittent occurrence of hemoptysis during the five weeks of combined chemotherapy and radiation therapy.

Five and a half weeks after the diagnosis was made, massive hemoptysis occurred. On admission to the ICU, the patient's blood pressure was 90/70 mm Hg; heart rate, 130

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beats/min; respiratory rate, 22/min; and with each breath he would cough bright red blood. Efforts to locate an experienced physician who could insert a Carlin's tube were unsuccessful. Accordingly, the patient was sedated with 5 mg intravenous diazepam and given 15 mg intravenous codeine just prior to nasotracheal intubation with a #8 tube. Emergency flexible bronchoscopy revealed bleeding that was localized to the left mainstem bronchus and aspiration of blood into the right lung. The hemorrhage spontaneously resolved, and a chest film demonstrated the unchanged left hilar mass and a new right lower lobe infiltrate. The patient received mechanical ventilator assistance, which maintained an arterial oxygen tension (PaO_2) of 80–100 mg on 30% oxygen.

The patient's initial hemoglobin level was 7.1 g/dL, and he required a blood transfusion of six units of packed red blood cells. All of the coagulation parameters were within normal limits, as were the fibrinogen level and fibrinogen degradation products. His blood pressure stabilized, and he remained alert, writing notes to his family.

Discussions with the attending physician and the patient, as well as the family, resulted in a plan to render supportive medical care without surgical intervention. Cautious suctioning was utilized to keep the nasotracheal tube patent for fear that blind suctioning would penetrate the clotted left mainstem bronchus. As a result, a modest elevation in the peak pressure on the ventilator was noted (35–50 cm H_2O). Twenty-four hours after admission, spontaneous hemorrhage through the nasotracheal tube occurred totalling 1,500 cc, with the patient again becoming acutely hypotensive to 80 mm Hg systolic. Life-threatening airway compromise from exsanguinating hemoptysis was apparent. In an attempt of blind tamponade of the region of hemorrhage in the left mainstem bronchus, a Swan-Ganz balloon flotation catheter was passed down the nasotracheal tube. With visual guidance provided by the flexible fiberoptic bronchoscope, the balloon flotation catheter was easily directed into the blood-filled left mainstem bronchus. The usual 1.5 cc of air to inflate the balloon did not result in immediate cessation of bleeding. An additional 0.5 cc resulted in no further hemorrhage, and the bronchoscope was removed with the catheter left behind in the lumen of the nasotracheal tube. A repeat arterial blood gas on 40% oxygen revealed a Po_2 of 99 mm Hg. A repeat chest film was not attempted for fear that movement of the patient would result in further hemorrhage or dislodgement of the balloon catheter. The patient received an additional transfusion of four units of packed red blood cells. Over the following 12 hours, the patient remained alert, and cautious suctioning of the tube continued, although the peak pressures on the ventilator continued to rise to unacceptable levels (90 cm H_2O). A repeat arterial blood gas disclosed a Po_2 of 54 mm Hg and Pco_2 of 59 mm Hg with a pH of 7.32. The patient was placed on 100% oxygen and suctioning, with saline lavage. Bronchoscopic use of forceps was unsuccessful to relieve an apparently occluded nasotracheal tube of a blood clot. The patient's blood pressure dropped to 80 mm Hg. With the family present and consenting, 100,000 U of streptokinase was instilled blindly down the nasotracheal tube. Within one minute, the ventilator pressure fell to 50 cm H_2O, blood pressure returned to 120 mm Hg, the patient was again arousable and alert, and the repeat arterial blood gas disclosed a pH of 7.36, Pco_2 of 41 mm Hg, and Po_2 of 82 mm Hg on 40% oxygen. Continued suctioning for the following four hours was difficult due to resistance, but no hemorrhage was noted after the streptokinase had been instilled. Once again, unacceptable peak pressure levels were reached on the ventilator, and four and a half hours after the streptokinase was instilled, exsanguinating hemorrhage through the nasotracheal tube and ventilator tubing occurred and the patient died. Inspection of the balloon postmortem showed that it was intact. No autopsy was obtained. Three hours after the patient's death, the blood in the tubing of the ventilator remained unclotted.

DISCUSSION

Massive hemoptysis is a life-threatening disorder with both medical and surgical intervention having limited success. Medical management often requires personnel with special training in controlling hemorrhage. In addition, the equipment necessary may not be readily available in cases of unexpected exsanguinating hemoptysis. The use of the Swan-Ganz catheter and streptokinase are, therefore, noteworthy for availability, simplicity in use, and effectiveness in management problems associated with massive hemoptysis.

The Swan-Ganz balloon flotation catheter is commonly used in critical care units for hemodynamic monitoring, and therefore it is readily available in the event of life-threatening hemorrhage for airway tamponade, as in this patient who had a nasotracheal tube. The use of this catheter for a nonhemodynamic related disorder, however, is not standard procedure. It has been utilized in bedside pulmonary angiography[8] and microvascular cytologic sampling of the pulmonary artery.[9] The Fogarty catheter, a catheter used principally for removal of arterial emboli, has been used as the principal device for tamponade of an airway compromised by serious hemorrhage. The Swan-Ganz catheter, however, offers several advantages over the Fogarty, including the former's availability, universal familiarity, and the spectrum of inflated balloon diameters related to the volume of air introduced. The potential limitation of the Swan-Ganz catheter is similar to that of the Fogarty catheter, in that bronchoscopy is required for immediate assurance of its proper placement. In addition, balloon rupture of the Swan-Ganz is possible if the balloon is inflated beyond the recommendations of the manufacturer (1.5 cc air results in 14 mm diameter; 3 cc air results in 16 mm diameter).

In addition to exsanguination and aspiration of blood, another complication of massive hemoptysis is occlusion of the airway or endotracheal tube with blood clots.[10,11] Occlusion of the nasotracheal tube occurred in this patient, even though he was suctioned frequently, lavaged with saline, and even unsuccessfully manipulated by bronchoscopy forceps to "piecemeal" out the clot. When the ventilator could no longer deliver the set volume, 100,000 U of streptokinase was slowly instilled down the tube. Although the immediate fall in airway pressures without hemoptysis for four hours acknowledged the effectiveness of streptokinase in clearing the tube, one must remain cautiously optimistic about its use for this condition. Three hours after exsanguinating hemorrhage occurred, or seven hours after streptokinase was instilled down the tube, the blood in the ventilator tubing remained unclotted. Inspection of the Swan-Ganz catheter postmortem revealed the balloon to be intact. Therefore, endobronchial absorption of streptokinase was likely with a probable lytic state resulting.

In conclusion, massive hemoptysis is associated with an extremely high mortality, resulting in exsanguination and death from associated asphyxiation. This report demonstrates that two major complications of massive hemoptysis in a patient with a nasotracheal tube can be initially managed effectively. The Swan-Ganz balloon flotation catheter can be manipulated through the tube with relative ease for temporary tamponade of an endobronchial hemorrhage. In addition, life-threatening blood clot occlusion of a nasotracheal tube can be temporarily lysed with direct instillation of streptokinase to allow resumption of mechanical ventilation. Because of the limitation of continuous balloon inflation on the integrity of the catheter, and the possible systemic lytic state from streptokinase, the use of these two interventions should be reserved for temporary life-saving measures.

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Trabecular (neuroendocrine) carcinoma of the skin

Report of four cases and review of the literature

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In 1972, Toker[1] reported five cases of a previously unrecognized cutaneous malignancy that he designated as trabecular carcinoma of the skin. Grossly, these lesions were pink, well-circumscribed, non-encapsulated, and firm. Microscopically, the tumor cells had conspicuous mitoses, scanty cytoplasm, and formed irregular sheets (or trabeculae) that infiltrated the dermis but tended to spare the epidermis. Leong et al[2] described a "ball-in-mitt" pattern represented by one or two crescentic tumor cells encircling an oval cell. Several years later, Tang and Toker,[3] using electron microscopy, provided further insight by describing membrane-bound, dense-core granules indistinguishable from those found in neuroendocrine tumors (APUDomas), and the authors characterized this entity as Merkel cell tumor. Still others have called this tumor "primary neuroendocrine carcinoma of the skin," because of reports of ectopic production of corticotropin (ACTH),[4] calcitonin,[5] and somatostatin.[6] Kroll and Toker[7] point out, however, that the most important diagnostic feature of this tumor is its cord-like structure; they therefore suggest that the term "trabecular carcinoma" is superior to other alternatives.

As rare or as new as this form of malignancy might be, it has generated much international interest, as recent reports from Great Britain,[8] Israel,[9] Australia,[10] and Japan[11] would attest. Trabecular carcinoma of the skin is more than an academic curiosity, for it poses a dilemma for both the pathologist and the clinician. In making this diagnosis, the pathologist must consider a differential diagnosis that includes small cell squamous carcinoma, malignant melanoma, histiocytosis X, eccrine sweat gland carcinoma, malignant small cell lymphoma, and primary or metastatic cutaneous neuroblastoma.[12] Kroll and Toker[7] emphasize that the majority of these tumors can be diagnosed by simple histologic analysis in the clinical setting, reserving electron microscopy for only the most difficult cases. The medical practitioner, then, must take the diagnosis of trabecular carcinoma and provide appropriate management for the patient afflicted with the disease. This has not been an easy task because, until relatively recently, the literature has not contained sufficient information on which to base decisions.

Four cases of trabecular carcinoma of the skin are presented here, demonstrating the spectrum of the disease. A review of the literature is also included to provide insight into the natural history, prognosis, and therapy associated with this tumor.

CASE REPORTS

Case 1. In 1980, a 73-year-old woman was seen at another medical center for an ulcerated lesion of her left lower thigh, with associated inguinal adenopathy. Results of a metastatic workup were negative, and the patient was taken to the operating room for excision of this ulceration and lymph node dissection. Pathology reports described a small cell-undifferentiated epithelial tumor—trabecular carcinoma. No adjuvant therapy was given.

Nine months later, the patient presented to the same institution complaining of fever, left flank pain, and abdominal pain. Evaluation at that time included an intravenous pyelograph (IVP), computed tomographic (CT) scan, and gallium scan that detected a mass obstructing the left ureter, with resultant hydronephrosis. A diagnosis of pyelonephritis secondary to an obstructing uropathy was made. The patient's pyelonephritis responded to intravenous antibiotics, and she was referred to Montefiore.

On presentation, the patient's examination was notable for a 10 × 5-cm mass slightly to the left of the umbilicus. No other peripheral lymphadenopathy was appreciated. The lesion was presumed to be metastatic trabecular carcinoma, and a regimen of radiotherapy was begun. After a total of 2,500 rad, the mass was described as "barely palpable." The patient went on to receive a complete course of 4,800 rad. She has remained free of tumor by physical examination, IVP, and CT scan for the succeeding five years.

Case 2. In 1982, an 87-year-old man with multiple medical problems and dementia was brought to Montefiore from a local nursing home. A lesion was noted on the patient's left upper eyelid. An excisional biopsy was subsequently performed, and the lesion was found to be "Merkel cell carcinoma." However, because of the patient's advanced age and generally fragile state, further treatment was deferred.

In 1984, the patient presented at Montefiore, complaining that he was unable to see from his left eye. A globular, red mass, measuring 8 × 16 mm, was found on his left upper eyelid. No cervical nodes were present, and liver function studies were within normal limits. The patient received 6,000 rad of radiotherapy. On completion of this course,

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the lesion had resolved.

Seven months later, the patient returned to the hospital; he had a 2-cm parotid mass and two palpable supraclavicular nodes. Needle aspiration of the parotid mass demonstrated necrotic malignant cells. He was then admitted to the hospital with bilateral pulmonary infiltrates. A core biopsy of the parotid mass demonstrated a "metastatic Merkel cell" tumor. Further evaluation, including a bone survey, suggested diffuse bone and pulmonary metastases. He received an additional 2,400 rad to his parotid with resolution of the mass. He recovered from his pneumonia and was returned to the nursing home, where he died some weeks later.

Case 3. In 1983, an 84-year-old woman presented at Montefiore Medical Center complaining of a lesion on her left calf that had been present for approximately three years. On examination, a red, friable mass, measuring approximately 1 cm, was found. There was no lymphadenopathy present. An excisional biopsy was performed, showing a "Merkel cell tumor, margins clear." The patient received 6,860 rad to the area after surgery.

Two years later, a nodule was noted in the treatment field without any associated adenopathy. Initially, the nodule was firm, red, and seemed to be stable in size. When it began to grow, it was biopsied again and proved to be recurrent Merkel cell tumor. The patient received additional radiotherapy in the form of implanted irridium seeds, from which she received 4,800 rad. At the time of this writing, the patient had a 2 × 2-cm ulcerated area at the site of the implants, and still had no appreciable adenopathy.

Case 4. In 1986, a 78-year-old woman presented to her dermatologist complaining of a five-month history of a mass growing over her left eyebrow. The lesion was biopsied and was diagnosed as a trabecular carcinoma. She was then referred to Montefiore.

On examination, the patient had a 3-cm lobular, erythematous mass with central umbilication over the left brow. It was nontender, and there was no discharge or adenopathy. The patient underwent excision of the lesion that was "3.0 × 1.5 cm, light ivory, glistening in appearance and rubbery in consistency." On microscopic evaluation, this was a trabecular carcinoma. The patient underwent reconstructive surgery by means of local rotation and forehead flaps, and she was followed closely in the postoperative period.

Five months after excision of her lesion, the patient was found to have an 8 × 5-mm movable, subcutaneous mass inferior to the medial canthus of her left eye, and a 2 × 1-cm mass in the left parotid gland. There was no evidence of recurrent primary tumor, nor was there any appreciable cervical adenopathy. Needle aspiration biopsies of each mass were performed, and the pathologic findings were consistent with Merkel cell tumor (Figs 1–3).

In March 1987, the patient underwent a left parotidectomy and local excision of the tumor of the left infraorbital area. The parotid specimen contained normal salivary gland with an encapsulated lymph node invaded by a small cell, anaplastic tumor. The infraorbital lesion was also an anaplastic tumor. Findings in both these lesions were consistent with Merkel cell tumor. The patient is currently receiving radiotherapy to the face and neck.

A summary of these cases is presented in Table I.

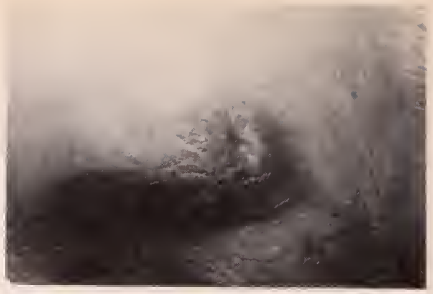

FIGURE 1. Case 4: Preoperative view of lesion on left eyebrow.

Discussion

Although trabecular carcinoma of the skin has been recognized as a distinct entity for less than 20 years, the literature currently contains enough information for this tumor to be adequately and accurately characterized.

Initially, trabecular carcinoma was thought to be a low-grade malignancy. As late as 1986, it was reported that this tumor "rarely disseminates to distant sites and rarely causes death."[13] However, it is interesting to note that the patient described in this same case report died only 15 months after initial presentation, with metastases to the left lung, liver, left kidney, abdominal lymph nodes, and bilateral cerebellar hemispheres.

The first clinical series describing trabecular carcinoma began to appear in 1982. At that time, Kroll and Toker[7] had treated 30 patients who carried this particular diagnosis. A follow-up of greater than one year (range, 1 to 10 years) was obtained in 23 of these cases. The authors described this tumor as one that afflicted patients in their seventh and eighth decades, and as one that demonstrated a predilection for the head, neck, and upper extremities. After extirpative surgery, 48% of patients were completely free of disease, and only 22% died as a direct consequence of the tumor. In addition, the authors noted that tumor response to both radiation and chemotherapy was the same, ie, rapid regression followed by recurrence within six to 12 months. Concurrently, Ecker et al[14] and Wick and his associates at the Mayo Clinic[15] reported the propensity of trabecular carcinoma to recur and metastasize to the liver, lung, bone, and brain.

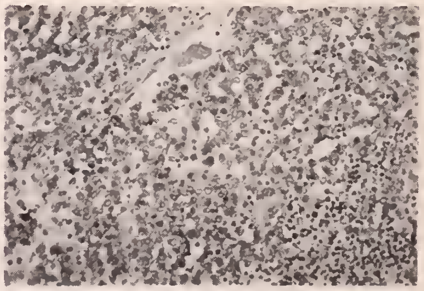

FIGURE 2. Case 4: The trabecular pattern of the tumor is seen here; this feature is not seen in all fields. The size of the tumor cells can be compared with the lymphocytes in the right lower corner (hematoxylin-eosin stain; original magnification × 100).

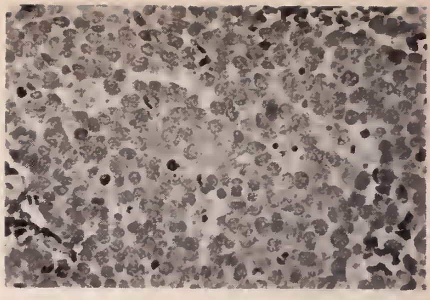

FIGURE 3. Case 4: The tumor cells characteristically have scant cytoplasm. The nuclei have finely stippled chromatin and inconspicuous nucleoli. Increased mitotic activity and degenerating individual cells, as seen here, are also features of this entity (hematoxlin-eosin stain; original magnification × 200).

Raaf and his colleagues[16] suggested that the mortality figures in the early literature did not adequately reflect the lethal nature of this malignancy. After reviewing their personal experience with four cases and combining this with an additional 76 cases described in the literature, these authors echoed Kroll and Toker in characterizing trabecular carcinoma as a disease of the elderly that appears, in order of decreasing frequency, on the head and neck (44%), leg (28%), arm (16%), or buttock (9%). A single case of trabecular carcinoma has also been reported in the vulva.[17]

Raaf et al[16] further noted the rate of local recurrence to be 36%, of regional metastatic disease to be 53%, of distant metastases to be 28%, and of death due to metastatic disease to be at least 25%. They therefore recommended that patients with primary lesions should undergo wide excision of the tumor, with concurrent prophylactic regional node dissection. Similar statistics regarding the nature of the malignancy were pro-

TABLE I. Summary of Four Cases of Trabecular Carcinoma

Patient No.	Age/Sex	Primary Site	Treatment/Primary Tumor	Recurrence/Metastasis	Treatment Regional and/or Metastatic Disease	Status
1	73/F	Left lower thigh + inguinal adenopathy	Excision + lymph node dissection	Intra-abdominal mass (nine mo later)	Radiotherapy, 4,880r	Disease-free for four years
2	87/M	Left upper eyelid	Excision	a. Left upper lid (two yr later) b. Parotid mass and supraclavicular nodes (seven months after a)	Radiotherapy, 6,000r	Died three yr after presenting with disease
3	84/F	Left calf	Excision, radiotherapy, 6,860r	Left calf (two yr later)	Irridium seeds, 4,800r	Local ulceration four yr after presenting with disease
4	78/F	Left eyebrow	Excision	Left medial canthus + left parotid node (five mo later)	Local excision + left parotidectomy	Radiotherapy nine mo after presenting with disease

vided by Cotlar et al,[18] Meland and Jackson,[19] Goepfert and colleagues,[20] Sibley and associates,[21] and Pajor et al.[22] The results of these studies are presented in Table II.

In addition to surgical excision of the primary lesion, radiotherapy proved somewhat efficacious in a number of the cases reported by Kroll and Toker. DeWolf-Peeters, in Belgium,[23] had even more striking success and reported his series of five patients, all of whom remained in good health during a period of 18 months following external beam radiotherapy. This encouraged Cotlar and his colleagues,[18] and prompted them to suggest, as the proper course of treatment, wide surgical excision coupled with postoperative irradiation to both the local site and regional lymphatics.

Our own experience with trabecular carcinoma comports with the currently available literature. We have found the disease to be a cutaneous malignancy of the elderly that presents on the head and lower extremities. We have also found this tumor to be rather aggressive because of its propensity for local recurrence and regional and distant metastases. Still, the appearance of a local recurrence or metastasis does not necessarily presage death, as only about 25% of patients will succumb to the disease.

However, we do treat trabecular carcinoma with great caution and attention. There have been reports of this malignancy arising in conjunction with a squamous carcinoma,[24,25] a basal cell carcinoma,[26] and in the scar of a previously excised basal cell carcinoma of the nose.[27] It has also been described as a source of superior vena cava obstruction,[28] fulminant hepatic failure from liver metastases,[29] malignant pleural effusion,[30] and small bowel metastases.[31] We therefore use a team approach: the lesion is surgically excised with adequate margins, and radiotherapy is employed at the site of both the primary tumor and for any recurrence or metastasis. While we have had no experience with management by chemotherapy, recent positive reports about this modality[7,32,33] may provide a key for the treatment of the metastatic component of the disease.

TABLE II. Summary of Reported Cases in the Literature

Author	Year	No. Patients	Local Recurrence	Regional Lymphadenopathy	Metastases	Deaths
Murphy et al	1988	4	75%	75%	50%	25%
Raaf et al[16]	1986	80 (4 personal, 76 literature)	36%	53%	28%	25%
Cotlar et al[18]	1986	147 (8 personal, 139 literature)	37.5% 30%	50% 50%	37.5%	25% 18%
Meland and Jackson[19]	1986	23	43%	52%	26%	35%
Pajor et al[22]	1986	10	—	—	—	30%
Sibley et al[21]	1985	43	30%	65%	40%	33%
Goepfert et al[20]	1984	41	—	79%	—	—
Kroll and Toker[7]	1982	23	39%	22%	26%	22%

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FROM THE LIBRARY

PRESENT STATUS OF THROMBOLYTIC AGENTS

Recently interest has been focused on the possibility of using active thrombolytic agents to produce a dissolution of intravascular clots. It is generally recognized that if a safe, effective substance, standardized in its action from one lot to another and capable of producing a uniform response, can be evolved, it will represent a new experimental and therapeutic tool of great importance. Today several preparations are available which are suitable for investigational use. The most widely used are those which contain streptokinase, urokinase, a mixture of streptokinase and plasmin, or, in the views of other, plasminogen-activator combinations, each possessing varying degrees of activity. Many workers have been experimenting with their use in animals and in man, and new knowledge has been gained from these studies. Nevertheless, they still remain unsatisfactory for widespread clinical use for a variety of reasons. These should be known to the clinicians who, without experimental facilities, might be induced to use them in their practices.

The materials which have been marketed in the past have not been adequately standardized, either in terms of fibrinolytic activity or in their individual components. They have been capable of producing pyrogenic and other unpredictable and undesirable reactions. The problem is rendered more complicated by the variability in the patient's blood, where the antistreptokinase and antifibrinolytic titers range widely and may be far more than enough to counteract the amount of fibrinolytic preparation which may be safely injected for therapeutic purposes.

All of this is not intended to discourage investigation in this area. On the contrary, such investigation appears to hold promise of greatly enlarging our knowledge of one of the fundamental processes of biologic life, the fibrinolytic-antifibrinolytic balance, and it may well contribute a widely used therapeutic technic. Every encouragement should be extended to those who are embarked on these fact-finding missions.

At this time, however, clinicians are being overexposed to suggestions and intimations of therapeutic effectiveness and safety which are not based on solid basic or clinical evidence. These agents should be regarded as research and experimental tools until they are sufficiently purified and standardized, until we have secure methods of determining the correct dosage available to all who would use them, and until our knowledge as to precise indications and contraindications has reached a more sophisticated level.

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LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Low-dose subcutaneous heparin therapy

TO THE EDITOR: I read with more than great interest the commentaries by Rooke[1] and Mendelsohn[2] as well as the research paper by Hollander et al[3] on the use of heparin in the prevention of thromboembolism in the July issue of the *Journal*. I was more than a little surprised that in none of the aforementioned was reference made to my original highly successful subcutaneous low dose heparin method.[4-6] I am pleased to see, however, that all of the above find that my method is most reliable.

I recommend the low dose subcutaneous method chiefly in operative patients but found it valuable in others as well.

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Cocaine

TO THE EDITOR: We read with interest Dr Gay's July 1989 commentary on cocaine,[1] and while we agree with the need to treat toxicity aggressively, we take issue with some of the measures advocated.

The author recommends, for example, that "dantrolene should be stocked in the event of malignant hyperthermia." The use of dantrolene to treat cocaine-induced hyperthermia is not indicated, in our view. Dantrolene is an inhibitor of skeletal muscle contraction used in the treatment of malignant hyperthermia, which is a rare complication of general anesthesia seen in genetically predisposed patients. Malignant hyperthermia has never been reported in association with cocaine use. The hyperpyrexia associated with cocaine intoxication, on the other hand, is a result of hypermetabolism combined with severe peripheral vasoconstriction and probable hypothalmic alterations in thermoregulation.[2] Aggressive cooling measures, as the author mentions, are the mainstay of treatment; dantrolene has no proven role.

In addition, we disagree with the author's recommendation for bicarbonate infusion for metabolic acidosis. While it is true that acidosis depresses myocardial function and potentiates ventricular dysrhythmias, the use of bicarbonate remains very controversial.[3] Studies have shown that intravenous bicarbonate may further impair depressed myocardium and may paradoxically decrease arterial and intracellular pH.[4-6] Therapy should be directed at the underlying metabolic derangements.

Lastly, we wish to point out the hazards of urinary acidification to enhance renal excretion of cocaine. Rhabdomyolysis is a well-described complication of cocaine intoxication[7] which may lead to acute renal failure, particularly in the setting of an acid pH.[8] Given the relatively short serum half life of cocaine together with the potential danger of myoglobinuric renal failure, administration of NH_4Cl is contraindicated.

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The new health code regulations concerning postgraduate medical education in New York State

TO THE EDITOR: The article by Steven C. Reiner in the August 1989 issue, "The impact of new code regulations on postgraduate medical education in New York State," may leave your readers believing that the reforms involving residency training in New York State are an aberrant development, out of line with thinking elsewhere in the nation.[1] This is incorrect. National residency boards are adopting similar standards to be applied nationwide.

One should keep this in mind in evaluating Dr Reiner's effort to present an objective evaluation of the New York reforms, their probable impact on patient care, residents' education, and New York's response.

In the section on sleep deprivation and the quality of care, Dr Reiner cites a number of studies which failed to demonstrate impairment of cognitive and motor functions of residents. This suggests to me that the testing methodology must be flawed rather than that sleep deprivation has no effect on performance. Certainly the common sense experience of every reader of this *Journal* will corroborate that 36 hours of demanding activity with an average of 2.4 hours of sleep (Greater New York Hospital Association Survey) every third or fourth night for months on end would compromise function adversely.[2]

Dr Reiner made no mention of the psychological and social effects of the current residency training that may lead to depression, marital breakups, suicide, and deep resentment toward patients. Further, biological studies on the diurnal rhythm have clearly demonstrated that the biological clock requires the winding which is provided by sleep. Finally, is it not surprising, if sleep deprivation has no adverse effects, that airplane pilots and long distance bus drivers are restricted in their hours of continuous service? Think how our airfares might be reduced if the captain and crew of our next air flight were in the 32nd hour of continuous piloting instead of just beginning eight hours of restricted time on.

In the section on perspectives on reform, Dr Reiner cites some affirmative comments on the new regulations but, in the main, suggests that they impair residents' education, produce major disruptions in patient care, and cause increased costs of medical care. Some of Dr Reiner's evidence is outdated. The comments by the American College of Physicians had reference to a preliminary formulation in the report which was later abandoned. (In testimony to the New York State Department of Health Ad Hoc Advisory Committee on Emergency Services, on August 17, 1987, the American College of Physicians criticized the June 2, 1987, proposal of the ad hoc committee, which proposed a 16-hour workday limit. This was revised and made more flexible in the subsequent and final report of the ad hoc committee which recommended the 24-hour scheduling limit, and an average of 80-hour weeks over a four-week period.) At the present time, both the Accreditation Council for Graduate Medical Education (ACGME)[3] and the Residency Review Committee for Internal Medicine have come forward to urge an 80-hour work week.[3] These groups and the Pediatric Residency Review Committee call for one full 24-hour period off each week, and other national organizations as well as states are now actively debating reforms.[3]

It is an error to believe that one tragic case led to this tremendous local and national debate on residents' working conditions and supervision. In New York State there have been many other instances of adverse outcomes for hospitalized patients in which overworked, unsupervised house officers have played an unhappy and unjustified role. No doubt the same experience is found in other states.

Dr Reiner concludes in the section on implementing reform that some type of reform is due, but questions how it should be done. New York State has provided direct funds for ancillary personnel and already many are in place and others are in training. This reform alone is likely to improve residents' care of patients and their education. It is suggested that the best solution to the new regulations is to increase the number of house staff. In fact, this has been done, particularly in internal medicine, where the number of PGY-1 residents matched increased from 803 in 1987 to 900 in 1989. In contrast, the number of residency positions in medicine nationally declined from 5,341 to 5,123. If, as Dr Reiner suggests, the answer to reasonable regulations is to increase the number of residents, then residents are being used for service, not for education. This should make us all think again about the purposes of graduate medical education!

The disruption of the continuity of patient care has often been cited as a major drawback of the new regulations. This, in part, is a red herring. Unless a resident is to be on call 24 hours every day, continuity of patient care will necessarily be interrupted at some point. Nevertheless, on the basis of discussions with program directors in several of the major disciplines, the commissioner of health has introduced flexibility into the regulations which allows time for the orderly transfer of information from those residents going off duty to those coming on. Also, it was always recognized that a resident in the midst of caring for a critically ill patient, such as one experiencing diabetic ketoacidosis, or in the middle of an operation, would be expected to continue the care of the patient as indicated.

Finally, Dr Reiner has reservations about the "surprising speed" by which the regulations were enacted and also implies a question about the "nine distinguished New York physicians" who were members of the committee chaired by Dr Bertrand Bell. The Ad Hoc Advisory Committee on Emergency Services' printed report[4] appeared in 1987 after one year of frequent meetings and study, and after extensive public hearings and wide discussion with national as well as New York-based leaders in medical education. The State Hospital Review and Planning Council, which enacted the regulations, also had public hearings. The July 1989 implementation date followed a long term process.

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3. Residency Work Hours and Supervision, Changes in Special Requirement, Medical Education Group, Accreditation Council for Graduate Medical Education, Chicago, Ill, March 1989.
4. Final report of the Ad Hoc Advisory Committee on Emergency Services, Albany, New York State Department of Health, October, 1987.

In reply. Mr Slocum states that the failure to demonstrate impairment of function of residents after sleep deprivation can be explained by the testing methodology's being flawed. Although this debate continues, the fact is it has proven impossible to demonstrate that an otherwise healthy, energetic, and vigorous house staff member, motivated to learn and perform, will deteriorate as a result of 24 or 36 hours of continuous duty. Since I assume that a major motivation on the part of the New York State Department of Health in implementing these regulations was to improve quality of care, it would behoove it to prove that quality of care does indeed deteriorate after 24 hours prior to implementing its rigid regulations. Mr Slocum refers to the common sense experience of every reader that being on call every third or fourth night for months would adversely compromise function. The medical literature is replete with hard data refuting many common sense experiences. For example, common sense tells us that the treatment of severe hypertension with thiazide diuretics should reduce the risks of heart attacks. Indeed, after careful study the opposite has proven to be true.[1] Common sense has also suggested to us that thalidomide might be a good sleeping pill, that interferon might cure cancer, and that coronary bypass surgery always prolongs life. Such assumptions have been proven incorrect. There is no doubt that sleep deprivation and overwork make residents an unhappy, unsociable, and perhaps divorce-prone group. Indeed, as I mentioned, reform of working conditions is necessary. But it is a far cry from a blanket statement that the reduction of resident work hours will result in an automatic improvement in quality of care. This is a supposition that the health department has made, placing health care institutions in financial jeopardy, and the quality of medical education at risk.

Mr Slocum states that “it is suggested that the best solution to the new regulations is to increase the number of house staff.” While this may indeed be a solution to the problem of compliance with state regulations, it is not equivalent to an improvement in medical education. As I stated in my article, there is clearly a relationship between the volume and diversity of patient and didactic material that residents are exposed to and their ultimate competency as physicians. It remains to be shown that diluting the house staff experience is an avenue towards improved quality.

Mr Slocum also states that the number of residents matched in New York State in internal medicine programs increased in 1989. Although this may be a local aberration, the facts show that there is a continued national decline in matching in all primary care specialties including internal medicine, pediatrics, and family practice since 1987.[2] In 1989 3% fewer positions matched in the National Resident Matching Program in internal medicine than in 1988. Perhaps New York State is robbing Peter to pay Paul. If the state health department theory is correct, we should notice decreasing quality nationwide and an increasing quality of house staff performance in New York State.

The thrust of my article was to emphasize that those who should be deciding residents’ work hours, residents’ work rules, the presence or absence of resident impairment, and the residents’ stress load should indeed be the medical educators. Most of the rest of the nation seems to be headed in this direction, and only New York State (and perhaps Massachusetts) has decided to imprison the medical education process in rigid rules. In California, a careful, cooperative process among the state’s ten medical schools and the department of health has begun a *four year* process to “implement their own voluntary reforms of resident work hours.”[3] Under the California plan, a committee of representatives from the state’s schools has begun a reform process expected to be implemented by July 1993. This plan leaves room for interpretation, suggests caps on residents’ work hours, but leaves considerable flexibility, including a provision that each institution define the limit on the number of continuous hours worked.

My major objection to the new health code regulations is with the process more than the content. No one would disagree that reform is due. However, it never fails to astonish me that the New York State Department of Health has a solution for each and every health care problem in the state. It produces a never ending stream of rules and regulations against the backdrop of a medical care system that is clearly in chaos. I stated at the conclusion of my article that the state has begun the largest uncontrolled experiment in medical education ever attempted. I challenge the New York State Department of Health to return to us follow-up studies which demonstrate that as a direct result of new 405 regulations, the number of house staff errors has decreased, the overall morbidity and mortality due to physician error in New York hospitals has decreased, the scores of residents on national or specialty certifying board examinations have increased, and that the divorce rate, suicide rate, and, perhaps, the reading level of physicians’ children have all been improved as a result of this far reaching legislation. After all, this is the process by which all medical experimentation is evaluated. I suggest that the state department of health propose a one- or two-year period of study for its new health code regulations. Failing to demonstrate an improvement in overall quality of house staff care, and diminished house staff error, this expensive experiment should be terminated. Control of the medical education process should be wrested from the hands of the health bureaucrats and returned to the deans, program directors, and house staff representatives at the local level.

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Alcohol and public health: Implications for New York State

TO THE EDITOR: Congratulations on the extensive coverage which the *Journal* provided to the New York State Department of Health’s report, “Alcohol and Public Health: Implications for New York State.”[1-3]

As the report documents, alcohol-related illnesses and injuries account for an estimated 6,500 deaths in the state each year, making them the fourth leading cause of death. And although alcohol is believed to be involved in up to 50% of all hospital admissions, it is diagnosed on only 6% of the discharges.

By calling attention to this topic, the *Journal* is increasing the medical community’s awareness of the extent of “the alcohol connection” to those problems. Through its Hospital Intervention Services Project, the Division of Alcoholism and Alcohol Abuse has contracts and 18-month pilot projects with 11 hospitals in the state to improve their abilities to diagnose alcohol problems and refer patients for appropriate treatment.

The division also has recently agreed on an “Action Plan” with the state department of health to intensify the involvement of state and local health authorities in improving alcoholism services.

Dr David Axelrod, Commissioner of Health of the State of New York, has demonstrated his leadership in this area, and we look forward to working with all sectors of the medical community to improve the state’s health and safety by understanding “the alcohol connection.”

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Birth outcomes in New York State (Part II)

This article is the second part of a two-part series that examines various birth outcomes in New York State. It includes those maternal variables such as age, race, socioeconomic status, level of education, parity and absence or inadequacy of prenatal care that have been associated with poor birth outcomes.[1]

The study population for this analysis consisted of 725,775 live births recorded in New York State from 1983 through 1985. Logistic regression analysis was used to confirm the impact of the maternal variables on birthweight (low birthweight is defined as less than 2,500 grams and normal birthweight defined as 2,500 grams or above). Odds ratios were calculated relative to the most favorable subgroup within each variable; the most favorable group was assigned the odds ratio of one. The odds ratio represents the relative risk of those exposed to a factor (eg, inadequate prenatal care) compared to those not exposed (eg, adequate prenatal care). An odds ratio of one would indicate no difference; the larger the ratio, the greater effect that variable has on the outcome.

Table I summarizes the results of the logistic regression analysis. Level of prenatal care was determined by considering the date of first visit, number of visits, and length of gestation.[2] Prenatal care was then defined as adequate, intermediate, or inadequate using these criteria. Inadequate care was associated with more than a 100% increase in the risk of delivering a low birthweight infant when compared to the groups receiving adequate care. The odds ratio of 2.08 was adjusted for differences in maternal age, race, education, and parity. The other odds ratios affirm the risk of low birthweight infants among women with no previous live births, the poorly educated, younger and older women and nonwhites.

TABLE I. Adjusted Odds Ratios for Low Birthweight, New York State, 1983-1985

Level of Prenatal Care	
Adequate	1.00
Intermediate	1.34
Inadequate	2.08
Maternal Age	
10-19	1.78
20-24	1.13
25-29	1.00
30-34	1.14
35 and over	1.31
Race	
White	1.00
Nonwhite	2.04
Previous Live Births	
0	1.26
1	1.00
2	1.07
3 or more	1.17
Maternal Education	
0-11 years	1.89
12 years	1.32
13 and over	1.00

Live Birth/Pregnancy Ratio

During 1987, 64.8% of New York State residents reported pregnancies which resulted in live births. The 1987 ratio of live births to pregnancies was higher for upstate residents (73.3%) than for women residing in New York City (56.7%). There has been little change in the state live birth to pregnancy ratio since 1977.

Maternal Age

In contrast to the general population, there has been a decrease in the live birth to pregnancy ratio in the adolescent population in New York State from 47.7% in 1977 to 44.3% in 1987.

During 1986, 11% of the births among adolescents 17 years of age and younger were among those who had previously given birth; 19% were among those who had previously been pregnant. Of births among teens 18-19 years of age, 25% were women who had already given birth, 36% were those who had previously been pregnant. There is little difference in pregnancy histories between adolescents from New York City and those from the rest of the state.

Between 1977 and 1987, the pregnancy rate among teenagers in the state increased from 41.1 per 1,000 to 48.8 per 1,000. This increase occurred in both New York City and the rest of the state.

Maternal age and race have been associated with low birthweight infants. In 1987, the low birthweight rate was 10.1% for births among mothers 19 years of age and younger compared to a rate of 7.1% for all ages. Nonwhite mothers under age 20 delivered 12.8% of their babies weighing under 2,500 grams.

Prenatal Care

The established relationship between prenatal care and pregnancy outcomes seen in Table I and in the literature suggests that improvement in prenatal care to an adequate level would result in a general improvement in the health status of newborns. However, many New York State women continue to receive inadequate prenatal care. Data for the period 1977 to 1987 showed no significant increase in the percent of pregnant women receiving prenatal care in the first trimester. At the same time there was an increase in women who did not begin prenatal care until the third trimester or received no prenatal care.

The New York State Department of Health has established a service network across the state to provide prenatal care and nutritional support to improve pregnancy outcome. This intervention strategy seeks to identify those areas and people having the highest rates of poor outcomes and to target activities to those areas. The goal of such efforts is to provide preventive service, family planning, prenatal care and nutritional support to improve the health status of all newborns.

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Residential and home health care for people with AIDS and HIV-related illnesses

Residential Health Care

The clinical profile of AIDS and HIV-related illnesses has evolved from a relatively short term, fatal trajectory, punctuated by episodes of acute illness, to a more extended course of incurable illness, often characterized by months of physical and mental disability prior to the terminal stage. Through the AIDS Intervention Management System (AIMS), the New York State Department of Health has documented the need to manage the chronic manifestations of HIV infection in residential health care facilities (RHCFs).

RHCFs have not been widely used by people with AIDS and HIV-related illnesses. Only 3.6% of 1987 AIDS hospital discharges in New York State were to chronic care or other nonacute facilities. The Coler and Goldwater facilities, operated by the New York City Health and Hospitals Corporation, provide 68 specialty hospital beds offering care similar to that in skilled nursing facilities (SNFs). These facilities have documented the complex care need of the long term HIV patient with costly medication requirements, higher nurse staffing patterns and drug abuse treatment needs.

In a 1988 20-hospital study of people with AIDS or HIV-related illnesses, almost one quarter of clinically stable patients hospitalized for ten days or more were identified as needing subacute care in RHCFs upon discharge. Of these, 16.8% needed care in SNFs and 7% in health-related facilities (HRFs). People with AIDS and HIV-related illnesses are significantly different from typical long term care clients; they are younger (average age 35); much more likely to be drug users, without a suitable home or any home at all; and require greater and more complex clinical and supportive care. Advances in medical care strongly suggest that the proportion of all people with HIV-related illnesses requiring long term care will increase.

In response to the identified need for AIDS-specific residential health care, the State Hospital Review and Planning Council passed regulations in June 1988 to establish HIV-specific skilled nursing and health-related facilities and dedicated HIV beds in existing facilities. The regulations for the operation of these RHCF beds amend the existing code for nursing home and health-related facilities with specific requirements to assure appropriate care for people with HIV-related illnesses.

Like traditional RHCFs, AIDS-RHCFs will provide rehabilitative care involving active treatment of coexisting illnesses and disabilities and palliation for those who choose relief of pain and mental anguish. However, AIDS-RHCF care will differ from traditional RHCF care in several ways. Case management services will generally be provided in cooperation with an AIDS center. AIDS-RHCFs must also provide or make formal arrangements for special services, as needed, to include substance abuse, mental health, and pastoral counseling. Counseling and supportive services will be made available to assist staff members with problems related to caring for people with HIV-related illnesses. Also, staff are expected to attend inservice and continuing education programs at least every three months which address the medical, psychological, social problems, and care needs specific to HIV disease.

As of June 1989, the New York State Department of Health received applications for 732 AIDS-SNF beds from eight applicants. A total of 182 of these have been approved, and 44 have been opened at the Terrance Cardinal Cooke Health Care Center. An additional six AIDS-SNF beds have been opened at the Monroe Community Hospital in Rochester, New York. The department has received applications for 509 AIDS-HRF beds from four applicants. A total of 165 of these beds have been approved.

Several other potential applicants have approached the Department of Health in anticipation of developing increased bed capacity in existing AIDS-SNFs and HRFs in the near future.

For each AIDS-RHCF application, the department, the applicant, and the Medical Care Facilities Financing Agency are working together to overcome barriers to capital financing, property acquisition, community acceptance, and program development. These barriers are significant because few applicants have experience in providing residential health care to people with the need for frequent medical oversight and intervention and drug treatment.

Home Health Care

Care to people with HIV-related illnesses is largely provided in hospitals and at home. Certified home health agencies made 10,800 nursing visits to AIDS patients in 1987. Ninety percent of these visits occurred in New York City. As of September 1988, the New York City Human Resources Administration, the Visiting Nursing Service of New York Inc (VNS), and other certified and licensed home care providers were providing home care services to approximately 750 people with AIDS and HIV-related illnesses.

Several specialized home care programs for those with HIV-related illnesses have developed, most notably the VNS of New York's AIDS home care services under arrangement with New York City's Human Resources Administration. Intensive staff training for both nursing, home health aides, and home attendants has been necessary and essential for all home care programs serving people with HIV-related illnesses.

In addition, a Long Term Home Health Care Program specifically for people with AIDS and HIV-related illnesses was enacted by legislation passed in September 1988. The department is currently drafting regulations and defining the process of establishment in order to implement this first AIDS-specific home care program. Patients serviced by the AIDS Home Care Program will receive a comprehensive coordinated array of home care services, including some waived services not traditionally covered by Medicaid.

AIDS and HIV-related illnesses exacerbate the demands for labor experienced by the home health care system. A study of the home care resource needs of people with AIDS and HIV-related illnesses was conducted in 1987 by the Home Care Association of New York State for the department. It was determined that AIDS patients need 13% more nursing time per visit than terminal cancer patients and 29% more than the average home care patient. As a result of that study, the Department of Health established separate payment rates for nursing services provided to people with AIDS. Nursing rates were set using regional service prices increased by a case mix adjustment factor to reflect the additional resource utilization needed in home care treatment of AIDS patients. The New York City average nursing visit rate for AIDS patients served by certified home health agencies was increased in 1988 by 30%.

The AIDS center concept has been followed by other innovations and resource allocation in AIDS health services delivery including enhanced outpatient reimbursement, maternal pediatric services, residential health, and home health care. These advances are a critical extension of the AIDS center concept of a coordinated continuum of care supported by hospital-based case management. Furthermore, the effectiveness of AIDS centers is dependent on the availability of a full range of services. Quality and utilization monitoring similar to that in AIDS centers is carried out to evaluate resources and quality of care in each setting.

Cancer incidence among NYC Hispanics 1982–1985

Hispanics constitute a significant segment of the New York City population. The incidence of cancer among this population has not been previously documented. This article compares cancer incidence among Hispanics with that among white non-Hispanics in the city.

In the 1980 US Census, Spanish origin was determined by a question that asked each respondent to identify whether or not they were of Spanish origin or descent.[1] Individuals selecting any one of the four Spanish categories (eg, Mexican, Puerto Rican, Cuban, or other Spanish) are collectively referred to as individuals of Spanish origin or Hispanics.

Approximately 1.4 million individuals of Hispanic origin or descent were identified among New York City residents in 1980, comprising 20% of the city's population.[2] Only five cities in the United States have populations larger than 1.4 million, the number of Hispanics residing in New York City. Most Hispanics living in New York City immigrated after World War II; until 1965 a large majority were Puerto Rican. Immigration from countries in South and Central America increased rapidly by 1970 because of significant changes in the immigration laws in 1965.

The data source for this article was the New York State Department of Health Cancer Registry. Cancer cases diagnosed during 1982–1985 among New York City residents were designated as Hispanic if they were of any race with Spanish origin reported as either Puerto Rican, Mexican, Cuban, Central, or South American or other. These cases were tabulated by sex, age, and cancer site and they form the basis for this article. An earlier study was conducted to validate the accuracy of information contained within the New York State Cancer Registry concerning place of birth and country of origin among Puerto Rican-born residents of New York City. The study found over 90% of the information within the Cancer Registry to compare with that contained on hospital records.[3]

Cases of cancer diagnosed among white non-Hispanics (whites) were tabulated in an identical way and used as a comparison group. Whites were defined as individuals of white race and not of Spanish origin or of white race with Spanish origin recorded as unknown.

Revised population counts were calculated to address the problem of the 1980 US Census under-enumeration of minority residents.[4] These revised counts were used to calculate age-, sex-, and site-specific average annual rates for both whites and Hispanics. Sex and site-specific rates were then age-adjusted to the 1970 US population.

For major cancer sites, the average annual age-adjusted cancer incidence rates for Hispanic and white males in New York City were examined (Fig 1). The overall cancer incidence rate per 100,000 for Hispanic males (319.7) was substantially lower than for white males (392.1). In particular, colon cancer in Hispanic males was nearly half that of white males (24.8 vs 46.7), as was cancer of the rectum (13.0 vs 20.2). Malignant melanoma was found to be six times lower in Hispanic males (1.4 vs 8.7). With the exception of buccal cavity and pharynx as well as larynx, Hispanic rates were substantially lower for the smoking-related sites including the lung and bronchus (51.5 vs 73.2), urinary bladder (16.8 vs 31.1) and kidney (5.3 vs 10.2). The rate of stomach cancer was found to be higher in Hispanic males (18.7) as compared to white males (13.4).

A lower incidence rate in Hispanic females was observed for all sites combined (224.7 vs 317.6) (Fig 1). Similar patterns to those observed in males were present in females for stomach, colon, rectum, and malignant melanoma. Lower rates for Hispanic females were observed for the smoking-related sites of lung and bronchus (14.1 vs 31.8), urinary bladder (4.4 vs 8.4) and kidney (2.6 vs 5.0). The rate for cancer of the gall bladder was higher in Hispanic females (3.4) as compared to whites (2.0). Cancers in female sites were generally lower for Hispanics, with

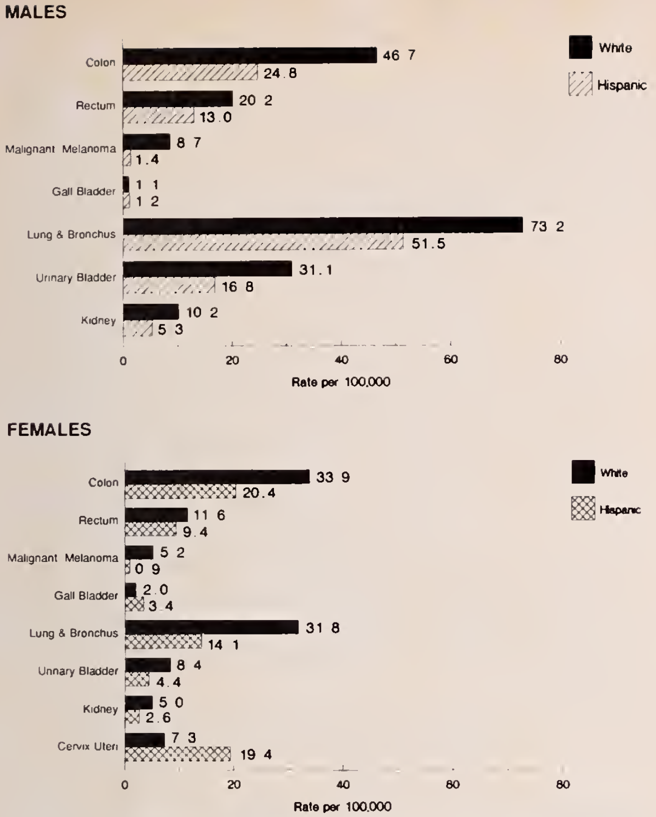

FIGURE 1. Average annual cancer incidence per 100,000 in whites and Hispanics, New York City, 1982–85 (from New York State Cancer Registry).

the exception of cancer of the cervix uteri (19.4 vs 7.3).

Little has been published on the cancer incidence among Hispanics. However, available data indicate generally lower cancer incidence rates in Hispanics than in the white non-Hispanic population of the United States. Colon and rectal cancer have previously been reported as lower in Puerto Rican-born residents of New York City than in other whites residing in the city.[3] The low incidence of malignant melanoma among Hispanics observed in this study is also seen in New Mexico and Los Angeles, with rates for Hispanics six times lower than in whites in these areas.[5,6]

Other studies have also reported a deficit of smoking-related cancers in Hispanics and suggest that patterns of smoking behavior may account for the current differences.[5,7–11] Department of Health survey data indicate that cigarette smoking is less prevalent among Hispanics than among non-Hispanic whites.[12] However, the sample was based on a small number of respondents. There is some evidence that suggests smoking rates may be increasing in Hispanic men.[10] Stomach cancer rates have been consistently higher in Hispanics than in white populations, including Puerto Rican-born residents of New York City and Hispanics in Los Angeles and Denver.[4,5,8,9]

The high incidence rate for invasive uterine cervical cancer has been observed in other Hispanic populations in the United States and is consistent with that observed in New York City.[5,8,9,13] It is the most common malignancy in women in Columbia as well as in Hispanic women in New Mexico[7] and Mexican-born immigrants residing in Los Angeles.[8] It is the second most common cancer in Hispanic women in Los Angeles County.[8] The known risk factors for uterine cervical cancer are low socio-economic status, marriage, or sexual activity before age 20 and multiple marriages or sexual partners.[14] For example, black women in the United States have a two-fold greater risk than whites; these racial differences have been attributed to socioeconomic factors. Hispanic women in New York City had substantially lower socioeconomic status in 1980 than women in the city as a whole and an average of three children by age 35–44 (2.2 for all New York City women).[2] The total population of Hispanic women were an average of 7.4 years younger than all women living in New York City. In addition, they were 44% more likely to have children under the age of 18 and out of wedlock. Differing practices in hysterectomy may account for some of the increased risk. For example, fewer hysterectomies are performed on Mexican-Americans than on whites in Los Angeles County.[8]

Cancer incidence in Hispanics living in New York City is similar to published accounts of cancer incidence in other Hispanic groups throughout the United States. In general, cancer incidence rates among this population are lower than among whites residing in the city. Rates for most smoking-related sites are lower among Hispanics, most likely the result of different smoking habits. Rates for cancer of the colon and rectum, urinary bladder, kidney and renal pelvis and malignant melanoma of the skin are well below those for whites. Stomach cancer rates in men and women and cervix uteri and gall bladder rates in women are substantially higher.

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Risk factors for teenage suicide

More than 6,000 young persons, aged 15 to 24 years, intentionally kill themselves each year in the United States, making suicide the second leading cause of death among this age group. Occasionally, multiple youth suicides occur in groups relative to time or location; these events have been called suicide clusters. Authors Davidson, Rosenberg, and Mercy, from the Centers for Disease Control (Atlanta, Ga), and colleagues investigated two suicide outbreaks and evaluated the risk factors for teenage suicide during a suicide cluster (*JAMA* 1989; 262:2687–2692).

One prominent hypothesis for youth suicide clusters has been "that the exposure of susceptible young people to an actual suicide or the idea of suicide leads some of those young people to commit suicide." Direct exposure to suicide, defined as firsthand knowledge of the suicide victim, or indirect exposure, through word of mouth or media coverage of the event, may produce an imitative response.

In this matched case-control study, the authors did not find greater exposure to media accounts of suicide among suicide victims, compared to control youths. In addition, direct exposure to suicide was not necessarily more common among case versus control subjects. There were, however, several variables that pertained more often to the suicide victims than to the control population. These findings resulted from the data obtained through questionnaires administered to parents and parent interviews.

An evaluation of the two suicide clusters revealed common characteristics: 46% of the suicidal acts occurred in the afternoon, with none occurring between 12:00 AM and 6:00 AM; seven of the 14 suicide victims shot themselves, five died from carbon monoxide poisoning, and two deaths were the result of hanging; the weapons in all the shootings were guns kept in unlocked storage areas; 54% of the suicides occurred in a car or garage; in both clusters, all the suicide victims after the first were aware of the preceding suicide.

Characteristics shared by the case subjects that are known to increase the probability of suicide included previously threatening or attempting suicide, having been hospitalized for treatment of a mental illness or substance abuse, and losing or anticipating the loss of a girlfriend or boyfriend.

Other important variables that distinguish youths vulnerable to suicide from the "classically depressed teenager" include:

- experiences with violence or force intended to harm oneself or others
- having physically hurt themselves other than in suicide attempts
- having previously been physically violent toward others
- having experienced the death of a relative or close friend
- having been arrested or charged with an offense
- seeming to become emotionally hurt or offended relatively easily
- changing residences more often and attending a greater number of schools
- having had more than two adults serve as parents.

It is the authors' hope that these findings might help identify youths who are at increased risk of suicide. Although exposure to media coverage of teenage suicide was not a factor differentiating case from control subjects in this study, the effects of direct or indirect exposure to suicide could have a greater impact on a youth already more vulnerable to suicidal thoughts and actions. Sensationalized accounts of suicide that build up the victims' importance, and memorial services held at schools, which focus the attention of classmates and peers on the event, may have a role in imitative behavior. The authors believe that it is "prudent to curtail the excesses in public exposure to suicide."

Metaproterenol inhaler taken off market

The recently reformulated, prescription metered dose inhaler, Alupent®, containing the asthma drug metaproterenol, has produced an alarming rate of adverse side effects. In response to reports of coughing with a choking or gagging sensation, experienced primarily following the first dose of the reformulated drug by users of the old formulation, the drug's manufacturer—Boehringer Ingelheim (Ridgefield, Conn)—is removing the new formulation of Alupent® from the market.

While the active ingredient in both drugs is the same, the manufacturers replaced the suspending agent sorbitan trioleate with soya lecithin in the new formulation. Alupent® containing soy lecithin has been prescribed in other countries for several years, and the cause of the adverse reactions to the reformulated product distributed in the US has not yet been determined. In addition to reports of coughing and gagging, some patients have complained that the new formulation is not as effective against their asthma and, in some cases, may even exacerbate their symptoms. None of the reported adverse reactions has been fatal or has required a hospital stay for treatment.

Boehringer Ingelheim is removing all lots of the new formulation from the market. Available lots of the old formulation are safe, and the manufacturer plans to reintroduce the old Alupent® formulation into the US market.

Lung cancer rates decreasing for young people in US

"Although overall mortality from lung cancer continued to increase through 1986, the latest year for which data are available, recent decreases in both incidence and mortality have been reported among young white men" (*J Natl Cancer Inst* 1989; 81:1568–1571). This conclusion is derived from a recent study of lung cancer incidence and mortality data by race, sex, age, and cohort year of birth, performed by the National Cancer Institute. In addition to the substantial decreases documented for

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young white men, modest reductions were also evident for young black men as well as for young white and black women.

Authors Devesa, Blot, and Fraumeni, Jr, attribute these optimistic findings largely to reductions in smoking prevalence among young people. For example, in the 1950s, proportionally more whites smoked than blacks, whereas now, smoking is more prevalent among black men than among white men. Similarly, during the period between 1950 and 1986, the lung cancer mortality rate for nonwhite males surpassed the rate for white males.

For the decade from 1975 to 1985, the greatest increases in lung cancer mortality affected the 65 and older age group. Men aged 45–54 years exhibited a declining mortality rate of 7%–10%. However, the most striking decline in lung cancer mortality—29%—was seen for white men aged 35–44 years. For that same age group, black men experienced a 14% decrease, with a 5% drop for white females and 9% for black females.

The authors contend that since lung cancer survival rates have remained low throughout the study period, the declining mortality rates indicate a decrease in lung cancer incidence for these populations. These findings emphasize the importance of antismoking efforts: "targeting young people for prevention programs has the potential to result in major reductions in incidence and mortality from lung cancer and other smoking-related diseases."

Too many surgeons for too few operations?

Health policy experts have suggested that the number of surgeons in the United States is now so much greater than the demand that surgeons, primarily in over-staffed urban area, may not be performing sufficient procedures to maintain their skills at peak proficiency (*New York Times*, November 7, 1989, pp C1,6). Professional organizations of surgeons dispute this observation, citing the need for more surgeons in rural areas and in the inner cities, and claiming that current caseloads do not threaten surgical competence. They do agree, however, that a certain minimum amount of surgical experience is essential to maintain skills.

Two main factors are contributing to the increasingly competitive market for surgeons: the ever-growing popularity of surgery as a specialty, with its promise of prestige and financial reward, and the dwindling numbers of operations. Conditions involving the stomach, intestines, kidneys, gallbladders, heart, and blood vessels that were formerly treated with major forms of surgery are now being treated with less invasive methods. For example, drugs being used to treat peptic ulcers have virtually eliminated the need for surgical treatment. Other new drugs are proving effective in treating coronary artery disease, thus reducing the need for coronary artery bypass surgery.

Advances in medical technology have forced many surgeons to extend their expertise to include less invasive, nonsurgical therapies. Endoscopy is now the primary invasive method for diagnosing and treating stomach and intestinal disorders such as ulcers, polyps, and tumors. Laparoscopy is now a common method for diagnosing and treating pelvic and intestinal disorders and is gaining acceptance as a method for gallbladder removal. "For the last two years, endoscopy experience has been required in all general surgery training programs, and seminars to teach older surgeons these new tricks have flourished."

Lithotripsy has replaced surgery for kidney stone removal in many cases. Thin-needle biopsy, of breast masses for example, has proven an effective diagnostic alternative to surgery. Balloon angioplasty has increased the role of radiologists in treating blocked vessels and coronary arteries.

As surgical subspecialties increase, general surgeons in particular feel the pinch of the decreasing need for invasive procedures. "Studies have shown that the number of operations done by the average general surgeon dropped 25 percent from 1982 to 1985." Other studies suggest that for some complicated surgical procedures, improved results and decreased mortality, by as much as 40%, are associated with going to a surgeon who performs such operations relatively often.

In regions with too many surgeons, the options being chosen by surgeons are to limit their practices to one or a few procedures that they can perform frequently and more skillfully, to expand their practices to include more primary care, or to become proficient in endoscopic and other less invasive techniques that they can offer to patients as therapeutic alternatives.

Promoting bicycle helmet use in children

Five hundred to 600 children die each year in the United States due to bicycling injuries, with many of these injuries affecting the skull and brain. It would seem obvious that head protection while bicycling could save many lives. In fact, a recent study demonstrated that wearing a bicycle helmet can lessen the risk of a head injury by 85%. Why, then, do less than 5% of school-aged children wear bicycle helmets? Common reasons include lack of knowledge, inconvenience, peer pressure, and financial considerations.

The results of a recent case-control study suggest that a community-wide campaign promoting bicycle helmet use can significantly increase helmet use among school-aged children (*JAMA* 1989; 262:2256–2261). The campaign focused on three goals: "to increase parental awareness of the need for helmets, to promote use by children, and to reduce financial barriers to their purchase." The methods used to achieve these goals included public service announcements on television, newspaper coverage of the campaign, press conferences, distribution of educational material in schools, bicycle shops, physicians' offices, and health departments, and implementation of a bicycle safety program in elementary schools. Discount coupons for helmet purchases were distributed through physicians' offices, schools, youth groups, and community events.

After adjusting rates of helmet use for confounding variables such as race, bicycle type, and riding site type, the authors found that bicycle helmet use increased from 4.6% before the campaign to 14% after 16 months—a significantly ($p>$ 0.001) greater increase than the 1.0% to 3.6% change documented in a control community during the same period.

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AIDS

Statistical Analysis and Mathematical Modelling of AIDS. Edited by J. C. Jager and E. J. Ruitenberg. 167 pp, illustrated. New York, Oxford University Press, 1988. $56.50 (hardcover)

Simple Acts of Kindness: Volunteering in the Age of AIDS. Edited by John Griggs. 122 pp. New York, United Hospital Fund, 1989. $5.00 (paperback)

CRITICAL CARE

Manual of Critical Care Procedures. Edited by Lyle D. Victor, MD. 215 pp, illustrated. Rockville, Md, Aspen Publishers, Inc, 1989. $67.00 (hardcover)

FORENSIC MEDICINE

Unnatural Death: Confessions of a Medical Examiner. By Michael M. Baden, MD, with Judith Adler Hennessee. 209 pp. New York, Random House, Inc, 1989. $17.95 (clothcover)

GENETICS

Human Chromosomes: Manual of Basic Techniques. By Ram S. Verma, MD, and Arvind Babu, MD. 256 pp, illustrated. Elmsford, NY, Pergamon Press, Inc, 1989. $28.95 (paperback)

HEALTH CARE

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Formula for Life: The Anti-Oxidant, Free-Radical Detoxification Program. By Eberhard Kronhausen, EdD, and Phyllis Kronhausen, EdD. 623 pp. New York, William Morrow & Co, Inc, 1989. $20.95 (clothcover)

Take One as Needed: 50 Capsules of Humor for Temporary Relief of Misery Due to Low-Fat Diets, Safe Sex, and Aerobic Exercise. By Oscar London, MD. 150 pp. Berkeley, Calif, Ten Speed Press. $8.95 (paperback)

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1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925-927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337-338.

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References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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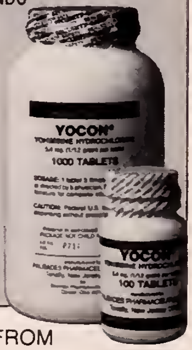

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Index to Advertisers

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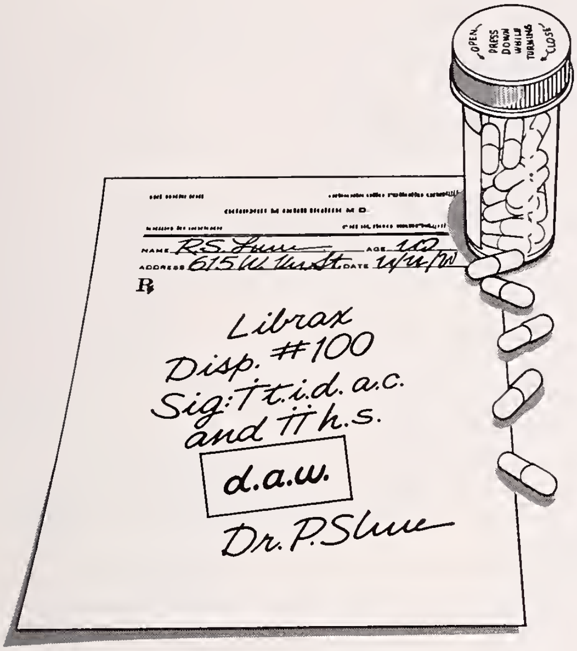

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Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

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FEBRUARY 1990 • Volume 90, Number 2

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Contents

COMMENTARIES

The health and health care challenges for New York City in the 1990s 51
PASCAL JAMES IMPERATO, MD

Bile duct carcinoma 53
RICARDO L. ROSSI, MD

RESEARCH PAPERS

An intervention to reduce the sale of cigarettes to minors 54
MICHELLE T. SKRETNY; K. MICHAEL CUMMINGS, PHD, MPH; RUSSELL SCIANDRA, MA; JAMES MARSHALL, PHD

Assessing the damage from cigarette smoking in New York State 56
PERRY F. SMITH, MD; JAMES M. SCHULTZ, PHD; DALE L. MORSE, MD

SPECIAL ARTICLE

The Alaskan community health aide scheme: A successful rural health program 61
SIXTEN S. R. HARALDSON

THE CPC

Congophilic angiopathy 64

CASE REPORTS

Bile duct carcinoma simulating choledocholithiasis 69
JOSEPH N. T. DYTOC, MD, FERNANDO V. ONA, MD

Carcinoma of the colon associated with systemic sclerosis 73
CATHERINE MARINO, MD; ELIZABETH McDONALD, MD; ARTHUR ALUNDAY, MD

Systemic embolization from left atrial ball thrombus: The case for early surgical intervention 74
DAVID WRISLEY, MD; IRA LEE, RDMS; FREDERICK PARKER, MD; WILLIAM BROWNLEE, RDMS; MOHAMMED ISMAIL, MD

LETTERS TO THE EDITOR

Student participation in mixed gender sports competition 76
ROSANNE HOLTZMAN, RN, BS

Physician recredentialing 76
PAUL E. REEVES, MD

Eliminating the requirement for medical libraries in hospitals 76
PETER ROGATZ, MD
LUCRETIA McCLURE, MA
FELIX WRÓBLEWSKI, MD
MARVIN GOLDSTEIN, MD

Gallstone appendicitis 77
JANE SIEGEL, MD; BENJAMIN W. PACE, MD; IRVING B. MARGOLIS, MD

Open supramalleolar amputation of the infected foot: An underutilized, two-stage procedure 77
JERRY J. SVOBODA, MD, MALUR R. BALAJI, MD

ESSAY REVIEW

Practicing clinical preventive medicine 79
ALLEN D. SPIEGEL, PHD

AN APPRECIATION

Our referees 83

LEADS FROM EPIDEMIOLOGY NOTES 85

BOOK REVIEWS 88

OBITUARIES 91

HOUSE OF DELEGATES

Sessions; Officers, Councilors, and Trustees; Members; Nominating and Reference Committees; Report of the House Committee on Bylaws 95

MEDICAL MEETINGS AND LECTURES 8A

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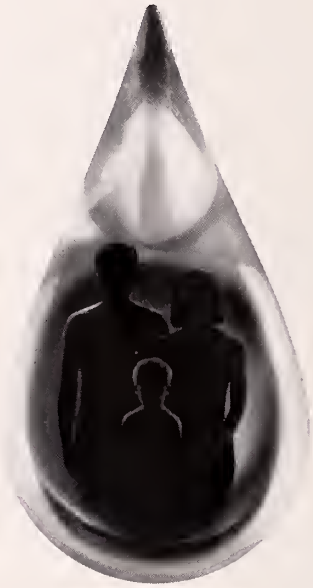

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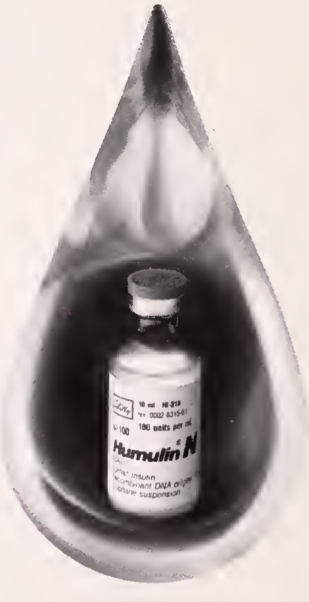

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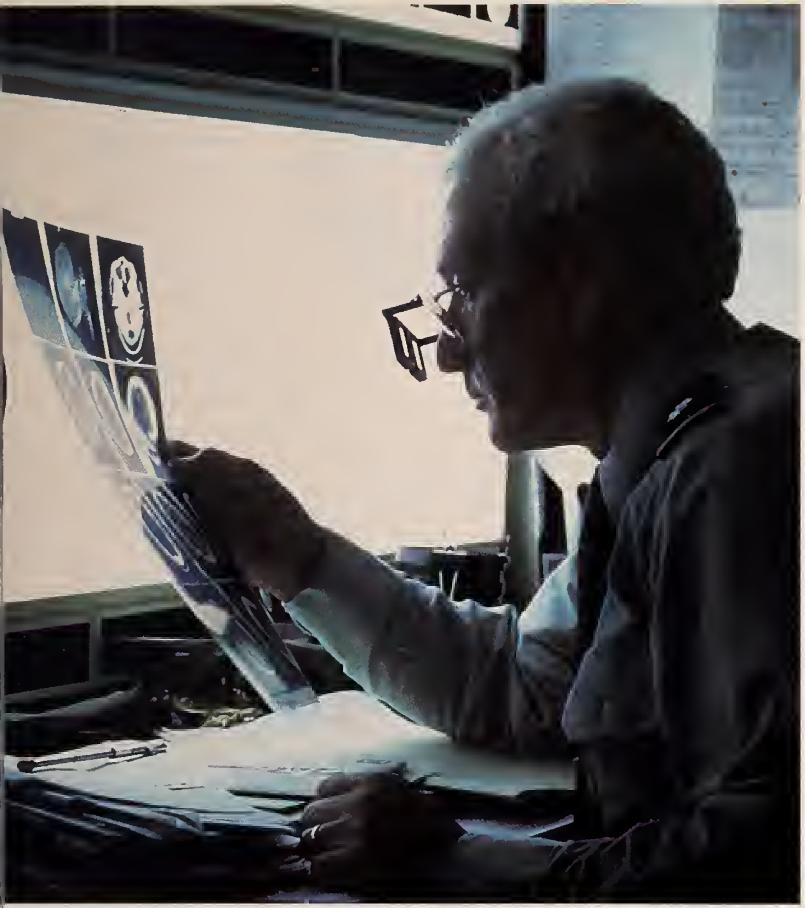

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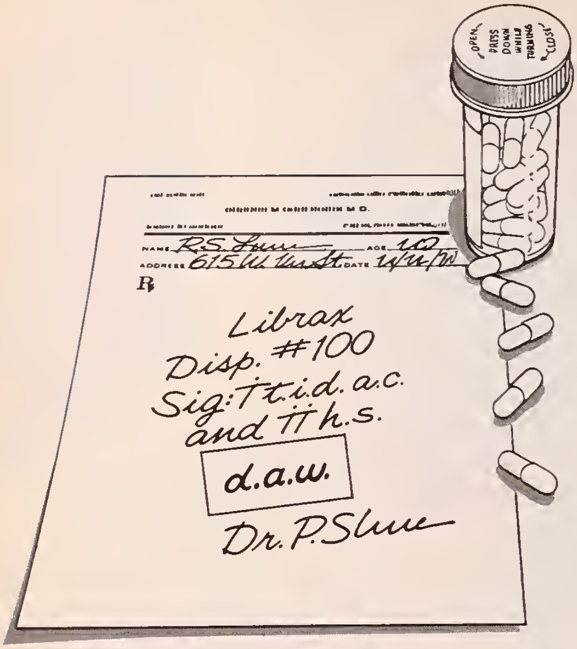

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Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.

As with all anticholinergics, inhibition of lactation may occur.

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

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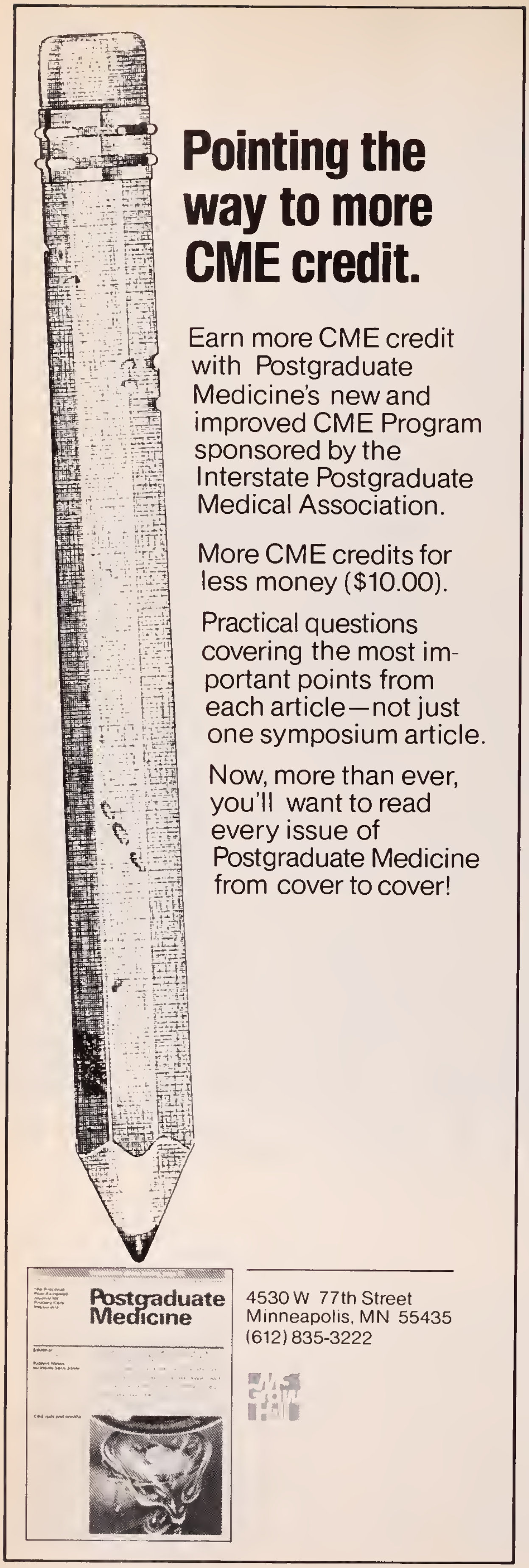

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MARCH 1990

AROUND THE STATE

ALBANY

Mar 22. **GI Teaching Day: Hypercholesterolemia.** Albany Medical College. Contact: Diane M. Martin, Conference Coordinator, Albany Medical College, Office of CME, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

BRONX

Mar 28. **Murder: Rockland County Style.** Lehman College Campus. Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 511 Hempstead Ave, Suite 2, West Hempstead, NY 11552. Tel: (516) 292-2300.

EAST ELMHURST

Mar 9, 10. **Emergency Medicine Oral Board Review Course.** 7 Cat I Credits. LaGuardia Marriott Hotel. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

MANHASSET

Mar 30. **Extrafamilial Child Sexual Abuse and Pornography: A Community Disaster.** 7 Cat I Credits. North Shore University Hospital. Contact: Mary B. Strong, MA, Conference Coordinator, North Shore University Hospital, 300 Community Dr, Manhasset, NY 11030. Tel: (516) 562-3045.

MANHATTAN

Mar 2–4. **Bellevue Hospital Center Psychiatry Board Review.** 23½ Cat I Credits. NYU Medical Center. *Mar 24–25.* **Cochlear Implants in Children: Rehabilitation Techniques.** 13½ Cat I Cred-

(continued on p 12A)

NEW YORK STATE
JOURNAL OF MEDICINE
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COMMENTARIES

The health and health care challenges for New York City in the 1990s

New York City enters the 1990s with a long agenda of social, economic, political, and health problems in need of solution. Newly inaugurated Mayor David N. Dinkins has pledged to do his best to address this wide range of challenges. However, effectively doing so in the environment of serious fiscal constraints that looms ahead will not be an easy matter. The new city administration faces a half billion dollar deficit in the current fiscal year and twice that amount next year. Preserving resources for human services will not be easy. As the new mayor has said, the city faces difficult times which require difficult choices.[1] These choices are likely to include less than optimal resources to deal with a number of ongoing and in some cases worsening health and health care problems.

In New York City these problems are often intricately intertwined in a web of social and economic determinants that include homelessness, poverty, drug abuse, unemployment, and crime. Many of them resisted resolution in the 1980s and have been passed on as a legacy to the 1990s. Among them is the drug epidemic, especially that of cocaine and crack. Ambulatory and inpatient hospital services are now overwhelmed by the medical and social consequences of drug abuse. Hospital emergency rooms in the city are stretched to the limits of effective functioning, not so much by rising numbers of visits, which have actually fallen, but by the nature of these visits, and because inpatient bed shortages cause a buildup of those requiring hospitalization. Over a third of all visits to hospital emergency rooms are currently related in some way to drug abuse. The violence spawned by both drug abuse and trafficking is often similar to that seen in warfare where massive gunshot injuries are common. These types of injuries place enormous burdens on emergency rooms in the city.

While drug abuse and the violence associated with it have escalated, the drug treatment infrastructure in the city has not kept pace with need. During the 1990s both the number of drug treatment programs and their overall patient capacity must be expanded to meet this need. Drug abuse is tragedy enough. However, the tragedy is compounded when someone desiring or in need of treatment is denied access or made to wait because of insufficient resources.

The epidemic of acquired immunodeficiency syndrome (AIDS) will continue to be a major challenge in the city during the 1990s. Its linkage to substance abuse has produced serious consequences for many infants born to mothers infected with human immunodeficiency virus (HIV). While there is evidence that prevention efforts among those men engaging in homosexual sexual contacts are succeeding, the same cannot be said thus far for AIDS prevention among intravenous drug abusers. Preventing AIDS among the latter is a major challenge for the 1990s as is reducing the numbers of HIV infected infants. The achievement of these goals will require an intensification of current efforts and the initiation of others. Special attention will have to be given to certain subgroups of teenagers who because of early age of first sexual contact, substance abuse, and other factors are at high risk of acquiring the infection and transmitting it to their infants.

The demand for inpatient beds for AIDS patients has steadily risen during a time when overall inpatient bed numbers in the city have been reduced. Innovative approaches to the management of HIV infected individuals hold promise for the 1990s for delaying the onset of clinical AIDS and for reducing reliance on inpatient services. Our current ability to monitor the progression of HIV infection through markers such as the CD4 (T4) lymphocyte counts and/or the CD4 percent permit more effective intervention with zidovudine (AZT) on an ambulatory care basis.[2] Similarly, *Pneumocystis carinii* pneumonia (PCP), which eventually infects 80% of those with AIDS, can be efficiently prevented on an outpatient basis with aerosolized pentamidine and trimethoprim-sulfamethoxazole.[3] All of this translates into a greater role for primary care in the management of HIV infected individuals and for a reduced reliance on hospital beds. The 1990s could be the decade when primary care gains ascendancy in the treatment of HIV infected individuals.

New York City's infant mortality rate is currently unacceptably high. There are several socioeconomic determinants in addition to drug abuse which have produced this high rate. These will have to be approached on a broad front by an array of human services organizations whose

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The opinions expressed in this editorial are those of the author alone and do not constitute an expression of official opinion of the Medical Society of the State of New York.

efforts must be coordinated to insure success. Perinatal services in the city are now inadequate to deal with the needs of many mothers and infants. Expansion of these services is only part of the solution. The recruitment and retention of health care workers to staff these services is also critical.[4] The provision of additional medical benefits to indigent pregnant women and their infants will help. However, the extra $123 million promised by the state for 1990 is insufficient.

Staff are in short supply not only in the area of perinatal care but also in the field of general nursing. The nursing shortage in general care hospitals in New York City is by no means unique. Despite improvements in entry salary levels, many nursing shifts are still inadequately staffed. The attractions of salary are soon lost on overworked nurses assigned to understaffed services. Not surprisingly, success in recruitment is frequently nullified by failure at retention. Nurses now have many more attractive career options outside of the hospital setting. These opportunities offer equal or better salaries, less stress, and regular working hours. While hospitals may compete on salary, they cannot match the other advantages of nonhospital nursing. The sharp decline in the number of applicants to nursing schools and the defection of many registered nurses from hospital care pose a very serious challenge for the 1990s. Hospitals in New York City and elsewhere will have to do more than raise salaries in the next few years to recruit and retain staff nurses.

Within the hospital sector, limited revenue growth through third party reimbursement will continue to place institutions in financial jeopardy. The impact of this limited revenue growth will be increasingly felt in ambulatory care services where losses are greatest at present. The 12% reduction in the number of general care beds that occurred in New York City during the 1980s has contributed to high occupancy rates compared to the rest of the country. This, coupled with lengths of stay longer than the national average and an increased demand for beds, has led to long waits for inpatient admission. Lengths of stay in New York City are frequently longer than the national average because of a number of medical and nonmedical characteristics of the inpatient population. These characteristics are not generally amenable to rectification, meaning that hopes for reducing waits for inpatient admission should not be placed on a reduction of length of stay. As the number of general beds fell in the 1980s, the demand for these beds rose because of AIDS, substance abuse, and an increased birth rate.

Solutions to the hospital sector problems will prominently figure in public policy debates in the 1990s. Some have already been put forth but have been shunned by state legislators and the state administration because they require increased revenues which can only be generated through tax increases. The New York State Department of Health's Universal New York Health Care (UNY-*Care) would, under whatever form adopted, require additional revenues. It would increase demand for and waits for inpatient admission because it would give access to those currently uninsured. It might shift ambulatory care visits from hospitals to private physicians' offices and could reduce hospital deficits. However, these deficits can only be appreciably reduced by improving ambulatory care visit payments from the current 1981 Medicaid base year. There will be an increased demand in the 1990s for an improvement in ambulatory care payments not only because they would reduce hospital deficits but also because they would shift patients from overburdened hospital outpatient departments to other ambulatory care settings. Beds could be freed up by extending outpatient care, decreasing lengths of stay, and by removing the burden from hospitals of managing nonmedical problems. However, none of these are easy objectives and have thus far proven unattainable. The New York State Department of Health announcement that inpatient reimbursement will go up 6.1% in 1990 is a step in the right direction. However, this increase is not likely to reduce inpatient deficits by very much.

The drug abuse epidemic has placed an enormous burden on both the inpatient and outpatient services of hospitals. In psychiatric services, the 1980s witnessed a 12% increase in psychiatric inpatient days. There was a 29% increase in the public hospitals.[5] This illustrates a medical problem with enormous social and economic determinants that cannot be resolved by medical means alone.

The New York City administration, facing as it does large budget deficits, will look to the state and federal governments for assistance in funding human services. It is unlikely that substantial help will be forthcoming in the early 1990s. What is likely is that more will have to be done with less in the public sector. In this scarce resource environment there is a high risk that the public debate will shift from focusing on solutions to fault finding. Local state and federal officials may seek to engender public support not by trying to solve problems but by convincing voters that it is someone else's responsibility to do so. This would be an unfortunate development but it looms large as a possibility from the perspective of 1990.

There are many other health and health care problems that need to be addressed in addition to the ones highlighted above. The national nature of the majority of these problems means that they cannot be addressed by local remedies alone. New York City's success or failure in dealing with them in the 1990s will in large measure be determined by the support and cooperation it receives from the state and federal governments.

PASCAL JAMES IMPERATO, MD
Editor

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Bile duct carcinoma

The paper on bile duct carcinoma by Drs Dytoc and Ona[1] in this issue of the *Journal* serves to remind us that bile duct carcinoma can mimic benign conditions of the biliary tree. Surgeons and gastroenterologists need to be aware that on occasion, patients who present with a picture of acute cholecystitis, acute or recurrent cholangitis, recurrent febrile episodes of unknown origin, or defects on cholangiography suggestive of stone can be suffering a malignant process of the biliary tree.

When the possibility of malignancy is suspected preoperatively in the patient who is a good surgical risk, a complete work-up is desirable. For proximal bile duct tumors, computed tomographic (CT) scan or ultrasound can reveal the presence or absence of metastatic disease. A percutaneous cholangiogram confirms the location of the lesion and its proximal extent. If it is felt that the patient is a candidate for resection, angiography should be performed. A proximal bile duct tumor involving the hepatic bifurcation should be considered for resection in a patient who is a good surgical risk if involvement of the secondary segmental ducts is unilateral, when the main hepatic artery and portal vein are not involved, if the tumor involves the hepatic artery or portal vein of the lobe affected but spares the vasculature of the opposite lobe. For distal bile duct tumors, if the CT scan does not show metastatic disease, an endoscopic retrograde cholangiopancreatography (ERCP) is performed in order to obtain tissue diagnosis, and on occasion, for retrograde decompression in patients in whom surgery has to be deferred.

For distal lesions, endoscopic papillotomy can occasionally be useful in the preoperative work-up, either for decompression, to differentiate a tumor from a distal impacted stone, to obtain tissue diagnosis, or to remove the stone if that proves to be the case.

When operating in the biliary tree for presumed benign disease, the surgeon has to be aware that the finding of stones does not necessarily rule out the presence of a carcinoma, as both conditions can coexist. One should try to obtain a complete cholangiogram that opacifies not only the distal biliary tree, but both the right and left hepatic ducts and hepatic bifurcation. Atypical findings at surgery, such as the absence of gallstones or common duct stones, or a decompressed gallbladder in a jaundiced patient, should increase the suspicion of a malignant process. Cholangiography and choledochoscopy are helpful.

If the clinical and radiological features in a case are those of malignancy, but a positive tissue diagnosis cannot be obtained in the operating room, the patient should be treated as having a carcinoma, as in most instances that will prove to be the case.

Prospective randomized trials comparing resective procedures with palliative endoscopic and percutaneous techniques in the management of bile duct cancer are scarce. There are retrospective data suggesting that the best results in the management of bile duct cancers in regard to quality of life and length of survival are achieved by resective operations.[2–4] At the time where major hepatic resections and pancreatoduodenectomies can be accomplished with mortalities under 5%,[3,5] we believe that patients with biliary malignancies who are good surgical risk patients should have the benefit of a surgical consultation, prior to being treated by palliative percutaneous or endoscopic means, with or without associated adjuvant therapies such as radiotherapy or chemotherapy.

In this writer's view, the final therapy of choice in the case presented by Drs Dytoc and Ona[1] would have been a pancreatoduodenectomy, which has an associated five-year survival rate of 30–60%.[5] It appears especially indicated in a fairly young patient in otherwise good health. Radiotherapy, as used in this case, has only achieved occasional long term survival and the use of chemotherapy continues to be controversial.

If at the time of exploration for a possible resectable tumor, this appears unresectable, palliation can be achieved either by proximal internal drainage (cholangiojejunostomies, proximal hepatico jejunostomies, etc) or stenting techniques. Although the role of liver transplantation for proximal bile duct cancer remains very controversial in the selection process of possible candidates, it is of importance that the patient's disease be adequately staged at the time of the first operation. This includes nodal sampling of the lymph nodes of the hilum of the liver, celiac nodes, and nodes of the peripancreatic area. Patients with negative nodes appear to fare better and therefore could be better candidates for transplantation, while patients with positive nodes would do poorly and would not be candidates for this therapeutic modality.[6]

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An intervention to reduce the sale of cigarettes to minors

MICHELLE T. SKRETNY; K. MICHAEL CUMMINGS, PHD, MPH; RUSSELL SCIANDRA, MA;
JAMES MARSHALL, PHD

ABSTRACT. This study reports the results of an intervention to stop the illegal sale of cigarettes to minors. Sixty-two of 120 stores in Erie County, New York, were randomly selected to receive in the mail an informational packet requesting their help in stopping the illegal sale of tobacco to minors. The packet included a letter to the store manager citing the law prohibiting the sale of tobacco products to minors and requesting their assistance in observing the law; a supply of warning signs; and a tip sheet designed to assist store managers in educating their employees about the law. Two weeks after the mailing, stores in the study were visited by study teenagers aged 14 to 16 who attempted to purchase cigarettes. The intervention resulted in an increased number of stores posting warning signs, but had no effect on deterring the sale of cigarettes to minors. Minors purchased cigarettes in 77% of stores that received the special mailing and in 86% of nonintervention stores. Warning signs were visible in 40% of stores that received the special mailing and in none of the nonintervention stores. The findings from this study suggest that a merchant education program to increase awareness of the law and posting of warning signs is not sufficient to discourage the sale of tobacco products to minors.

(NY State J Med 1990; 90:54–55)

In an effort to prevent children from using tobacco many states have enacted laws to restrict the sale of tobacco to minors.[1] In New York State it is against the law to sell tobacco products to anyone under 18 years of age (Penal Law § 260.20).[1] New York State law requires businesses selling tobacco to post a sign stating: "SALE OF CIGARETTES OR OTHER TOBACCO PRODUCTS TO PERSONS UNDER 18 YEARS OF AGE IS PROHIBITED BY LAW" (General Business Law § 399-e).[1] The mere existence of a law prohibiting the sale of tobacco to minors, however, does not guarantee that such sales will not occur. A number of studies conducted throughout the United States have demonstrated that illegal sales of tobacco products to minors are commonplace.[2–5]

In 1987, retail business establishments in the Buffalo, New York, area were contacted to determine their awareness of the law prohibiting the sale of tobacco to minors.[6] In 40% of the business surveyed, owners or store managers either did not know of the law or were misinformed as to the legal age for purchasing tobacco products. Only 29% of stores claimed to have a copy of the law posted on the premises as required by the law.

Several recent studies have suggested that increasing merchant awareness about laws prohibiting the sale of tobacco products to minors can result in increased compliance.[2,3,6] The purpose of this study was to determine whether tobacco sales to minors in the Buffalo, New York, area could be reduced through an education program designed to increase merchant awareness of laws prohibiting the sale of tobacco to minors and requiring the posting of a warning sign.

METHODS

The study population included 120 stores located in the Buffalo area, including 22 supermarkets, 55 convenience food stores, and 43 pharmacies. Stores were selected out of the Buffalo Area Telephone Directory Yellow Pages and were randomly allocated to intervention or nonintervention conditions. To prevent contamination of the intervention among chain stores, all stores within a particular chain were assigned to either intervention or nonintervention conditions.

The 62 stores assigned to the intervention group were sent an educational packet by first class mail. This packet included a letter to the store manager citing the New York State law prohibiting the sale of tobacco products to minors and requesting their assistance in observing the law, a supply of warning signs to be placed around cash registers and in store windows, and a tip sheet for educating employees not to sell tobacco to minors. The tip sheet was presented in a question/answer format that included information on penalties for selling tobacco to those under age, the circumstances under which a customer may purchase tobacco, placement of warning signs, and facts about youth and tobacco. District managers of chain stores assigned to the intervention group were sent a similar packet of information and encouraged to assist individual store managers in complying with the law. The 58 nonintervention group stores received no information.

Two weeks following the mailing of the educational packet to intervention stores, all stores in the study were visited by a minor, operating under adult supervision, who attempted to buy a pack of cigarettes. A total of seven minors aged 14 (n = 3), 15 (n = 2), and 16 (n = 2) years visited the stores to purchase cigarettes. Upon entering the store, the minors were instructed to observe whether the warning sign about tobacco sales was posted and to purchase one package of Marlboro Light Cigarettes. They were instructed that if asked, they should state their actual age and indicate they were buying the cigarettes for their own use. If a sale was made, the minor left the store with the package of cigarettes and returned to the car to complete a brief questionnaire about the sale.

From the Department of Cancer Control and Epidemiology, Roswell Park Cancer Institute, Buffalo, NY (Ms Skretny, Dr Cummings, Mr Sciandra), and the Department of Social and Preventive Medicine, State University of New York at Buffalo (Dr Marshall).

Address correspondence to Dr Cummings, Department of Cancer Control and Epidemiology, Roswell Park Cancer Institute, Elm and Carlton Streets, Buffalo, NY 14263.

TABLE I. Cigarette Sales to Minors and Posting of Warning Signs by Type of Store and Intervention Group

	Percentage of Stores Where Minors Bought Cigarettes (N)			Percentage of Stores with Warning Signs Posted (N)		
	Intervention	Control	p*	Intervention	Control	p*
Supermarket	63 (5/8)	93 (13/14)	NS	62 (5/8)	0 (0/14)	<0.01
Convenience store	79 (23/29)	85 (22/26)	NS	28 (8/29)	0 (0/26)	<0.01
Pharmacy	80 (20/25)	83 (15/18)	NS	48 (12/25)	0 (0/10)	<0.01
Total	77 (48/62)	86 (50/58)	NS	40 (25/62)	0 (0/58)	<0.01

* NS indicates not statistically significant at $p < 0.05$.

RESULTS

Results of the study are summarized in Table I. Overall, minors were successful in purchasing cigarettes in 82% (98/120) of the stores visited. Sales of cigarettes to minors did not differ between those stores receiving the mailing and those that did not (77% versus 86%, $p = 0.55$). Sales did not differ by type of store, the sex of the merchant, or the age and sex of the prospective purchaser.

There was a significant difference in the posting of warning signs associated with merchant education. Forty percent (25/62) of the stores that received the special mailing had warning signs posted. None of the nonintervention stores had warning signs posted. The results were consistent across type of store.

DISCUSSION

The findings from this study confirm the results of earlier investigations which have demonstrated that illegal sale of tobacco products to minors is common.[2-5] Results from this study also suggest that merely informing merchants about the law is not effective in reducing such sales. Although our intervention increased the number of stores posting required warning signs, the presence of a warning sign was not related to compliance with the law.

The findings from this study differ from those of Altman et al[2] who found that a voluntary merchant education program, combined with a media campaign and grassroots community organization effort, was effective in significantly reducing illegal over-the-counter sale of cigarettes to minors in Santa Clara County, California. There are several important differences between the intervention tested in this study and the program evaluated by Altman et al.[2] The California study involved a pre-post test design. Information obtained during the pre-test was used to create a media campaign designed to heighten public awareness about the illegal sale of tobacco products to minors. The support of community groups and local business leaders was also solicited to help promote awareness and observance of the law. The merchant education program was also much more extensive in the California study. Certified letters were sent to the chief executive officers of company-owned chain and franchise operations asking them to take specific actions to encourage store managers and employees to observe the law. Finally, store managers were informed of the pre-test findings in which minors had attempted to buy cigarettes in their store.

Increasing community awareness about the problem of illegal sales of cigarettes to minors appears to be an essential element in promoting merchant compliance with the law. Clearly, the norms in communities about minors' access to and use of tobacco need to change. The prevailing attitude regarding the sale of tobacco products to minors is illustrated by an interaction which occurred between a clerk and one of the minors in our study. During one sale, the clerk stated, "It's terrible that you are buying cigarettes. It is wrong, but I guess you could be doing worse things."

No single strategy is likely to be effective in stopping illegal tobacco sales to minors. In addition to community and merchant education efforts, the laws pertaining to the illegal sale of tobacco to minors need to be strengthened. The legal age for purchasing tobacco products should be increased from 18 to 21 years. We recommend that retail tobacco vendors be licensed annually, as are alcohol vendors. Licensure would provide an opportunity to educate vendors about state laws before their licenses are renewed. Retailers found to violate the law would be in danger of having their licenses revoked. The threat of losing the license to sell tobacco products would likely do more to motivate compliance than the existing penalties, which involve relatively small monetary fines.[1] Finally, enforcement of the tobacco access law is needed. Income generated from licensing retail vendors and from fines levied for illegal tobacco sales could pay for community and merchant education programs and for development of monitoring mechanisms to ensure vendor compliance with the law.

Acknowledgments. The authors thank Kevin Wiley, Nathan Gunn, Rebecca Marshall, Christine Marshall, Kristin Tripp, Anna Sciandra, and Kelly Toy for assisting with this project.

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Assessing the damage from cigarette smoking in New York State

PERRY F. SMITH, MD; JAMES M. SHULTZ, PHD; DALE L. MORSE, MD

ABSTRACT. Although there is clear evidence that smoking is the leading preventable cause of death in the United States today, almost one third of American adults continue to smoke. To promote public awareness of smoking's adverse effects, we applied a previously established methodology to estimate the impact of cigarette smoking in New York State. Using New York State-specific mortality data, cigarette smoking prevalence rates, and total health care costs, we found that cigarette smoking caused almost 27,000 deaths in 1985, resulting in 335,000 years of potential life lost (mean, 12 years per person). The estimated annual cost of smoking totaled $4.7 billion, including direct (health care) and indirect (lost income and productivity) costs from smoking-attributable deaths and illness. The New York State Department of Health publicized these findings to gain support for a statewide regulation for smoking control in public places. This methodology can easily be applied elsewhere to assess the adverse impact of smoking and to support smoking control activities.

(NY State J Med 1990; 90:56–60)

There is unequivocal evidence for the adverse effects of smoking on health. Recent annual surgeon general's reports have estimated that smoking causes 30% of deaths from cancer,[1] 30% of deaths from coronary heart disease,[2] and 80%–90% of deaths from chronic obstructive lung disease[3] each year. The estimated annual number of deaths attributable to smoking from these three causes totals 350,000—more than all the American lives lost in World War I, Korea, and Vietnam combined.[4] Determinations such as these have prompted the surgeon general to conclude that smoking is "the single largest preventable cause of premature death and disability in the United States."[5] However, despite widespread publicity about the dangers of smoking, nearly one of every three American adults continues to smoke.[6]

To promote public awareness of smoking's adverse effects at a state level, we estimated the impact of cigarette smoking in New York State. Using readily available state data and recently developed computer software, we determined the number of smoking-attributable deaths, years of potential life lost, and the economic costs resulting from smoking in New York in 1985. The results of this analysis provided support for a statewide regulation to restrict smoking in public places.

We present here the findings from this study. They not only document the huge impact of smoking at a state level but also illustrate the use of computer software that is readily available to state and local health departments.

METHODS

For these calculations we used a methodology that was developed by Rice and colleagues[7] and a microcomputer software package that was developed by the Minnesota Department of Health for use by state and local health departments.[8] A brief summary of these methods is presented here.

To calculate the number of smoking-attributable deaths, we used 1985 New York State gender-specific and age-specific mortality data for 20 diagnoses, which were selected because of their strong association with cigarette smoking.[7] We determined the number of smoking-attributable deaths by multiplying the total number of deaths from each disease, coded according to the ninth revision of the International Classification of Diseases (ICD-9-CM, 1978), times the percentage of deaths that were attributable to smoking. This percentage, defined as the smoking-attributable risk fraction, was derived from gender-specific smoking prevalence rates for current and former cigarette smokers and from relative mortality risk values for each diagnosis for current and former smokers compared with nonsmokers, according to the following formula:[8]

Smoking-attributable risk fraction =

$$\frac{(p_0 + p_1(RR_1) + p_2(RR_2)) - 1}{(p_0 + p_1(RR_1) + p_2(RR_2))}$$

where p_0 = prevalence of never smokers
p_1 = prevalence of current smokers
p_2 = prevalence of former smokers
RR_1 = relative risk for current smokers compared with never smokers
RR_2 = relative risk for former smokers compared with never smokers

For this calculation we used 1985 New York State smoking prevalence rates from the Behavioral Risk Factor Surveillance System[9] for persons 20 years old or older. The relative risk values were derived by Rice et al[7] from four prospective studies.

We estimated the number of years of potential life lost due to smoking by multiplying the number of deaths from smoking in each gender-specific five-year age group times the gender-specific number of expected years of life remaining for each age group. Values for the years of life remaining were based on New York State-specific vital statistics data and were determined from the midpoint of each age group to life expectancy.

Smoking-attributable economic costs were divided into three categories: direct health care, indirect mortality, and indirect morbidity.

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Direct health care costs were defined as personal health expenditures for the diagnosis and treatment of smoking-attributable diseases. To estimate these costs, we first determined the total costs in New York State for hospital care, physicians' services, nursing home care, medications, and other professional services (1982 data from the Health Care Financing Administration,[10] inflated to 1985 dollars). We next estimated the combined costs for neoplastic, circulatory, and respiratory diseases in New York State using the proportion of 1980 national direct health care costs that were attributed to these three disease categories. Finally, we estimated smoking-attributable direct health care costs by multiplying the combined costs for these three disease categories times smoking-attributable risk fractions for health care utilization. These risk fractions were based on the gender-specific prevalence rates for current smokers and the differential rates of medical care utilization for current smokers relative to never smokers.[7] Former smokers were not included in this calculation.

Indirect mortality costs were defined as the cost of lost income and productivity resulting from premature death due to smoking-attributable disease. We estimated these costs by adding the totals of the present value of future earnings in 1985 dollars, stratified by five-year age groups and gender, times the number of smoking-attributable deaths for each of the 20 diagnoses by age group and gender. We used 1984 estimates of the present value of future earnings with a 4% discount rate to adjust for future inflation. (AA Scitovsky, DP Rice, J Showstack et al, unpublished data, 1986). For our calculations, we inflated these figures to 1985 values.

Indirect morbidity costs were defined as the cost of lost income and productivity for persons who were disabled by smoking-attributable illness. Since these data were not readily available, we estimated these costs using national data on total smoking-attributable economic costs.[7] We assumed that the ratio of indirect morbidity costs to direct health care costs and indirect mortality costs in New York State was the same as the ratio observed in the United States. Using this ratio and the values for the direct health care costs and indirect mortality costs calculated for New York, we estimated the indirect morbidity costs for the state.

To assess the impact of maternal smoking on the fetus and infant, we also estimated the number of deaths, years of potential life lost, and indirect mortality costs for four fetal and infant diagnoses that have been causally linked to maternal smoking.[11,12] For these calculations we used the same methodology as presented above. However, because of the requirements of the software program, years of potential life lost were calculated from national estimates of life expectancy rather than from New York State-specific estimates, and the smoking-attributable risk fractions for these diagnoses were based on the smoking prevalence rates in New York State for women aged 20–64 years, as an approximation of the smoking prevalence rate for pregnant women.

RESULTS

The smoking prevalence rates used in our calculations are shown in Table I. Overall, for males and females combined, 32.3% were current smokers, 22.5% were former smokers, and 45.2% had never smoked.

The smoking-attributable risk fractions, total deaths, and estimated smoking-attributable deaths in New York in 1985 are shown by gender and diagnosis in Table II. In sum, there were almost 27,000 smoking-attributable deaths in the state. These deaths comprised 26% of the 104,220 deaths from the 24 smoking-related diagnoses, and 16% of all deaths in New York for that year. Over 17,000 (65%) of the smoking-attributable deaths occurred in males, consistent with the higher male smoking rates in the over-65 age cohort, which bears the primary burden of fatal smoking-attributable diseases. Overall, 20% of male deaths and 11% of female deaths were attributable to smoking in 1985. Three diagnostic categories—neoplastic, circulatory, and respiratory diseases—accounted for 26,229 deaths, or 98% of all smoking-attributable deaths. Among 8,898 smoking-attributable cancer deaths, 6,344 (71%) were from malignant neoplasm of the lung. Smoking was a factor in 80% of male and 47% of female deaths from lung cancer. Among 12,598 smoking-attributable deaths due to circulatory disease, 8,909 (71%) were from ischemic heart disease, with 6,263 (70%) of these occurring after age 64.

TABLE I. Smoking Prevalence Rates by Gender and Age Group, from the Behavioral Risk Factor Surveillance System, New York State, 1985

	Males		
Smoking Status	20–64 Yrs (N = 374)	≥65 Yrs (N = 92)	All Ages ≥20 Yrs (N = 466)
Current	35.3%	23.9%	33.6%
Former	24.7%	50.2%	28.6%
Never	40.0%	25.9%	37.9%
	Females		
Smoking Status	20–64 Yrs (N = 519)	≥65 Yrs (N = 150)	All Ages ≥20 Yrs (N = 669)
Current	36.1%	12.1%	31.3%
Former	15.6%	24.8%	17.4%
Never	48.3%	63.1%	51.3%

Smoking-attributable deaths resulted in the loss of approximately 335,000 potential years of life for New Yorkers in 1985. The mean number of years lost per person dying of smoking-attributable disease was 12. Males contributed 209,000 (62%) years to the total. The number of years of life lost varied by disease category (Fig 1). Of all the diseases in the circulatory category, ischemic heart disease contributed most heavily, with 102,000 years of life lost. Similarly, carcinoma of the lung contributed most heavily to the category of neoplastic disease, with 88,000 years lost.

Direct health care costs for treatment of smoking-attributable diseases were estimated at $1.9 billion for New York State for 1985 for current smokers (Table III). Males accounted for $1.1 billion or 57% of direct costs, a smaller contribution than males made to all other disease impact measures because of the higher rates of medical utilization by females. Hospital costs of $1.4 billion accounted for 76% of direct costs and 30% of the total estimated smoking cost of $4.7 billion.

Indirect mortality costs were valued at $1.9 billion in 1985 dollars. Males accounted for $1.4 billion (75%). This marked gender disparity resulted from the excess of male deaths, the greater number of men in the work force, and the higher male mean incomes in the calculation of the present value of future earnings. Smoking-attributable neoplastic and circulatory diseases accounted for 80% of total indirect mortality costs.

Smoking-attributable indirect morbidity costs were estimated at $910 million, with a striking differential by gender, as noted above for indirect mortality costs.

Overall, we estimated the total cost of smoking-attributable illness in New York in 1985 to be $4.7 billion. Direct health care costs comprised 40% of this total; indirect mortality costs, 41%; and indirect morbidity costs, 19%.

DISCUSSION

This study documents the huge impact of smoking at the state level. Comparing smoking with other public health problems provides perspective on the importance of the smoking problem. For example, the epidemic of acquired immunodeficiency syndrome (AIDS) has received great publicity. In 1985, the Secretary of Health and Human Services stated that AIDS was the number one public health priority in the nation.[13] Massive amounts of public resources have appropriately been directed against this disease. The magnitude of the AIDS epidemic is clear

TABLE II. Smoking-Attributable Risk Fractions and Deaths by Diagnosis and Gender, New York State, 1985

Disease Category and Diagnosis	Males			Females			Total
	Risk Fractions	Total Deaths	Smoking-Attributable Deaths	Risk Fractions	Total Deaths	Smoking-Attributable Deaths	Smoking-Attributable Deaths
Infectious							
Respiratory tuberculosis	.443	102	45	*	40	*	45
Neoplastic							
Lip, oral cavity	.693	477	330	.455	237	108	438
Esophagus	.594	582	346	.579	204	118	464
Stomach	.176	783	138	.289	592	171	309
Pancreas	.306	931	285	.164	1,028	168	453
Larynx	.814	291	237	.455	77	35	272
Lung	.801	6,007	4,810	.470	3,264	1,534	6,344
Cervix	NA[†]	NA	NA	.410	344	141	141
Urinary bladder	.378	634	240	.307	306	94	334
Kidney	.253	399	101	.138	307	42	143
Circulatory							
Hypertension	.160	914	147	.170	1,227	208	355
Ischemic heart disease, 20-64 yr	.288	7,066	2,035	.212	2,889	611	2,646
Ischemic heart disease, 65+ yr	.179	21,488	3,841	.092	26,441	2,422	6,263
Cardiac arrest	.402	926	372	.385	931	359	731
Cerebrovascular accident	.097	3,810	370	.159	6,320	1,008	1,378
Arteriosclerosis	.242	640	155	.350	1,004	351	506
Aortic aneurysm	.632	825	522	.509	387	197	719
Respiratory							
Pneumonia, influenza	.210	2,964	621	.107	3,077	331	952
Emphysema, chronic bronchitis	.855	726	621	.728	428	312	933
Airways obstruction	.855	2,113	1,807	.728	1,429	1,041	2,848
Digestive							
Ulcers	.487	350	171	.486	368	179	350
Fetal and Infant							
Short gestation/low birth weight	.215	176	38	.215	165	36	74
Respiratory distress syndrome	.215	188	40	.215	106	23	63
Other respiratory conditions	.215	212	46	.215	161	35	81
Sudden infant death	.153	169	26	.153	115	18	44
Total	—	52,773	17,344	—	51,447	9,542	26,886

* Insufficient data available to estimate smoking-attributable deaths from respiratory tuberculosis in females.

[†] NA = not applicable.

when one considers that there were 9,504 AIDS deaths among New York residents from the late 1970s, when the epidemic began, until July 1988.[14] However, the epidemic of smoking has had an even greater impact, causing several times this number of deaths each year. In 1985, AIDS resulted in approximately 2,700 deaths (New York State Department of Health, unpublished data) or less than 2% of the 170,193 total deaths in New York, while smoking caused a striking 16% of all deaths. AIDS resulted in the loss of approximately 71,000 potential years of life for New Yorkers in 1985 (New York State Department of Health, unpublished data), less than one fourth of the years lost because of smoking.

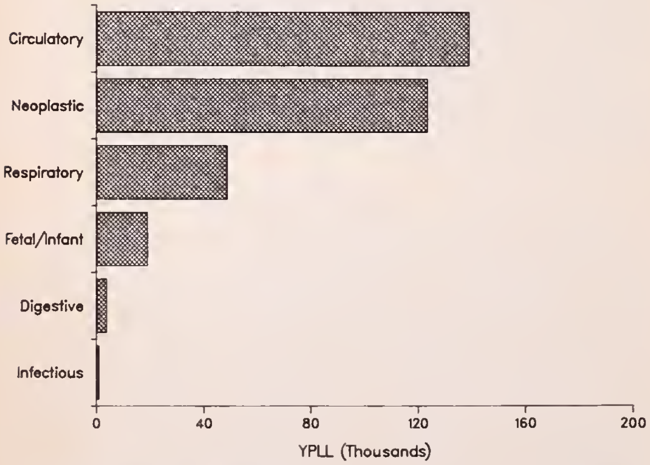

FIGURE 1. Years of potential life lost (YPLL) due to smoking, by disease category, New York State, 1985.

Automobile crashes are another cause of significant mortality in New York. They also have received much public attention, resulting in a state-wide seat-belt law in 1985. Automobile crashes resulted in the deaths of approximately 2,000 New Yorkers in 1985[15] and the loss of almost 76,000 years of potential life (New York State Department of Health, unpublished data). By comparison, smoking caused more than 13 times as many deaths and more than four times as many years of life lost.

Smoking has considerable economic impact as well. Our estimate of $4.7 billion for the total financial burden of smoking translates into an annual cost of more than $200 for every New York resident, smoker and nonsmoker alike. It is also clear that tax revenues generated by cigarette sales do not fully compensate for the cost of smoking. In 1985, an estimated 2.1 billion packs of cigarettes were

TABLE III. Smoking-Attributable Economic Costs[*] by Cost Category and Gender, New York State, 1985

Cost Category	Males	Females	Total
Direct health care costs			
Hospitalization	$ 865	$ 565	$1,421
Nursing home care	103	169	272
Physician services	84	43	127
Medications	24	14	38
Professional services	3	4	7
Subtotal	$1,070	$ 795	$1,865
Indirect mortality costs[†]			
Neoplastic	$ 586	$ 189	$ 775
Circulatory	578	180	758
Respiratory	152	63	215
Fetal/infant	77	47	124
Infectious	14	[‡]	14
Digestive	13	6	19
Subtotal	$1,420	$ 485	$1,905
Indirect morbidity costs			
Subtotal	$ 617	$ 293	$ 910
Total costs	$3,107	$1,573	$4,680

[*] Costs are rounded to nearest million in 1985 dollars.

[†] Specific diagnoses in each disease category are specified in Table II.

[‡] Insufficient data available to estimate indirect mortality costs for infectious diseases in females.

sold in New York State.[16] We estimate the tax revenues from these sales to be $441 million ($.21/pack) for New York cigarette tax, $336 million ($.16/pack) for federal cigarette tax, and $63 million ($.03/pack) for New York sales tax, yielding a total tax revenue of $840 million. This figure is less than half of the direct health care costs of smoking and is dwarfed by our estimate of $4.7 billion for the total cost of smoking.

Estimates of the adverse impact of smoking argue strongly for smoking control by encouraging smokers to quit and by mustering public support for legislative action. For this reason, the Minnesota Department of Health developed the microcomputer software program that we used for these calculations. The results in Minnesota provided a very convincing argument for smoking control legislation.[17,18] In New York, the results of our study were presented in public hearings before the New York State Public Health Council in 1987 to support an amendment to the New York State Sanitary Code. The amendment included one of the most comprehensive smoking control regulations in the nation by providing for a smoke-free environment in virtually all public places. Although the Public Health Council approved the amendment, it was later struck down by the New York courts on legal grounds. However, the state legislature subsequently passed the New York State Indoor Clean Air Act, which provides for similar smoking restrictions in public places (effective January 1, 1990) and in private and public worksites (effective April 1, 1990).

To assist other states and local communities in similar efforts to document the impact of smoking, the Centers for Disease Control of the Public Health Service has made the Minnesota microcomputer software program available to local health officials. The program is easy to learn and can be applied readily to local data.

Although the estimates of smoking's adverse impact are based on extensive epidemiologic research, there are limitations to this methodology. The central calculation is the smoking-attributable risk fraction, which depends in part on the relative mortality risk for smokers compared with nonsmokers. Estimates of relative risk for many smoking-related diagnoses vary from one study to another and may be affected by other behaviors that correlate with smoking, such as alcohol use. Furthermore, relative risk estimates vary for smokers in different age groups. Age-specific relative risks are not well defined for most smoking-related diseases. Therefore, age-specific smoking-attributable risk fractions were not determined for any of the smoking-related diagnoses except ischemic heart disease, for which only two broad age categories were considered.

Our economic calculations also have some limitations, since they are based on approximations of medical care utilization, incomplete data on the differential morbidity for smokers and nonsmokers, and on multiple economic assumptions. Furthermore, we did not include any estimates of the cost to society for persons who survive their full life expectancy rather than dying prematurely from smoking-attributable disease.

Despite these potential errors, however, there are several reasons why these calculations might underestimate smoking's impact. For example, our smoking prevalence rates included only cigarette smokers and not pipe or cigar smokers, who are also at increased risk for many of the same diseases as cigarette smokers.[4] Also, for each diagnosis, we assumed that deaths due to smoking-attributable disease were distributed by age in the same pattern as were all deaths from that cause. However, smokers are not only more likely to die from certain smoking-attributable diseases, but are also more likely to die younger from these diseases than their nonsmoking counterparts.[2,19] Since our calculations did not account for this premature mortality among smokers, our estimates of years of potential life lost and indirect costs are likely conservative. In addition, although our estimates of smoking-attributable mortality included the prevalence rates of former smokers, our calculation of smoking-attributable direct health care costs did not. Therefore, the cost of care for smoking-attributable disease in former smokers was not included.

This study dealt with the adverse impact of smoking on smokers, infants, and fetuses, and did not include the effects of passive tobacco smoke exposure on other children and adults. The 1986 surgeon general's report documents that nonsmokers who are exposed to environmental tobacco smoke have an increased risk of developing disease, including lung cancer and respiratory infections.[5] We did not include smoking-attributable deaths among these nonsmokers, since the relative mortality risk from passive smoke exposure is not yet precisely defined. However, as additional information about this hazard becomes available, it will be possible to estimate the impact of smoking on nonsmokers as well.

We conclude that smoking causes enormous costs to Americans, both in preventable deaths and in economic loss. Efforts to increase public awareness of the hazards of smoking and to promote a smoke-free environment by restricting smoking in public places would likely decrease the prevalence of smoking and result in substantial health benefits. Estimates of the adverse impact of smoking provide strong support for such efforts.

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FROM THE LIBRARY

THE PHYSICIAN'S OBLIGATION IN THE SMOKING ISSUE

Seven score and fifteen years ago . . . Abraham Lincoln, the great emancipator was born. By a remarkable coincidence, Charles Darwin, who emancipated science and men's minds, was born the same day and year. Today, we are in need of further emancipation of men's minds and of their lungs and hearts.

Now we are involved with perhaps the greatest public health problem ever known. The world-wide cigaret habit has had such an insidious development and has become so firmly entrenched that we still have no proper perspective of it. Pulmonary cancer, emphysema, and cor pulmonale are causally related. Other degenerative diseases are aggravated by it; these include coronary spasm and thrombosis, circulatory disturbances, peptic ulcer, and so forth. Quantitatively, the resulting wide devastation is far greater than smallpox or the plague ever was. Only a few years ago, daily headlines screamed of epidemics of poliomyelitis. The death and the crippling of children is tragic but has never been economically important. The cigaret death, however, hits the head of the family, the wage earner, and the leader. It punches a hole in the great interlocking economic framework on which our modern complex life depends. Thirteen years ago, much publicity illuminated the loss through lung cancer of a great and guiding figure in our international relations. Only recently there have been scores of important and potentially great men cut down more quietly. The worlds of sport, medicine, communications, entertainment, the military, and others have paid human sacrifice to the cigaret. We will never know how many thousands have died on the threshold of accomplishment.

In contrast, advertising has created a smoking super-race which must be dethroned. Coaches have always known instinctively that cigarets diminish full physiologic performance, yet Saturday's heroes tell our young to light up. On the stage and screen, not only the sophisticated good guys but also the virile bad guys maintain a perpetual semaphore of smoke. The beautiful double-breasted movie star seductively suggests that the adolescent cuddle up with her and a full-flavored smoke, but she does not explain that the lung is nature's final filter.

Who will be the emancipator? Certainly, government cannot and will not. The Monday after the report of the Presidential Survey, that ubiquitous and anonymous "source close to the White House" said that if Health, Education, and Welfare started anything, they would find their appropriation cut so fast they would not only be short of funds but also short of breath. In this war of attrition which is going to last at least a generation, the medical profession must be the leader, the emancipator. We should take the initiative now and we must not lose it. . . .

RICHARD H. OVERHOLT, MD
(*NY State J Med* 1964; 64:1297-1300)

The Alaskan community health aide scheme

A successful rural health program

Sixten S. R. Haraldson

The only part of the United States in the arctic region is Alaska, with its 1.5 million square kilometers mainly located between 60° and 70° north latitude. The southwestern half of Alaska is subarctic, while the northeast is arctic. Much of the state is covered by an enormous permafrost tundra. Hunting and fishing still constitute the main occupations of the Alaskan natives.

Of the total Alaskan population, almost half a million (84%) are non-native immigrants living mainly in urban areas in the south and southwest. Sixteen percent are natives, consisting of about 75,000 Eskimos and Aleuts and 40,000 American Indians. The average population density of all Alaska is 0.3 persons per square kilometer, but if non-native settlement areas are excluded, the figure drops to $0.04/km^2$, which means an extremely low density of people. There are also enormous distances between some 200 native villages scattered over a gigantic wilderness with a harsh climate.

Villages vary in size from 30 to a few hundred inhabitants. The inhabitants of this region face many adversities that challenge the development of modern services such as education, health, transport, and communication.

MORBIDITY AND MORTALITY

A dramatic decline in morbidity and mortality rates among Alaskan natives, especially regarding tuberculosis, otitis media, and other infectious diseases over the last two decades, is partly due to general socioeconomic development, and partly to efficient rural health programs.

The morbidity/mortality pattern[1] among natives has successively become similar to that seen in the western world, with an increasing frequency of cardiovascular diseases and malignant neoplasms. This is seen in spite of the young age of the Alaskan native population. The native birth rate is more than double the average rate in the rest of the US. The total mortality figure for natives is lower than the average figure for the US as a whole and is unique when one considers the extreme living conditions of the Alaskan natives and compares them with scattered or similar adversely situated populations in other parts of the world, including arctic regions.[2]

Accidental deaths are still at the top of the list, and standardized rates of death from influenza and pneumonia are several times higher than among the total US population. Other major causes of mortality are suicide, homicide, alcoholism, and cirrhosis of the liver, as well as diseases of early infancy. The infant mortality rate of Alaskan natives had already decreased to 17/1,000 in 1981 from about 50/1,000 in 1960. The corresponding rate for all the United States was 12/1,000. Neonatal mortality is almost identical in the two groups. The somewhat higher numbers of postneonatal deaths among natives are closely correlated to low birth weight.[3] The low neonatal mortality and an almost negligible maternal mortality are largely due to the fact that practically all deliveries in Alaska are now performed in institutional settings. Native expectant mothers are sent, a few weeks in advance, by air to the hospital, where the waiting period before delivery is used for health education and intensive prenatal care.

THE COMMUNITY HEALTH AIDE SCHEME

The earliest health services for the natives of Alaska came in the 1930s with the creation of rural hospitals in several towns, mostly along the coastline, by the federal Bureau of Indian Affairs (BIA). In the 1950s, the responsibility for native health was taken over by the US Public Health Service, and in the 1960s, regular calls to native settlements began, focusing primarily on maternal and child health and on environmental sanitation.

A new type of health worker emerged during this period known as the Community Health Aide (CHA). The aide's purpose was to provide primary care to villagers. Since 1968, these aides have undergone standardized training, which is designed and organized by the Alaska Area Native Health Board in Anchorage, and carried out in each of seven service units. The village council for each of the about 200 native villages chooses a local woman (only a few men are aides) for training.

Training focuses on primary health care and on health problems and diseases that consume the major part of rural health services. Prevention, immunizations, and health education are important ingredients in the curriculum and in the practice of the CHAs.[4] The basic course for the trainees, who usually have completed about nine years of primary school when they are selected, lasts nearly three months, at which time the CHAs return to their own villages and start practicing. Each village clinic is equipped with a well-designed drug kit, holding essential remedies and vaccines.

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In her daily work, the CHA is supported by a manual, *Guidelines for Primary Health Care in Rural Areas*,[5,6] and by daily radio/telecommunication via satellite with doctors at the regional hospital. Difficult cases (5–15%) are transported by air to the hospital for more qualified diagnosis and treatment. Alaska has a unique air service system with 350 airports, all-weather airstrips, and well trained "bush-pilots."

A system of regular supervision exists, combined with assistance to the village clinic, by the public health nurse, and occasionally by the physician at the regional hospital, who, as a member of the team, is responsible for that particular clinic and CHA. Together with frequent refresher courses, such features are of vital importance for maintaining skills, creating a team spirit, and supporting the CHAs' self confidence as the "village doctor." The villagers know that she is continuously advised by the hospital doctors.

Native health services in Alaska are financially supported both by the federal government, through the Alaska Native Health Services (ANHS), the State of Alaska, and by 12 native regional health corporations. The CHAs are paid according to a contract between these authorities. Remunerations vary somewhat from place to place, but are attractive, and are about equivalent to the salary of a low level primary school teacher. The total cost or the per capita cost of health services for a village is complicated to calculate, as a number of activities and several authorities are involved. It has been estimated that almost half of the total expenditures are swallowed by rather generous air transport of patients and of health personnel.

In contrast to the situation in comparable regions of the world, the Alaskan natives generally wish to remain rural, living close to generous natural resources, in an arctic ecology to which they have adapted. In spite of the acceptance of western cultural and technical elements, the Eskimos in particular keep a strong cultural self-identity.

It has therefore been considered urgent in Alaska to create a satisfactory network of rural primary health care for natives. Thanks to cooperation from the natives' side, the present CHA scheme has developed well and functions well in its unique rural setting. The program is a front-line service to all villagers, affording full geographic coverage and accessibility. It is well accepted and fully utilized, although the average population density is extraordinarily low.

Twenty years ago, a basic medical training program for auxiliary health care personnel of less than half a year would have been put off as inconceivable. Today it is realistic, assuming that the training[7] and the working programs[5] have been carefully planned and maintained by dedicated public health personnel. Some of the latter have come from the contiguous lower 48 states, and have remained in Alaska with the native health service program. Thus, concepts and methods incorporated in the CHA scheme have been thoroughly tested and evaluated and are now well adapted to local conditions.

The World Health Organization (WHO) program "Health for All by the Year 2000" (HFA) is an appeal for improvement of rural health services, bearing in mind the fact that rural people everywhere have less access to health facilities than do people in urban regions. HFA aims at accessible health care (as defined at the Alma Ata world conference in 1978). Front-line health stations should offer a broad spectrum of services, with prevention, immunizations, and health education as significant ingredients. It is presumed that peripheral rural services ought to rely, to a large extent, on locally recruited auxiliary health workers.

It is evident that there will be obstacles in a project as ambitious as HFA. Some population groups will be difficult to reach and serve, for instance the more than 15 million refugees of the world and the mushrooming slum populations of numerous cities and megatowns. A third problem group includes peoples inhabiting difficult to reach locations, extending from 50 to 100 million nomads to indigenous and ethnic minorities, sometimes scattered over large areas with low population density and great distances between settlements and camps.[8–10] Apart from being geographically difficult to reach, tribal groups may, in addition, be distrustful because of cultural and linguistic barriers.

Scattered populations call for nontraditional design of health care services.[11] Front-line health services based on locally recruited, indigenous people with an educational background of only 5–6 years of primary school, and with one half to two years of medical training to become auxiliary health workers, have been tried in several countries, especially in the Third World, with varying success. Examples include "barefoot doctors" in China and the "aid-post-orderlies" of Papua, New Guinea.

The Alaskan model of health service has not attracted justified attention by countries with comparable scattered populations, neither in the Arctic,[2] nor in the developing nations of the world. It may therefore be useful to look in detail at some of the ideas and principles behind the Alaska CHA scheme.

Why has Alaska's health care program been so successful? It could be a fortunate consequence of ample financial and other resources from a rich mother nation, or it could be a result of some ingenious ideas, or a mixture of both assets, plus dedicated cooperation between Alaskan natives and non-native Americans.

The trainees are chosen by their own fellow villagers, and after training the CHAs live and work among their own people. Selecting trainees in this way seems to be of great psychological importance, as it tends to create a foundation of mutual confidence. Furthermore, the training is concentrated on a relatively small number of leading rural health problems. Other essential elements include team work, frequent consolidation courses, a splendid working manual, and daily, compulsory utilization of well-functioning radio/telecommunications.

Some features of the CHA scheme raise the standard of services without involving high cost. They are therefore transferable to places and countries with fewer financial means than those of Alaska, and are applicable in other arctic regions as well. Even in the absence of air and modern road transportation, improvements of existing front line health services can often be achieved. However, the limited resources in the Third and Fourth World countries will force the continued use of mules, camels, jeeps, and stretcher bearers as dominant means of transportation.

It is evident that authorities are often reluctant to launch and run services for people in extreme rural settings. Calculated per capita, cost for such services may be prohibitively high for a minimal national health budget. Other factors in many places have been the lack of primary schools and insufficient utilization of existing educational facilities. High rates of illiteracy greatly restrict the selection of suitable candidates for training as auxiliary health workers.[12]

Common drawbacks to the establishment of an adequate health care program are the absence of radio and telecommunications, adequate transportation facilities, the lack of extension and refresher courses for auxiliaries, and half-hearted cooperation from local villagers. Finally, inter-tribal friction may prompt members of one tribe to refuse to accept services provided or run by members of other tribes, which may impede the development of an efficient rural health service.

The exchange of experiences regarding health services ought to be universal, and not restricted to certain latitudes, such as the arctic or the tropical regions. Climatic dissimilarities are of minor importance and should not constitute barriers to the sharing of information.

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Tower of Verrazano-Narrows Bridge, New York, NY (photograph by Florence Kavaler, MD).

Congophilic angiopathy

Mary Anne Whalen, MD (Attending Physician): A 53-year-old woman with Down's syndrome died of a cerebral hemorrhage. This presentation focuses on the studies and information available relating to dementia in Down's syndrome, Alzheimer's disease, and the entity of cerebral amyloid angiopathy, also known as congophilic angiopathy.

Cerebral hemorrhage, the cause of death of this patient, accounts for approximately 10% of recognized cerebrovascular accidents. The antecedent risk factors are now fairly well established. They include hypertension, therapeutic anticoagulation independent of hypertension, bleeding diatheses secondary to a variety of other causes, cerebral vascular malformations, and factors such as cardiac dysrhythmia or structural disease, which predispose to embolism and cerebral infarctions—these may then become hemorrhagic.

Dr Richard Hood has cared for a number of these patients at the Pathfinder Village over the years. Dr Hood assured me that as far as he recalls, the patient presented here did not have any risk factors that would have predisposed her otherwise to experiencing cerebral hemorrhage.

More recently, we have come to appreciate that persons with Down's syndrome are particularly at risk for cerebral congophilic angiopathy associated with cerebral hemorrhage or independent of another identified hemorrhagic event.

Cerebral congophilic angiopathy in its most typical form is a condition in which beta amyloid protein infiltrates the walls of small- to medium-sized arteries in the brain, and, principally, those in white matter. The likelihood of this condition tends to increase with age and may be present in roughly half the general population over the age of 70 years.[1] In patients with Down's syndrome, it is said to be virtually a ubiquitous finding over the age of 30 or 40, though I must comment that the studies of this condition have not been completed in such a fashion that would permit such a blanket statement.

There is an association of congophilic angiopathy with dementia that appears to be independent of the association with age. Cerebral congophilic angiopathy is not associated with systemic amyloidosis or with the other risk factors for cerebral hemorrhage. It is reported to be the basis of about 10% of cases of cerebral hemorrhage in the general population, more frequently in lobar hemorrhage rather than hemorrhage into the basal ganglia or into the brain stem.[2] In general, the latter are the most frequent sites of hemorrhages. In particular, cerebral congophilic angiopathy is found to be the underlying pathology in cases in which lobar hemorrhages are recurrent. It may also be the underlying cause of brain infarction.

That genetic factors may underlie this condition is shown by the reports of dominant transmission in some families, largely from the Netherlands and from Iceland.[3] Cases of congophilic angiopathy in the general population are considered to be sporadic. The incidence of cerebral congophilic angiopathy is not known, although it is said to be very frequent in autopsied cases when its presence has been sought. The incidence of cerebral hemorrhage in Down's syndrome is also unknown, so I cannot argue from a firm statistical basis that congophilic angiopathy was the underlying cause of hemorrhage in the patient described here. Nonetheless, supported by the absence of any other identified risk factors in this patient, I have chosen to proceed on the assumption that congophilic angiopathy was present, because this occasion provides the opportunity to talk about the evidence that links cerebral amyloid proteins and Alzheimer's disease.

The accepted neuropathologic hallmark of Alzheimer's disease has long been the identifiable presence of neuritic plaques and neurofibrillary tangles, appearing both cortically and subcortically, with considerable variability of distribution throughout the brain. The plaques and tangles in brains are found in nondemented persons as well, and to a certain extent may be regarded as a normal consequence of the aging process. However, we are beginning to develop age-standardized criteria for this finding. It has been clear for some time that an increased density of plaques and tangles in the brain is present in patients with dementia. In fact, this density is necessary to the definition of Alzheimer's disease and must be paired with clinical evidence of dementia before that diagnosis can be made.

An increased density of plaques and tangles has been appreciated for 60 years in patients with Down's syndrome, causing numerous authors to infer that dementia was also present, but frequently with no clinical correlations that would justify such assumptions. Critical examination of the literature regarding dementia in Down's syndrome uncovers a genuine paucity of data on which this assertion is based. Rigorous definition of dementia would require data obtained from longitudinal observations of the same patient, preferably formal psychometric data that would permit appropriate quantification. Such data concerning patients with Down's syndrome are scarce, and certainly the more subtle manifestations of dementia may easily go unnoticed in a population of persons whose

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baseline is already one of some degree of impairment. Nonetheless, there seems to be good evidence that an increased incidence of dementia occurs at an earlier age in patients with Down's syndrome.

It should be noted, though, that both in the general population and in the population with Down's syndrome, the relationship between plaques and tangles and dementia cannot be demonstrated to be linear. That is, more or fewer plaques and tangles do not necessarily mean more severe or less severe dementia. It has been proposed that only a critical mass of plaques and tangles is necessary for intellectual dysfunction to become evident.

What relationships, if any, can be said to exist between patients with Alzheimer's disease, patients with Down's syndrome, and patients with congophilic angiopathy? First, it now appears to be established that the neuritic plaques essential to the definition of Alzheimer's disease are composed of a core of amyloid protein which, by immunologic assay and by amino acid sequencing, seem to be virtually identical to the cerebral vascular amyloid of congophilic angiopathy.[4] Second, although most instances of Alzheimer's disease occur sporadically, about 10% of such cases are apparently familial in nature. An analysis of the pedigrees of these families has resulted in tracking the gene for Alzheimer's disease, as expressed in these families, to chromosome 21. Independent work has localized the amyloid protein gene to the same region of this chromosome—the same chromosome whose genetic material is present in triplicate in Down's syndrome.[5]

Two different techniques have been used to map the involved gene location in the familial Alzheimer's disease cases, and for the beta amyloid protein found in neuritic plaques as well as in the arteries in the brain in persons with congophilic angiopathy.

Studies mapping familial Alzheimer's disease have taken advantage of a technique known as restriction fragment length polymorphism to study the hereditary patterns in these families, and to work out the region on the 21st chromosome where the gene appears to be located. This region in familial Alzheimer's disease has been localized within the 21st chromosome in the area known as the Q21 (11-21) region. Cell hybridization techniques on the other hand have been used to map the localization of the beta amyloid protein gene. This technique creates a DNA hybrid between mouse and man essentially, and then uses further technologic means to identify the region of gene localization. These studies have mapped the gene for beta amyloid protein, again to the 21st chromosome and to the same Q21 region.

It is felt that the liability to congophilic angiopathy and the apparent increased incidence of Alzheimer's disease in Down's syndrome are both secondary to the increased burden of the pertinent genomic region on chromosome 21, which persons with Down's syndrome carry in triplicate. It is further hypothesized that altered gene expression of this genome, through mechanisms not yet understood, accounts for the instances of dominantly transmitted Alzheimer's disease in the general population, and for the more widespread association of cerebral amyloidosis, neuritic plaques, and dementia.

To date, only one unequivocally established case of a person with Down's syndrome, documented dementia, cerebral hemorrhage secondary to cerebral congophilic amyloidosis, and neuropathologic evidence of Alzheimer's disease has been reported.[6] Several staff members of the Pathfinder Village, where the patient presented here resided, will offer their opinions about whether this patient did in fact have any evidence of dementia, premature senility, or loss of function over the period of time during which they observed her.

Ms Janet Wiswell: I am a nurse at the Pathfinder Village. We did notice that the patient was slowing down. She was less alert, more tired, and not as patient with the children as she had been. She certainly acted older than her age.

Dr Whelan: Was there any clear-cut loss of skills that you could identify? Any activity that she was formerly able to do and now was no longer capable of doing?

Ms Wiswell: Not that I am aware of.

Dr Whelan: Was there any alteration in her language ability?

Ms Wiswell: Not that I noticed. She had visitors almost every day and she would talk to them.

Dr Whelan: I think that someone else made an interesting comment about the different perceptions between people who saw her every day and people who had seen her at more infrequent intervals.

Ms Carol Kessler: I am the program director at Pathfinder Village. This patient seemed to act more like a normal 70-year-old. She wanted quiet and liked to go to bed early.

Dr Whelan: In general, have you observed many instances of what you would think of as premature aging in your population of older persons at Pathfinder Village?

Ms Kessler: Yes, often. By their 20s, most patients with Down's syndrome seem to reach a plateau in their development, and aging is seen by the time they have reached their early 30s. By their late 30s and early 40s, most act like normal people in their 50s and 60s, with graying of the hair and loss of agility.

Dr Whelan: How many patients over the age of 30 are in residence at the Pathfinder Village?

Ms Kessler: About 35 patients.

Dr Whelan: They would be a very interesting population to follow to document significant mental changes. This patient's record contains one instance of formal psychometric testing in August 1975, using the Wechsler Adult Intelligence scale, on which she obtained a verbal IQ of 54, a performance IQ of 50, and a full scale IQ of 50. The statement was made that "Her fund of knowledge was relatively good; she was able to tell me the shape of a ball, knows how many months there are in a year, and with help was able to tell me the names of four past Presidents."

I am anxious to know from the pathologist today whether this patient did have cerebral amyloid (congophilic) angiopathy, and whether an excessive number of neuritic plaques and tangles were present when the brain was examined.

CLINICAL DIAGNOSES

Down's syndrome.

Intracerebral hemorrhage most probably secondary to cerebral amyloid angiopathy (congophilic angiopathy).

PATHOLOGY PRESENTATION

Harlan D. Alpern, MD (Pathologist-in-Chief): When not due to trauma, intracerebral hemorrhage is most commonly due to hypertension. Less common causes include vascular malformations (especially arteriovenous malformations and cavernous malformations), blood dyscrasias, hemorrhagic brain infarction, arteritis, and cerebral amyloid angiopathy (also called congophilic angiopathy).

This 53-year-old woman with Down's syndrome died of a massive intracerebral hemorrhage, due to one of these less common causes. The brain had a mass of 1,200 g, with a rounded shape due to foreshortening. The superior temporal gyrus was thin, and the cerebellum was small. In the left frontal lobe was a 12 × 6 × 5-cm region of intraparenchymal and subarachnoid hemorrhage. The hemorrhage produced increased intracerebral pressure, causing herniation of the hippocampal gyrus into the tentorial notch between the brain stem and the free edge of the tentorium. This herniation produced compression of regional veins, leading to a secondary mid-brain hemorrhage, which compromised vital autonomic functions of respiration and heart rate with resultant death.

The cause of the hemorrhage was evident by microscopic examination of the brain. Numerous small- and medium-sized arteries in the brain and in the leptomeninges demonstrated a marked thickening and eosinophilic hyalinization of their walls, characteristic of amyloid deposition (Fig 1). In a section stained by crystal violet, metachromasia was evident; in a section stained by Congo red and visualized under polarized light, there was apple-green birefringence. Most dramatic was the strong fluorescence seen in the section stained by thioflavine T (Fig 2). These histochemical reactions confirm that the eosinophilic material seen in the vessel walls is indeed amyloid.

The histologic sections of the brain demonstrated large numbers of senile plaques, as well as some neurons with neurofibrillary degenerative changes, all characteristic of Alzheimer's dementia. The senile plaques, which may be seen by routine hematoxylin and eosin stain, are better demonstrated in the Jamarri stain (Fig 3). Senile plaques stained by the periodic acid-Schiff reaction and also stained for amyloid.

By electron microscopy, senile plaques have been demonstrated to contain amyloid within their central portions, while their peripheral neuronal processes, which are abnormal, contain enlarged helical fragments. Hirano bodies and granulovascular bodies are less frequent morphologic findings in cases of Alzheimer's disease.

FIGURE 1. Thickening and eosinophilia of cerebral arteriole wall due to amyloid deposition (hematoxylin-eosin stain; original magnification × 100).

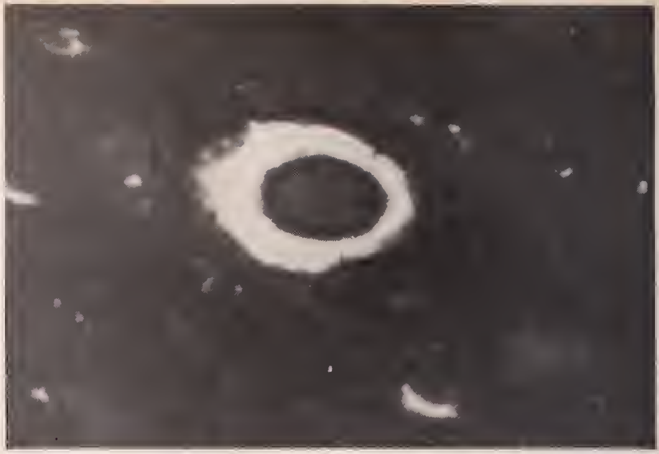

FIGURE 2. Fluorescence attributable to amyloid in wall of cerebral arteriole (thioflavin T stain; original magnification × 100).

The general autopsy performed on this patient was otherwise unremarkable, except for marked chronic superficial gastritis and colonic diverticulosis.

The gross appearance of the brain in Down's syndrome is more specific than are the microscopic findings. The weight of the brain in adults with Down's syndrome is low, generally about three quarters of normal size, averaging about 1,000 grams, and rarely exceeding 1,200 grams. The shape of the brain is unusual because of rounding of its contour, which may be secondary to the decrease in its anteroposterior dimensions, with a flattened occipital contour. Characteristically, the superior temporal gyrus is narrowed and the sylvian fissure is widened. There may be asymmetries of the two cerebral hemispheres. The cerebellum and brain stem are generally small, relative to the size of the cerebrum. The view has been expressed that the shape abnormalities of the brain are secondary to retarded skull development, which occurs in Down's syndrome.

The microscopic abnormalities that may be seen in Down's syndrome are not specific. They include decreased numbers of neurons in the third cortical layer, cerebral

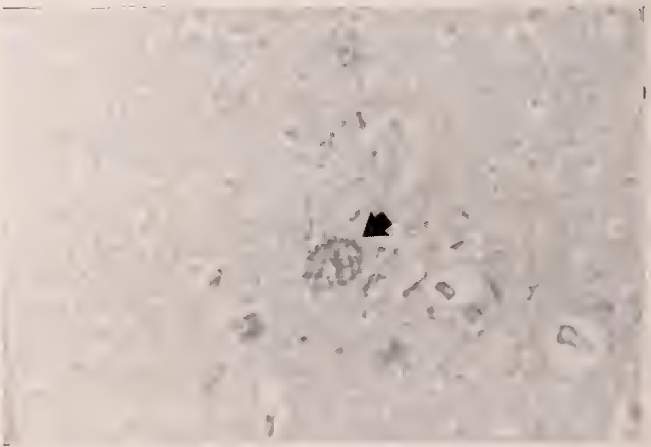

FIGURE 3. Senile plaque (arrow) in frontal cortex attributable to Alzheimer's disease (Jamarri stain; original magnification × 100).

calcifications, decreased myelin, reduced numbers and complexity of dendrites, and white-matter gliosis. Middle-aged patients with Down's syndrome generally show clinical signs of Alzheimer's premature dementia, which are histologically reflected by the presence of senile plaques and neurofibrillary changes.

Down's syndrome is characterized by chromosomal abnormalities which include trisomy of the 21st chromosome, translocations, and mosaicism. Trisomy 21 occurs in over 90% of cases, translocations in 2–4%, and mosaicism in 2–3%. Most familial cases are associated with the translocations. Trisomy 21 represents the presence of a third chromosome 21. Translocation is the shifting of a segment of one chromosome into another part of a homologous chromosome or into a nonhomologous chromosome. Mosaicism is the presence in an individual of cells of differing chromosomal constitution.

The pathogenesis by which the additional genetic material can cause a wide range of abnormalities with variable expression remains unknown. It has been suggested that Down's syndrome is associated with one third of the long arm of chromosome 21, and that the occurrence of the triple dose of the gene products of this chromosome may influence the premature aging seen in Down's syndrome.

Cerebral amyloid angiopathy, also referred to as congophilic angiopathy, is a pathologic condition characterized by the deposition of amyloid in the walls of small- and medium-sized arteries in the cerebral cortex and meninges, in the absence of systemic amyloidosis.[7] This entity is one of the less common causes of massive intracerebral hemorrhage. Amyloid of the vasculature is only rarely associated with systemic amyloidosis, and in the general population is present as an incidental finding, increasing in frequency with age: about 5% of patients in the seventh decade, 9% of patients in the eighth decade, and 17% in the ninth decade, according to one report.[8]

In a series of 73 patients with cerebral amyloid angiopathy, the incidence of massive cerebral hemorrhage was 3%.[8] This idiopathic condition has been most commonly seen in association with the dementia of Alzheimer's disease, but has also been seen in cases of post-radiation necrosis, in hereditary progressive ataxia, in Creutzfeldt-Jakob disease, in the hereditary Icelandic form of amyloidosis, and in one case report from Stanford University of an instance of the entity occurring in Down's syndrome.[6]

Although this entity of cerebral congophilic angiopathy was first described in 1909 by Oppenheim[9] and was further characterized by Dirvy[10] in 1927, it has been recognized relatively recently as an important cause of spontaneous intracerebral hemorrhage. In a series by Lee et al,[11] seven of 75 cases (9.3%) of spontaneous intracerebral hemorrhage were due to congophilic angiopathy.[11] The characteristic hemorrhage resulting from cerebral amyloid angiopathy usually originates in the cortex or subcortical white matter, and dissects through the brain tissue into the subarachnoid space. Presumably, the amyloid deposition results in vessel-wall fragility, destruction of the elastic lamina, and subsequent rupture. Destruction of the elastic lamina was demonstrated in this case.

There is a tendency in this disorder to repeated hemorrhages, varying in location and time of occurrence. Because of the peripheral location of most of these hemorrhages, in general they carry a better prognosis than do the generally deep hemorrhages associated with hypertension.

The causative factors of cerebral amyloid angiopathy are not well understood. One study of patients with the hereditary Icelandic form of the disease showed some evidence of lymphocyte dysfunction, especially an imbalance of lymphocyte subsets, reduced T-cell mitogen reactivity, and aberrant membrane properties. The author postulated an immune dysfunction perhaps related to a membrane defect affecting cell-cell interaction.[12] In one series, 92% of patients with cerebral amyloid congophilic angiopathy had Alzheimer's disease.[13] The nature of the association remains unclear. A possible direct link has been suggested by the similarity in the immunologic stains between the amyloid cores of the senile plaques and that of the amyloid in the vessels.

The clinical importance of congophilic angiopathy as a cause of spontaneous intracerebral hemorrhage is demonstrated in several series by the fact that congophilic angiopathy has been implicated as the cause in 5–10% of cases of spontaneous intracerebral hemorrhage, an incidence second only to hypertension.

In summary, this 53-year-old woman with Down's syndrome and characteristic pathologic changes of Alzheimer's dementia died of a massive intracerebral and subarachnoid hemorrhage, attributable to cerebral amyloid (congophilic) angiopathy in which the deposition of amyloid in the arterial walls predisposed to vessel rupture.

Dr Whelan: We can't make the diagnosis of Alzheimer's disease without good clinical evidence of dementia, in addition to the characteristic neuropathologic changes. The statements about the frequency of Alzheimer's disease in the population of patients with Down's syndrome have typically been inferred from the pathologic material, without good or established correlations by most authors that there was accompanying clinical evidence of dementia.

One author has even considered that in Down's syndrome there is a peculiar dissociation between the presence of plaques and tangles and the clinical dementia, reflecting double-gene dosage or else reflecting inadequate clinical tools to detect dementia in this population.[4]

John Davis, MD (Attending Physician): I assume that the age of 53 years for a patient with Down's syndrome might be equivalent to age 83 to 93 in a patient without that syndrome. This patient lived a long time for someone with Down's syndrome. The corollary question is, how often does one find a "normal" autopsy in a patient with Down's syndrome? Congenital heart disease is a frequent finding; the patients are also predisposed to a variety of infectious, inflammatory, and neoplastic disorders. It would seem to be a rarity that such a patient would live long enough to develop cerebrovascular amyloidosis and to die of one of its complications.

Dr Alpern: Before the conference, some of the staff from the Pathfinder Village were commenting on the fact that they have significant numbers of patients who are in the older age group for Down's syndrome. Could you give us further comment on that matter, as to how many of your patients with that condition are over the ages of 30 or 40?

Ms Kessler: As previously mentioned, about 35 patients are over 30 years of age. We have several who are in their 40s.

Dr Alpern: As was indicated, the incidence of Alzheimer's disease and congophilic angiopathy in older patients with Down's syndrome is extremely high.

William B. Guiney, Jr, MD (Attending Pathologist): Did you find evidence of other areas of previous hemorrhage, either hemosiderin or lacunae?

Dr Alpern: No, I found no evidence of previous hemorrhages, though as I indicated, these patients are prone to have recurrent hemorrhages. Apparently, this patient's first hemorrhage was so massive that she did not survive it.

Dr Guiney: You mentioned that 5–10% of cerebral hemorrhages were associated with congophilic angiopathy. What percentage of individuals of that same age dying from other causes have cerebral congophilic angiopathy?

Dr Alpern: One study[8] indicated that only about 3% of the patients who have congophilic angiopathy develop massive intracerebral hemorrhage, so that 97% who have the condition do not develop that complication. The incidence of congophilic angiopathy increases with age. According to this study,[8] 9% of individuals in the eighth decade and 17% of individuals in the ninth decade had the lesion on pathologic evaluation, without any relationship to hemorrhage.

Dr Whelan: In addressing Dr Davis' question about longevity in Down's syndrome, I think that we have a changing picture here, as medical facilities improve, and there appear more and stronger advocates for the rights of handicapped persons to receive full medical treatment. The curve of mortality, however, is probably a biphasic one; children with extreme congenital heart disease or other severe immunologic burdens die within the first ten years of life. Once these hurdles are cleared, then the curve of mortality more closely approaches the normal, although probably not fully comparable to those of age-matched persons in the population without Down's syndrome.

FINAL DIAGNOSES

Intracerebral and subarachnoid hematoma, large, due to cerebral amyloid (congophilic) angiopathy, left frontal lobe, with secondary mid-brain hemorrhage.

Down's syndrome.

Alzheimer's disease, brain.

Marked chronic superficial gastritis, stomach.

Diverticulosis, colon.

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Bile duct carcinoma simulating choledocholithiasis

JOSEPH N. T. DYTOC, MD, FERNANDO V. ONA, MD

Primary biliary carcinoma has been among the most difficult malignancies to diagnose and treat successfully. At the time of diagnosis, curative resection is achieved, at best, in only about 15% of cases with proximal duct tumors, in contrast to 78.5% of distal duct tumors.[1,2] However, the overall prognosis remains poor.

Often, the onset of illness is insidious. A syndrome of obstructive jaundice is the most common presentation.[1-4] Cholangiography is the most useful procedure for determining the site, the extent, and often, the etiology of obstructive jaundice.[5,6] The cholangiographic appearance of primary bile duct carcinoma is usually that of a circumferential stenosis of the duct. The stricture varies in length and degree of narrowing, and the obstructive margin tends to be fixed, abrupt, and irregular (Fig 1).[3,7-10] Rarely, the tumor may infiltrate the biliary ducts, producing an intraluminal polypoid or papillary appearance of the lesion.[11-13] In this instance, the cholangiographic picture can be deceptive and may even mimic benign biliary tract disease. This feature is illustrated in the following case of a carcinoma of the distal common bile duct masquerading as a calculus on endoscopic retrograde cholangiopancreatography (ERCP).

CASE REPORT

A 60-year-old man was admitted to the hospital with a one-month history of painless jaundice, dark-colored urine, and weight loss. His medical history was unremarkable. On physical examination, the patient was afebrile and had generalized jaundice and occult blood in the stools. The remainder of the examination was unremarkable. Pertinent laboratory findings included bilirubin of 16.2 mg/dL (normal, < 1.5 mg/dL), with direct bilirubin, 13.1 mg/dL (normal, 0–0.4 mg/dL); alkaline phosphatase, 562 units/L (normal, 45–110 U/L); aspartate transaminase, 205 U/L (normal, 20–45 U/L); alanine transaminase, 212 U/L (normal, 15–85 U/L); cholesterol, 261 mg/dL (normal, 140–280 mg/dL); albumin, 2.4 mg/dL (normal, 3.5–5.0 mg/dL). Bile was present in the urine.

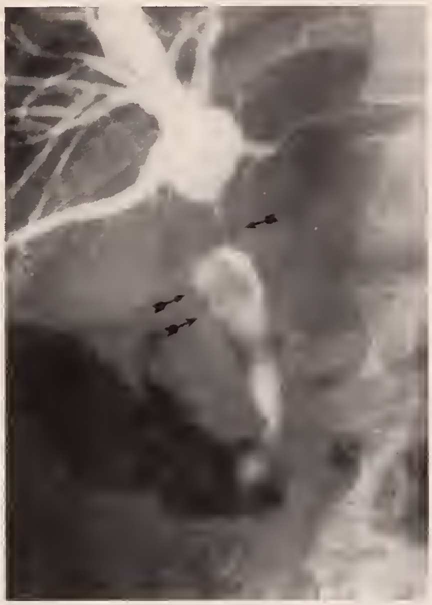

FIGURE 1. Strictured appearance of a proximal bile duct carcinoma (single arrow) with a polypoid intraluminal extension (double arrows) (courtesy of Jovitas Skucas, MD, University of Rochester School of Medicine and Dentistry, Rochester, NY).

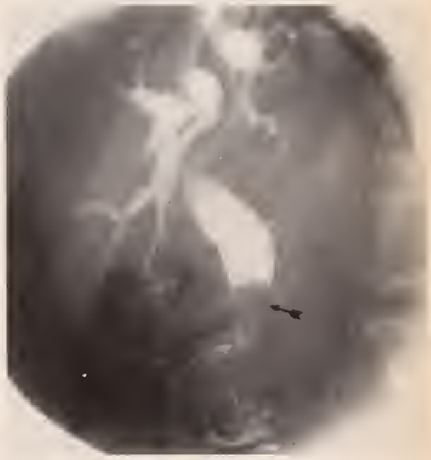

FIGURE 2. Retrograde cholangiogram showing a rounded lucent defect at the distal common bile duct (arrow) simulating an impacted calculus.

Ultrasonography revealed dilation of the biliary tree extending down to the distal common bile duct. The gallbladder was only slightly dilated. Although no stones were noted in the gallbladder, the presence of a calculus was suggested at the distal common bile duct. The pancreas appeared normal. A computerized axial tomographic (CAT) scan demonstrated mild intrahepatic biliary dilation with a normal-appearing pancreas. No abnormal calcifications were noted in the region of the gallbladder or common bile duct. An ERCP showed proximal dilation of the common bile duct and a rounded, well-demarcated lucent defect within the distal common bile duct (Fig 2). The superior margin of the lesion was angular and simulated the appearance of an impacted calculus causing almost complete obstruction of the distal common bile duct. No attempt at sphincterotomy was made, since the clinical history was thought to be more consistent with a malignant obstruction.

At surgery, no calculus was identified on choledochotomy. There was, however, a hard, dense, fibrotic lesion that was obstructing the distal common bile duct. Frozen sections from curettage specimens of the lesion revealed papillary structures embedded in a dense fibrous matrix. A choledochoduodenostomy with cholecystectomy was performed. Permanent sections from the previous curettage material later revealed a well-differentiated papillary adenocarcinoma (Fig 3). Sections from the gallbladder did not show any evidence of cancer. The cystic duct and a few lymph nodes excised from the region of the gallbladder were free of tumor.

The patient declined a second operation for possible curative resection. After surgery, radiation therapy with radiosensitizing chemotherapy using mitomycin-C and 5-fluorouracil was administered. The patient has remained well two years after surgery.

DISCUSSION

Biliary carcinoma is an uncommon,

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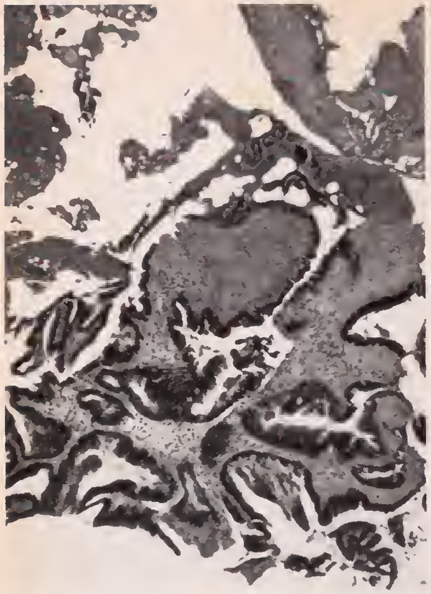

FIGURE 3. Microscopic sections of curettings from the distal common bile duct showing a well-differentiated papillary adenocarcinoma (hematoxylin-eosin stain; original magnification × 100).

TABLE I. Symptoms of Adenocarcinoma of Bile Duct (259 Patients)[1-4]

Symptoms	Patients with Symptoms
Jaundice	91%
Weight loss	57%
Abdominal pain	51%
Nausea/vomiting	51%
Acholic stools	50%
Dark urine	50%
Fatigue	50%
Pruritus	46%
Anorexia	31%
Fever/chills	20%

if not rare, type of tumor occurring in 0.01%–0.85% of autopsy subjects.[1,5,11] The peak incidence occurs among persons between 60 and 70 years of age.[1,3,14,15] There is a male preponderance, with a male-to-female ratio of 3:2.[5]

A definite causative factor has not been identified. However, a relationship has been suggested between this condition and such diseases as ulcerative colitis,[3,5] parasitic infections of the biliary tract such as *Clonorchis sinensis*[16] and *Schistosoma japonicum*,[3] congenital anomalies such as choledochal cyst,[17] and administration of Thorotrast.[18] Bile duct carcinoma and cholelithiasis have been estimated to coexist in 20% to 57% of patients; however, no apparent difference has been reported between the incidence of calculi in patients with extrahepatic bile duct carcinoma and the expected incidence of calculi in comparable age groups.[5,19]

In a review of four series reporting 259 cases of bile duct tumors,[1-4] jaundice was the most common finding heralding the onset of this illness. The symptoms are presented in Table I. Ninety-one percent of the patients had jaundice. Weight loss was the next most common symptom (57%). Abdominal pain was present in 51% and was accompanied by nausea and vomiting. Acholic stools and dark urine accompanied clinical jaundice in 50% of cases. Pruritus, which occurred concomitantly with abdominal pain or jaundice, was noted in 46%, and was most helpful in directing attention to the biliary tract. Chills and fever, which suggested sepsis, were found in 20% of cases.

The physical findings in the same four series (Table II) showed hepatomegaly as the most common abnormality (52%). Less than 10% of patients presented with splenomegaly or ascites. Occult blood in the stools was found in more than a third of the patients (37%). Abdominal tenderness was present in 22%. A palpable abdominal mass was found in 17% and was often attributed to a distended gall bladder.

The laboratory data were strongly indicative of obstruction to bile flow. As shown in Table III, the most frequent abnormal laboratory findings included elevations of alkaline phosphatase (95%) and serum bilirubin (95%). Alkaline phosphatase ranged from 184 to 1,850 IU/L with simultaneous total bilirubin levels of 3.4 to 47.2 mg/dL. Modest elevations of the transaminases, low serum albumin, and prolonged prothrombin times were noted in the majority of patients. Serum cholesterol was elevated in about half of the cases.

In most cases, the initial evaluation of patients with obstructive jaundice begins with some noninvasive diagnostic tests such as ultasonography or computed tomography, which can confirm the existence of dilated intrahepatic ducts and, occasionally, provide information regarding the gallbladder and the extrahepatic ducts.[20,21] Table IV shows the diagnostic studies employed in 146 patients with bile duct carcinoma.[2-4] Percutaneous transhepatic cholangiography was performed in 61 patients and was helpful in making a diagnosis in 96% of these cases. It has been the procedure of choice when proximal lesions are suspected. It can reveal the site of tumor and define the anatomy of the ducts proximal to the lesion. When, on the basis of ultrasound or CT scan, a distal obstruction is suspected because of a dilated gallbladder, a dilated extrahepatic bile duct, the possibility of a peri-ampullary lesion, or in patients with severe ascites or coagulopathy, ERCP would be the procedure of choice.[5] ERCP was performed in 19 patients (Table IV) and was helpful in 68% of the cases. It provides information about the biliary anatomy distal to the tumor. In addition, it has the advantage of direct visual assessment of the duodenum and the ampullary area, with the capability of obtaining adequate tissue samples for brush cytology and biopsy for immediate histologic diagnosis. It can be useful in defining the anatomy of the pancreatic duct to help differentiate between pancreatic malignancy or benign conditions, such as chronic pancreatitis. ERCP provides the added benefit of possible sphincterotomy and endoscopic decompression of the obstructed bile duct using the biliary catheter or stents for biliary drainage. However, depending on the degree of the malignant stricture, it may fail to delineate the ducts proximal to the tumor.

TABLE II. Physical Findings of Adenocarcinoma of Bile Duct (259 Patients)[1-4]

Findings	Patients with Symptoms
Hepatomegaly	52%
Heme-positive stools	37%
Abdominal tenderness	22%
Abdominal mass	17%
Splenomegaly	8%
Ascites	2%

TABLE III. Laboratory Findings in Adenocarcinoma of Bile Duct[3]

Laboratory Study	Abnormal
Alkaline phosphatase	95%
Direct bilirubin	95%
Total bilirubin	93%
Lactate dehydrogenase	87%
Serum glutamic oxaloacetic transaminase	82%
Albumin	72%
Prothrombin time	61%
Cholesterol	55%

Ultrasonography and computed tomography were often useful in determining the extent of local invasion[20,21]; however, in some cases, the actual site of obstruction could not be adequately visualized by these techniques.[3]

Radionuclide liver scanning and upper gastrointestinal barium studies were generally not helpful in this series.

Although not used routinely, arteriography was occasionally helpful in establishing the extent of local tumor invasion and nonresectability by revealing vascular encasement or portal vein invasion.[22]

Recently, percutaneous bile duct cytologic studies have proven useful for diagnosis in many patients.[23] In comparison, percutaneous liver biopsy had failed to show tumor even in patients found to have liver metastases at surgery.[2]

In a review of 95 cases, Alexander and coworkers[1] found that adenocarcinoma was the predominant histologic type (Table V). These tumors comprised 82% of the cases. Thirteen percent of adenocarcinomas were well differentiated, 47% moderately differentiated, and 40% poorly differentiated. Other unusual histologic types included adenosquamous carcinoma, leiomyosarcoma, and mucoepidermoid carcinoma, comprising about 4% of the cases.

Bile duct carcinoma frequently evokes a strong desmoplastic reaction,[3] giving rise to the sometimes rock-hard consistency of the lesion. In addition to this sclerosing characteristic, this carcinoma also tends to infiltrate in a submucosal fashion, so that adequate sampling and interpretation of biopsies on frozen section may be extremely difficult.[5] These features were demonstrated in the case presented here.

Tumors of the bile duct have little tendency to metastasize, although spread to contiguous structures such as the liver, pancreas, gallbladder, and duodenum is not that uncommon.[5] The most common sites of distant metastasis include the regional lymph nodes, lungs, kidneys, and peritoneum.[3]

Table VI shows the distribution of the lesions according to location based on a series of 109 cases by Alexander and coworkers.[1] Most of the tumors involved the proximal bile ducts (76%). Tumors of the distal and mid-portion of the ducts comprised 24% of the cases. Proximal duct tumors were commonly situated at the bifurcation of the common hepatic duct, with some extension into either the right hepatic duct or left hepatic duct.

TABLE IV. Diagnostic Studies (146 Patients)[2-4]

Procedure	Number Performed	Number Helpful
Transhepatic cholangiography	61	59
Ultrasonography	24	17
Liver-spleen scan	23	11
Upper gastrointestinal series	20	9
Endoscopic retrograde cholangiopancreatography	19	13
Computed tomography	19	13
Percutaneous liver biopsy	9	6
Percutaneous bile duct biopsy	8	8
Arteriography	4	2

TABLE V. Histology of Bile Duct Carcinoma (95 Cases)*

Histology	Frequency
Adenocarcinoma	82%
Well differentiated (13%)	
Moderately differentiated (47%)	
Poorly differentiated (40%)	
Adenosquamous carcinoma	1.8%
Leiomyosarcoma	0.9%
Mucoepidermoid carcinoma	0.9%

* From Alexander, Rossi, and O'Bryan et al[1] (with permission of the Lahey Clinic).

TABLE VI. Location of Carcinoma of the Bile Ducts (109 Patients)[1]

Location	Frequency
Proximal duct	76.0%
Bifurcation, right and left hepatic duct	72.0%
Bifurcation alone	9.5%
Bifurcation, left hepatic duct	7.0%
Bifurcation, right hepatic duct	1.0%
Right hepatic duct	3.5%
Left hepatic duct	2.0%
Common hepatic duct	3.5%
Mid-portion	11.0%
Distal	13.0%

TABLE VII. Relation of Gross Shape of Cancer to Its Histologic Type (43 Cases)*

Gross Shape and Histology	Frequency
Papillary or polypoid	
Papillary adenocarcinoma	100%
Nodular	
Papillary adenocarcinoma	53%
Tubular or poorly differentiated	47%
Flat or ulcerated	
Papillary adenocarcinoma	11%
Tubular or poorly differentiated	89%

* From Kozuka, Tsubone, and Hachisuka[14] (with permission of the authors).

A relationship between the gross shape of the tumor and the histologic type has been reported by Kozuka et al.[14] The shape of the cancers was divided into three types: papillary or polypoid; nodular; and flatly elevated or ulcerated (Table VII). All the papillary or polypoid tumors were adenocarcinomas on histologic evaluation, while most of the flatly elevated or ulcerated tumors were tubular adenocarcinoma or poorly differentiated carcinoma. The nodular tumors were divided between papillary adenocarcinoma and tubular or poorly differentiated carcinoma. The papillary or polypoid cancers were mostly noninfiltrating, ie, they did not penetrate beyond the fibromuscular layer of the bile ducts; while most cases of nodular and flat cancers spread into the deeper layers beyond the fibromuscular layer.[14]

Since radical surgery tends to offer the best prognosis,[1] the aim of therapy is resection of the tumor whenever feasible. The surgical approaches are dictated by the extent and location of the lesion as well as the condition of the patient.

In cases in which the risk of surgery is acceptable, surgical exploration is favored to obtain a tissue diagnosis, determine resectability, and decide preferable therapy.[5] In poor risk patients or in patients without duodenal obstruction in whom nonresectability can be established without laparotomy, it is reasonable to explore nonoperative methods for relieving jaundice.[24–27] Depending on the location of the tumor, placement of a drainage tube and subsequently a prosthesis through the tumor can be achieved either via the percutaneous transhepatic route[25] or via a duodenoscopic method of intubation.[24,28,29]

The latter technique is probably more feasible when the tumor is located more distally and has the advantage over the percutaneous route of sparing the patient from a permanent external fistula when the prosthesis cannot be placed.[24]

Proximal duct tumors are resectable in about 10% of cases.[1,2] The surgical approaches for these tumors consist of partial hepatectomy with reconstruction by hepatico-jejunostomy or by skeletonization with resection.[5,30] These types of resection are performed after making adequate assessment of resectability of the proximal tumor by careful dissection and exposure of the common duct, hepatic duct bifurcation, the right and left hepatic ducts, and after circumferential dissection of the portal vein, hepatic arteries, or its branches. Biopsy of the common duct and cystic duct nodes should be performed. The indications for liver resection should include the following criteria: 1) when the proximal tumor extends to the segmental intrahepatic ducts of either the right or left lobes with sparing of the segmental ducts of the opposite lobe, resection may be possible; 2) when there is no invasion of the portal vein and hepatic arteries; 3) when the distal extension of the tumor is limited to the common duct above its pancreatic portion; and 4) when metastasis is absent.[5] When the tumor is unresectable, palliative decompression can be performed using transtumor intubation techniques[27] or techniques of intrahepatic cholangiojejunostomy.[31–33] The mortality associated with surgical resection has been estimated to be approximately 16%.[1] Palliative percutaneous drainage is the treatment of choice for unresectable proximal tumors when the patient is considered to be at high risk for palliative surgery.[5] The five-year survival rate for proximal tumors has been estimated to be about 25%.[1]

Mid-duct tumors and distal duct tumors have resectability rates of 33% and 100%, respectively.[1] Mid-duct tumors, when resectable, can be extirpated by skeletonization and resection or by pancreaticoduodenectomy.[5] The mortality from these surgical procedures has approached 25% and there have been no five-year survivors.[1] Distal duct tumors can be resected adequately by the Whipple's procedure,[5,30] with or without pyloric preservation. The mortality rate reported by Alexander et al[1] in their report of 14 patients with distal duct tumors treated with surgery between 1965 and 1978 was 21% with a five-year survival rate of 20%. However, the current mortality associated with pancreaticoduodenectomy is less than 5%. The recent report by Roberts[4] showed a 1–2% mortality rate in 17 patients with ampulla of Vater carcinoma treated with Whipple's procedure. For unresectable mid-duct and distal duct tumors, palliative bypass surgery can be achieved by a hepaticojejunostomy proximal to the site of obstruction or by one of the transtumor intubation techniques via either the percutaneous or the duodenoscopic method.[5,24,25,28,29]

In addition to the influence of tumor site and resectability, tumor grade has also been shown to affect survival. The five-year survival of patients with well-differentiated tumors was reported to be 15%, compared to zero in patients with poorly differentiated tumors.[1]

The issue of the effectiveness of adjuvant therapy on bile duct cancer remains unsettled. Clinical experience has been reported in the use of postoperative radiation using implantable iridium-192,[34] external beam radiation,[2] and adjuvant chemotherapy.[4] The results have been encouraging in some patients, although further experience is required to assess the benefits of these treatments.

In summary, we have presented a case of a papillary adenocarcinoma of the distal common bile duct simulating a calculus by ERCP to illustrate a potential pitfall in the cholangiographic diagnosis of these obscure tumors. An awareness of this rare radiologic appearance of these tumors will be helpful as more cholangiographic studies are performed in the preoperative evaluation of obstructive jaundice.

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Carcinoma of the colon associated with systemic sclerosis

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The association between an increased incidence of malignancy and the development of a connective tissue disease is well established in polymyositis/dermatomyositis. The increased risk of malignancy in systemic sclerosis (scleroderma) is not as clear, though an association with lung, esophageal, and breast carcinomas has been noted.[1-8] We report a case of cutaneous sclerodermatous changes occurring within six months of an adenocarcinoma of the colon.

CASE REPORT

A 77-year-old man presented in December 1987 with the complaint of abdominal pain for the past several months. A right lower quadrant mass and anemia were present. Barium enema revealed a fungating mass at the area of the ileocecal valve. At surgery, the tumor mass was adherent to the right ureter, and there was lymph node involvement. Pathologic evaluation documented a moderately differentiated adenocarcinoma of the cecum with transmural infiltration into pericolic fat, with metastasis to eight of nine lymph nodes. The patient was referred for chemotherapy.

Treatment with 5-fluorouracil (5-FU), 1,500 mg intravenously, was initiated on April 11, 1988, and 750 mg was administered weekly thereafter, except for a dose of 1,500 mg given on May 26, 1988. With the second infusion, the patient began to complain of pruritus and a tight feeling in the skin around the infusion site. "Tension of the skin" over body surfaces was noted on May 26, 1988, by a house officer. A referral to the rheumatology clinic was made in June 1988.

At this time the patient complained of pruritus with tightening of the skin of the hands and legs. He denied Raynaud's phenomenon, dysphagia, or change in bowel habits. He did have weight loss of 10 lb over the past several months.

The physical examination was remarkable for advanced skin changes, compatible with systemic sclerosis of the hands and fingers, face, trunk, upper and lower arms, as well as the thighs and lower legs. Hand grip was poor. There was no telangiectasia. Blood pressure and urinalysis were within normal limits. Results of anti-nuclear (ANA) and anti-centromere antibody tests were negative. The carcinoembryonic antigen level was 0.3 ng/mL (normal). The patient refused a skin biopsy or esophageal motility studies.

Photographs of the patient taken on the previous Thanksgiving (November 1987) revealed normal appearance of the face and hands, compared to photographs taken in June 1988 (Fig 1).

The patient was begun on nifedipine, 10 mg three times a day, continued with the 5-FU because of the severity of his malignancy. Over the next two months, he experienced dysphagia for solids, Raynaud's phenomenon, and multiple telangiectasia. The patient died in February 1989 of metastatic colon cancer. Permission for a postmortem examination was denied.

DISCUSSION

Connective tissue diseases may precede, accompany, or follow the presentation of a malignancy in a given patient. In the prototypic disease, adult-onset polymyositis/dermatomyositis,

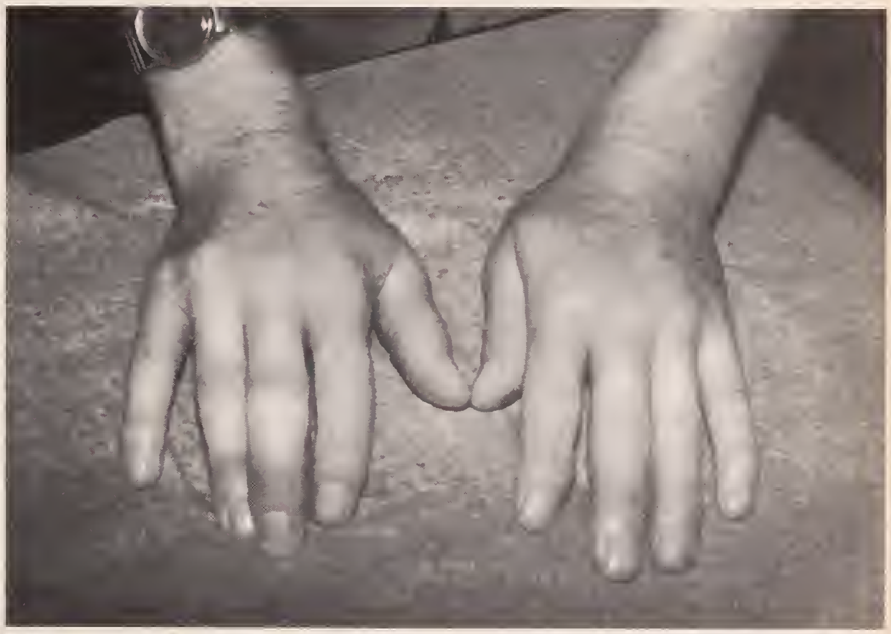

FIGURE 1. Hands demonstrating skin tightening.

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malignancy is of sufficient significance to warrant continued surveillance for an occult neoplasm.[9] Systemic sclerosis has been associated with an increased risk of cancer, predominantly lung neoplasias.[4,6,10] These have been reported in the setting of long-standing pulmonary fibrosis. Esophageal carcinomas, squamous cell adenocarcinoma, and undifferentiated carcinoma have also been noted.[8,11]

It has been suggested that within the lung and esophagus, chronic fibrosing changes may predispose to metaplasia and neoplasia. However, uterine, rectal, and breast malignancies have also been reported in association with the temporal development of systemic sclerosis.[1,5,12,13] Conflicting epidemiologic studies have shown no increased relative risk[14] or an increased relative risk[1,4,5] of the development of malignancy in patients with this disease. Basten and Bonnin[15] noted that the majority of neoplasms were adenocarcinoma and carcinoid tumors, and that 100% of the patients had cutaneous manifestations but less than half had systemic disease.

In the case presented here, the onset of sclerodermatous cutaneous involvement followed within six months of the diagnosis of advanced colonic adenocarcinoma. Serologic studies (ANA and anti-centromere antibody) were negative, as in patients exposed to vinyl chlorides in whom scleroderma-like symptoms developed. However, the incidence of antinuclear antibodies in scleroderma patients with and without malignancies was similar in the report by Roumn and Medsger.[4]

Chemotherapeutic agents, namely bleomycin, have been noted to cause scleroderma-like skin changes.[16] We could find no reports of 5-FU associated disease and, in fact, Casas et al[17] reported improvement in 12 women with systemic sclerosis treated with this drug. It seems more likely, therefore, that this patient's disease was temporally related to the adenocarcinoma of the colon, though an association with 5-FU cannot be excluded.

Speculation as to altered immunologic response due to tumor antigens and the development of connective tissue diseases remains unconfirmed. Demonstration of an association with chemotherapeutic agents other than bleomycin requires further surveillance.

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Systemic embolization from left atrial ball thrombus

The case for early surgical intervention

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Left atrial ball thrombus is an uncommon but potentially catastrophic occurrence. Transient obstruction of the mitral valve can produce acute cardiac decompensation[1,2] or syncope.[3,4] Systemic embolization, occasionally multiple, has been described.[5] Obstruction of the mitral valve may not resolve and can result in cardiovascular collapse with sudden death.[6] Because of these potential outcomes, some authors[1,5] have recommended that such thrombotic masses, once they are detected, should be surgically removed as soon as possible.

We describe a patient who presented with a transient stroke-like syndrome, and who was found to have a loose left atrial mass by echocardiography. The patient subsequently experienced multiple systemic emboli during an in-hospital observation period, while waiting for her condition to stabilize prior to surgery.

Case Report

A 69-year-old woman was admitted to another hospital in August 1988, after becoming unresponsive at home. She had had mitral stenosis due to rheumatic fever for many years, and she was on long term anticoagulation therapy with coumadin because of atrial fibrillation. Approximately four weeks prior to admission she stopped taking coumadin.

On admission, right hemiparesis was noted. The patient was treated with intravenous steroid therapy, and she promptly recovered most limb function. An echocardiogram, performed with the Interspec Model XL3 sector scanner (Interspec Inc, Consho-

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hocken, Pa) demonstrated moderate left atrial dilation. Significant mitral stenosis, with a valve area of approximately 0.9 cm^2, as determined from the mitral valve diastolic Doppler pressure half-time, was also observed. Additionally, there was a free-floating body of approximately 1-1/2 cm × 1-1/2 cm within the left atrial cavity (Fig 1). This was believed to be a detached thrombus. The mass became impacted within the mitral valve orifice every three to four beats (Fig 2); no symptoms were associated with these impactions. Additional echocardiographic findings included marked dilation of the right ventricle and normal left ventricular size and function. The patient was transferred to St Joseph's Hospital Health Center for further evaluation and therapy.

Pertinent cardiopulmonary findings on admission included clear lungs and an irregular rhythm, with a ventricular rate of 65 beats/min. A grade II/VI systolic murmur was heard at the apex, and it radiated into the axilla. A diastolic rumble of variable intensity was also heard at the apex. Neurologic examination was normal with the exception of a finding of a mild degree of confusion.

Cardiac catheterization was performed on the day following admission, primarily to assess the coronary arteries. No significant coronary atherosclerosis was present. It was decided at that time to introduce anticoagulation therapy with heparin and to optimize the patient's medical status over the next several days with the eventual intent of performing sternotomy for evacuation of the left atrial mass and replacement of the mitral valve.

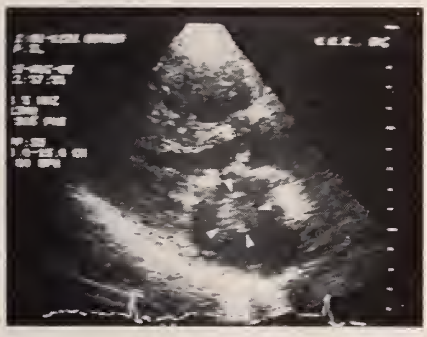

FIGURE 1. Parasternal long-axis view showing dilated left atrium, calcified stenotic mitral valve, and a free-floating mass within the atrial cavity (small arrowheads).

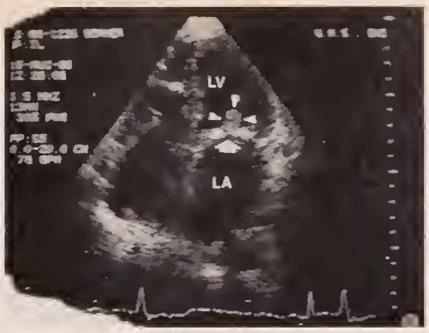

FIGURE 2. Apical four-chamber view showing temporary impaction of the free-floating mass (outlined by small arrowheads) within the stenotic mitral valve orifice (large arrow) (LA = left atrium; LV = left ventricle).

During this period there was fluctuation in her mental status, particularly in memory and orientation. A computed tomographic scan of the brain showed multiple small areas of what was interpreted to be recent infarction resulting from multiple emboli. On the sixth hospital day, the patient complained of sensations of "tiredness" and coldness of her left leg. Plethysmographic pulse volume recordings were consistent with complete obstruction of the left superficial femoral artery. An embolectomy was performed that evening, and the thrombotic origin of the obstructing mass was confirmed on histologic analysis. The following day the patient underwent median sternotomy with left atriotomy. Several pieces of thrombus were removed from the atrial appendage, which was then sewn over. The mitral valve was excised and replaced with a number 29 porcine bioprosthesis.

DISCUSSION

A free-floating, left atrial thrombus can present with unexpected sudden death when permanent mitral valve obstruction occurs, or with intermittent acute heart failure when occlusion is transient. Multiple systemic emboli from such thrombotic masses have also been described. Clinically, the patient described here appears to have experienced at least three discrete periods of embolization. The first included her original presentation with right hemiparesis, the second, her fluctuating in-hospital mental status, attributed to embolic cerebral showers, and the third, her episode of left superficial femoral artery obstruction. The absence of a loose thrombus at the time of atriotomy suggests that the material removed from the femoral artery comprised the bulk of the original left atrial mass. This presumably had been whittled away progressively, releasing multiple small emboli, until the thrombus itself was small enough to pass through the stenotic mitral valve orifice and travel to the periphery.

Complications, such as those found in this patient, would appear to be inevitable in cases of free-floating intra-atrial masses. Therefore, in the absence of significant medical instability, delaying surgery does not seem justified. Indeed, this patient experienced most of her embolic complications while in the hospital, after identification of the thrombus, and while under surveillance and on therapy intended to optimize her medical status in preparation for surgery. We agree with previous authors in suggesting that once identified, free-floating intra-atrial masses should be promptly removed.

Acknowledgments. The authors thank Karen Semak for her expert word processing of the manuscript, and Alessandro Giambartolomei, MD, for reading the manuscript and providing helpful and welcomed criticism.

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Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Student participation in mixed gender sports competition

TO THE EDITOR: As a registered nurse working in a pediatric outpatient clinic and as a mother of three teenaged children, I looked forward to reading the guidelines, "Student participation in mixed gender sports competition,"[1] with anticipation and interest. Instead, I was disappointed.

Because these guidelines were prepared by the Committee on School Health and Sports Medicine and approved by the Council of the Medical Society of the State of New York, they will be much sought after by parents and school personnel as they move toward incorporating mixed gender sports competition into their programs. The guidelines, therefore, should provide helpful recommendations, and they do when considering physical size, physical fitness, past activity and experience, and protective equipment.

They are not helpful when considering adequate locker rooms, and the attitudes of the coaches and team members. If locker room facilities are not adequate, these guidelines should encourage that changes be made to make them adequate. A coach who has an attitude incompatible with the participation of members of the opposite sex on the team should be counseled about this attitude. If this person, a role model for the young team members, cannot change then she/he should be replaced as coach. It is also important to monitor the attitudes of the team members and put to rest any of their misconceptions about mixed gender sports competition. The attitudes cultivated by these young people on the playing fields will be the attitudes that accompany them for the rest of their lives. In these areas, the guidelines do nothing to encourage mixed gender sports competition. Without saying so, they really favor the status quo.

What I find most disappointing about these guidelines is the very subtle sexist tone that pervades them. Boys are superior athletes and girls are put at risk if they play sports with boys. Girls have to measure up to boys in order to play on their team. I would like to see these guidelines reconsidered and rewritten in order to make them what they are supposed to be—guidelines for people seeking to make mixed gender sports competition an option for their children.

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1. Student participation in mixed gender sports competition. *NY State J Med* 1989; 89:526.

In reply. The guidelines were written to encourage mixed gender participation in a responsible manner, paying proper respect to the health and well being of the participants.

Adequate locker rooms and attitudes of players and coaches are factors that must be considered by a physician subjecting an athlete to mixed gender competition. No attempt was made to resolve unsuitable conditions and attitudes since this is a matter for local schools.

Prior to puberty the risk of bodily harm in mixed gender contact sports competition is equal for both sexes. Following puberty girls have less lean body mass than boys. Girls will absorb more of a force created by a collision. Females are relatively weaker than males in upper body strength. Postpubertal females on the average have 15% to 25% less aerobic capacity than males. These are scientific facts, not legal or social issues, and are not subject to opinion. We appreciate the highly developed skills of both male and female athletes but wish to point out the greater chance of the female being injured in contact sports where both males and females participate.

The Committee on School Health and Sports Medicine encourages greater interest in those sports where participants of both sexes and all ages may compete in safety and with a minimum of risk. These sports are the endurance and noncontact sports.

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Physician recredentialing

TO THE EDITOR: I believe that in their reply to Dr Atkinson's letter, Drs Gelhorn and Cherkasky devalued many of the best points he made.[1-2] I feel that his criticism was well thought out and presented. They did not answer what he said about residency programs and the need to better address some of the problems at the training level. Certainly this would be more corrective than setting up yet another certification examination.

Drs Gelhorn and Cherkasky cite the 16 specialty boards that have a recertification process. Noteworthy is the fact that in inviting family practitioners to retake their boards, the American Board of Family Practice considers that there may be certified FPs who disagree with the process.

Finally, most physicians do not doubt that Dr David Axelrod, New York State's Commissioner of Health, would be subjected to recredentialing or that he would pass whatever test is devised. This, however, would not put an end to his regulatory fervor which already has done so much damage in this state.

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Eliminating the requirement for medical libraries in hospitals

TO THE EDITOR: Dr Peter Farnsworth has sounded a very important alarm in his commentary, "The elimination of the requirement for medical libraries in hospitals."[1]

It is shocking that both the Health Care Financing Administration and the New York State Department of Health wish to eliminate the requirement that hospitals maintain a medical library. It seems almost too obvious to state that a medical library is a necessary but not a sufficient condition for assuring continuing education of physicians. Although the Department of Health is correct in its conclusion that existence of a medical library does not guarantee the quality of patient care, it ignores the fact that ready access to a medical library is one essential component of continuing medical education and therefore of high quality of patient care. It would seem that the health department has failed to recognize the distinction between "necessary" and "sufficient" conditions.

We must hope that Dr Farnsworth's warning will be heeded.

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[1] Farnsworth PB: The elimination of the requirement for medical libraries in hospitals. *NY State J Med* 1989; 89:601-602.

TO THE EDITOR: I am writing to comment and to express appreciation to Dr Peter B. Farnsworth for his commentary on libraries in hospitals.[1] He makes a strong case for the existence of libraries to serve the educational and patient-related information needs of health practitioners. He also makes clear the relationship between competency of the physician/nurse and the quality of care provided. Current and accurate information and literature are essential to those practicing in the medical professions, for their continuing education and for the well being of patients.

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[1] Farnsworth PB: The elimination of the requirement for medical libraries in hospitals. *NY State J Med* 1989; 89:601-602.

TO THE EDITOR: Dr Farnsworth's[1] commentary in the *Journal* underscores the poor judgment shown by the New York State Department of Health in eliminating the requirement that hospitals maintain medical libraries. A hospital library is part of the central nervous system of the hospital.

Hospital libraries need to adapt to changing technology, such as the availability on-line of the National Library of Medicine and various software programs. The cost of these is modest and their use results in decreased space requirements for hospital libraries. Their use also results in a decrease in need for certain journals, reference texts, and achieves savings in personnel costs and other costs such as binding. A computer network in a hospital can bring the medical library to doctors' lounges, nursing stations, the pharmacy, and every locus where medical information is needed.

The Department of Health and the Health Care Financing Administration should require that hospital libraries expand electronically with access to the National Library of Medicine. This increased access to medical literature will reflect itself in better patient care and physician performance.

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[1] Farnsworth PB: The elimination of the requirement for medical libraries in hospitals. *NY State J Med* 1989; 89:601-602.

TO THE EDITOR: The information contained in Dr Peter Farnsworth's editorial is astounding and frightening.[1] The same line of thinking would clearly indicate that all public libraries should be closed, since there is no positive evidence that the presence of such institutions in communities enhances their cultural and intellectual climate in any fashion. Once all the libraries are closed then we can begin to close down the art museums, public symphonies, etc.

Perhaps we should feel fortunate that the Health Care Financing Administration and the New York State Department of Health only deal with the health of our society. Imagine if they had a truly free rein.

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[1] Farnsworth PB: The elimination of the requirement for medical libraries in hospitals. *NY State J Med* 1989; 89:601-602.

Gallstone appendicitis

TO THE EDITOR: Many etiologies have been described for acute appendicitis.[1] A review of major texts and current surgical literature fails to reveal any reference to gallstone appendicitis. Reported cases in the early literature were thought to be unsubstantiated.[2]

Case Report. We recently treated a 19-year-old female for sharp intermittent periumbilical pain, nausea, and vomiting. She had no history of illness relating to the biliary tract. Abdominal examination revealed tenderness in the right lower quadrant, without rebound, guarding, or palpable mass. The patient was afebrile, with a white blood cell count of 12,000/μL, and serum bilirubin, alkaline phosphatase, serum glutamic oxalo-acetic transaminase, and amylase values within normal limits. At surgery an acute, nonsuppurative appendicitis was noted. Palpation of the appendix identified a 0.6-cm hard appendicolith suspicious for a stone. Once off the operative field, the appendix was incised, revealing a stone with the consistency and color of a gallstone. The gallbladder was not explored intraoperatively in order to avoid the spread of contamination. Biochemical analysis demonstrated that the stone was composed entirely of cholesterol and bilirubin. A sonogram and oral cholecystogram failed to reveal any further evidence of cholelithiasis.

Discussion. Appendicoliths resembling gallstones have been reported. However, on analysis they invariably are found to consist of fecal material.[2] We present this case to document that gallstones can indeed precipitate appendicitis. Even in the absence of a history of symptoms referable to the biliary tract, we suggest that when appendicoliths appear to be stonelike they be sent for biochemical analysis. This should be done in order to identify those rare cases in which gallstone appendicitis may be the first indication of biliary pathology.

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[2] Fuller FW, Bernhard WG: Appendiceal calculi resembling gallstones. *Ann Surg* 1968; 167:101-105.

Open supramalleolar amputation of the infected foot: An underutilized, two-stage procedure

TO THE EDITOR: We live in a medical care climate emphasizing efficient use of hospital bed space and a growing reluctance to return a surgical patient to the operating room for any reason. Despite this, we submit a case report and opinion that the two-stage operative management of the unsalvageably infected foot remains the optimum treatment for certain patients.

Case Report. A 62-year-old diabetic

man on chronic dialysis was admitted for treatment of an infected left foot five weeks following minimal trauma to the great toe. There was a history of heavy smoking and insulin dependence. The left femoral pulse was absent, the left fourth and fifth toes were gangrenous, a lymphangitic streak was present above the ankle, and angiography confirmed left iliac artery occlusion. After three days of treatment with intravenous antibiotics without signs of improvement, the patient underwent femoral-femoral arterial bypass and open amputation of the forefoot. Despite resolution of the lymphangitis and a favorable degree of bleeding seen during the forefoot debridement, the foot infection recurred and the forefoot amputation site became gangrenous. One week postoperatively, with a patent bypass, an open supramalleolar amputation of the left foot was performed. At this surgery, pus was seen streaming from the fascial spaces of the calf. Continued intravenous antibiotics and frequent saline dressings to the open ankle-level stump allowed resolution of the infection. One week after the open supramalleolar procedure, a formal below-knee amputation was performed. This healed uneventfully and the patient was discharged five days after the below-knee amputation. He was subsequently fitted with a prosthesis as an outpatient.

Discussion. This case demonstrates a situation that most general and vascular surgeons will recognize: the unsalvageably infected foot. With adequate blood supply to the midcalf level, this situation is most commonly treated by a single-stage, closed, below-knee amputation. However, in situations where uncontrolled infection exists just distal to the below-knee site, such treatment will be accompanied by unacceptably high rates of stump infection (22%) and eventual revision to the above-knee level (11%). These infection and revision rates have been demonstrated to be drastically lowered (3% and 0%, respectively) by a supramalleolar open (guillotine) amputation that allows drainage of the infected fascial spaces and lymphatics of the calf.[1,2] Following this, the second stage of the reconstruction, the actual below-knee amputation, can usually be done about one week later.

The open ankle-level drainage operation can also be used to rid a patient with sepsis of the infective source, even when the site is severely compromised. The relatively minor first stage takes about ten minutes to perform, and allows the patient to recover from systemic sepsis before proceeding with the second stage below-knee amputation at a later date. The procedure is also useful if the patient has recently had a myocardial infarction and presents with a septic foot. In this situation, the second stage amputation is delayed until cardiac improvement permits the more major procedure.

In four years, we have performed 11 such procedures, one under local anesthesia. Nine patients underwent revision to a below-knee amputation without infection or subsequent revision to the above-knee level. Two of the 11 patients died from medical factors unrelated to the procedure before a revision was performed.

Two recent prospective studies support the use of the two-stage technique. Fisher et al[3] randomized a total of 47 patients with unsalvageable wet gangrene to either a one-stage below-knee amputation with delayed skin closure or a two-stage procedure. Wound complications developed in five patients (23%) of the one-stage group, which necessitated a higher level amputation in two patients, whereas none of the patients in the two-stage group experienced such wound complications. Desai et al[4] found the two-stage technique also useful with foot sepsis associated with ischemia. They treated all infected ischemic feet clinically judged to require a below-knee amputation by a one-stage procedure with delayed skin closure for six months (29 patients), followed sequentially by utilization of a two-stage procedure during the latter six months of the study (44 patients). Thirty-three percent of the one-stage group later required above-knee amputation, whereas only 7.7% in the two-stage group did. Despite this recent support from the literature, we believe that the supramalleolar amputation is underutilized today. We recommend open supramalleolar amputation to surgeons dealing with uncontrollable foot infections as a simple procedure which may well preserve the knee.

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ERRATUM

The January 1990 issue contained the following errors:

In the case report by Nakao, "Local low-dose infusion of streptokinase for massive pulmonary embolism when systemic thrombolysis is contraindicated (90:32-33)," the captions for Figures 1 and 2 were inadvertently transposed.

In the article by Jacobs, "Cervical angina (90:8-11)," the entry in the last line of Table V, column 2, should be 5/5.

Practicing clinical preventive medicine

Guide to Clinical Preventive Services. An Assessment of the Effectiveness of 169 Interventions. Report of the U.S. Preventive Services Task Force. Edited by Michael Fisher. 419 pp. Baltimore, Md, Williams & Wilkins, 1989. $19.95 (paperback)

It is a fact that the three major killers in the United States result from individual life style behaviors rather than from bacterial, viral, or other infectious diseases. Only one communicable disease (influenza/pneumonia) is among the top ten killers. These facts should lead to a concentration on clinical preventive services to reduce the mortality and morbidity occurrences. This up-to-date *Guide to Clinical Preventive Services* does exactly that. Arguments are strongly stated that recommend drastic changes in the practice patterns of physicians as they care for their patients:

> ... the most promising role for prevention in current medical practice may lie in changing the personal health behaviors of patients long before clinical disease develops. The importance of this aspect of clinical practice is demonstrated by a growing body of evidence linking a handful of personal health behaviors to the leading causes of death in the United States: heart disease, cancer, cerebrovascular disease, injuries, and chronic obstructive pulmonary disease.

Evidence on tobacco use unequivocally indicates that smoking contributes to one out of every six deaths in America. Injuries sustained in motor vehicle accidents account for almost 50,000 deaths per year. Clinical preventive services in those two areas alone may yield dramatic decreases in mortality statistics, in suffering, and in personal and social deficits.

How can health and medical care providers convince themselves and their patients to do the right things to protect and preserve their own physical and mental health? If the recommendations suggested in this *Guide to Clinical Preventive Services* are widely adopted in practice and in health care educational institutions, current patterns of medical/health practice and behavior would be considered out of date.

Critics may feel that the *Guide* goes too far too fast. Is it reasonable to expect "busy physicians and nurses to abandon their other clinical duties to become counselors or nutritionists?" This *Guide* posits that the "new morbidity" demands a "new paradigm" for physicians to follow in caring for their patients.

Others may feel that the limited recommendations are too conservative. However, the *Guide* authors comment that their first obligation is "to do no harm" and point out the gaps to be filled in as research provides additional information.

Can a happy medium be achieved? Let us look at a sampling of the clinical preventive interventions presented in this *Guide*.

After five years of debate, literature review, and synthesis of critical comments from expert reviewers, an independent 20-member US Preventive Service Task Force turned well-meaning verbiage into specific recommendations for practicing clinicians. Appointed by the Assistant Secretary for Health in 1984, the task force evaluated the scientific evidence and issued a report detailing recommended clinical practice patterns related to 60 targeted potentially preventable diseases and/or conditions. More than 100 specific clinical practice interventions (screening tests, immunizations, chemoprophylactic regimens, and counseling sessions) to be initiated by the attending physicians are listed and extensively explained.

This *Guide* is intended for primary care clinicians and the major primary care specialties: family practice, internal medicine, pediatrics, and obstetrics/gynecology. In addition, the *Guide* is intended for use by nurses, nurse practitioners, physicians' assistants, other allied health practitioners, and students. However, there should certainly be no inhibition by any physician, regardless of specialty, to incorporate clinical preventive services into standard operating procedures in the office setting and elsewhere. Obviously, any patient could be smoking, overweight, or driving a car.

Following a well-documented methodology, the 20 task force members, the 16 senior advisors, and the more than 325 expert reviewers undertook their investigations. Emphasis was given to services for asymptomatic persons that could be delivered in a clinical setting. That emphasis led to a focus on primary and secondary clinical preventive measures. Combining all that informational input, the task force agreed on a number of far-reaching conclusions possibly having an impact on the day-to-day office practice of physicians. Medical care encounters could be drastically changed by the following findings:

- A selective periodic health examination tailored to individual risks is a more appropriate clinical strategy than the annual physical, in which the same battery of tests is performed routinely on all patients.
- There is need for greater selectivity in the use of screening tests, with tests chosen on the basis of the unique risk profile of the individual patient.
- Addressing the personal health behaviors of patients through education and counseling is one of the most effective forms of prevention available to the clinician.
- Both clinicians and patients will continue to under-

go important role changes in the near future. Patients will assume greater responsibility for their own health. Clinicians will develop new skills in risk assessment, counseling, and patient education.

For each of the 60 potential interventions to prevent diseases and/or conditions, the recommendations consider age, gender, and risk factors. In a few short, succinct pages, each of the 60 target preventive interventions is reviwed and the *Guide* uniformly does the following:

1. States the recommendation.
2. Details the burden of suffering in terms of mortality and morbidity statistics, ages affected, and socioeconomics.
3. Describes the intervention's efficacy in detecting the condition or in reducing risk.
4. Evaluates the effectiveness of early detection or counseling in reducing morbidity or mortality.
5. Summarizes the recommendations of other groups relative to the specific intervention.
6. Discusses other relevant issues such as side effects or clinical approaches to the patient.
7. Suggests appropriate clinical interventions, including specification of high risk groups, dosages, and screening schedules (where supported by evidence).
8. Lists the primary literature used in the development of the specific recommended intervention.

Clinical preventive services are divided into nine screening groupings comprising 47 interventions, one counseling group of eight interventions, and one immunizations/chemoprophylaxis group of five interventions. These groupings are identified below, along with the diseases/conditions to be screened for in each category. In addition, a recommendation example is also given. Importantly, each recommendation is more fully clinically explained in each of the 60 interventions as described in item #7 above.

Clinical screening intervention services include the following generic groups, with the disease and/or condition to be screened for listed within each category:

Vascular Diseases: asymptomatic coronary artery disease; high blood cholesterol; hypertension; cerebrovascular diseases; and peripheral arterial disease.

Recommendation example: Clinicians should emphasize the primary prevention of coronary artery disease (CAD) by periodically screening for high blood pressure and high serum cholesterol and by routinely investigating behavioral risk factors for CAD such as tobacco use, dietary fat, and cholesterol intake. Secondary prevention of CAD (screening) by performing routine electrocardiography to screen asymptomatic persons is not recommended. It may be clinically prudent to perform screening electrocardiograms (ECGs) in certain high risk groups. Routine resting or exercise ECG screening before entering athletic programs is not recommended for asymptomatic children, adolescents, or young adults.

Neoplastic Diseases: breast cancer; colorectal cancer; cervical cancer; prostate cancer; lung cancer; skin cancer; testicular cancer; ovarian cancer; pancreatic cancer; and oral cancer.

Recommendation example: There is insufficient evidence to recommend for or against routine digital rectal examination as an effective screening test for prostate cancer in asymptomatic men. Transrectal ultrasound and serum tumor markers are not recommended for routine screening in asymptomatic men.

Metabolic Disorders: diabetes mellitus; thyroid disease; obesity; and phenylketonuria.

Recommendation example: An oral glucose tolerance test for gestational diabetes mellitus is recommended for all pregnant women between 24 and 28 weeks of gestation. Routine screening for diabetes in asymptomatic nonpregnant adult patients, using plasma glucose measurement or urinalysis, is not recommended for the general population, but it may be appropriate in selected high risk groups.

Infectious Diseases: hepatitis B; tuberculosis; syphilis; gonorrhea; human immunodeficiency virus; chlamydial infection; genital herpes simplex; asymptomatic bacteriuria, hematuria, and proteinuria.

Recommendation example: Screening for infection with human immunodeficiency virus (HIV) should be offered periodically to persons seeking treatment for sexually transmitted diseases, intravenous drug users, homosexual and bisexual men, and others at increased risk of infection. Testing should also be offered to pregnant women (or women contemplating pregnancy) who are at increased risk for HIV infection. Testing should not be performed in the absence of informed consent and adequate pretest and post-test counseling. Clinicians should be careful to use proper tests and qualified laboratories. Seropositive persons require adequate post-test counseling, and partners should be properly notified. Persons with negative test results also require counseling and repeat testing when appropriate.

Hematologic Disorders: anemia; hemoglobinopathies; and lead toxicity.

Recommendation example: All infants and pregnant women should be tested for anemia. Routine screening of other asymptomatic persons for anemia is not recommended in the absence of clinical indications.

Ophthalmologic and Otologic Disorders: diminished visual acuity; glaucoma; and hearing impairment.

Recommendation example: There is insufficient evidence to recommend routine performance of tonometry by primary care physicians as an effective screening test for glaucoma. It may be clinically prudent, however, to advise patients aged 65 and older to be tested periodically for glaucoma by an eye specialist.

Prenatal Disorders: intrauterine growth retardation; preeclampsia; rubella, Rh incompatibility; congenital birth defects; and fetal distress.

Recommendation example: Fetal heart rate should be measured by auscultation on all women in labor to detect signs of fetal distress. Electronic fetal monitoring should not be performed routinely on all women in labor. It should be reserved for pregnancies at increased risk for fetal distress.

Musculoskeletal Disorders: postmenopausal osteoporosis; and risk of low back injury.

Recommendation example: Routine radiologic screening to detect low bone mineral content is not recommended. Estrogen replacement therapy is discussed in chapter 59.

Mental Disorders and Substance Abuse: dementia; abnormal bereavement; depression; suicidal intent; violent injuries; and alcohol and other drug abuse.

Recomendation example: The performance of routine screening tests for depression in asymptomatic persons is not recommended. Clinicians should maintain an especially high index of suspicion for depressive symptoms in those persons at increased risk for depression.

Clinical Counseling Services: Counseling services for patients pay attention to the following specific behavioral pattern goals: prevention of tobacco use, exercise counseling, nutrition advice, prevention of motor vehicle injuries, prevention of household and environmental injuries, prevention of human immunodeficiency virus infection and other sexually transmitted diseases, prevention of unintended pregnancy, and the prevention of dental disease.

Recommendation example: Tobacco cessation counseling should be offered on a regular basis to all patients who smoke cigarettes, pipes, or cigars, and to those who use smokeless tobacco. The prescription of nicotine gum may be an appropriate adjunct for some patients. Adolescents and young adults who do not currently use tobacco products should be advised not to start.

Immunization and Chemoprophylaxis Prevention: Immunization and chemoprophylaxis preventive services in clinical practice include the following: childhood immunizations; adult immunizations; post-exposure prophylaxis; estrogen prophylaxis; and aspirin prophylaxis.

Recommendation example: Low-dose aspirin therapy should be considered for men aged 40 and over who are at significantly increased risk for myocardial infarction and who lack contraindications to the drug. Patients should understand the potential benefits and risks of aspirin therapy before beginning treatment.

In addition to the 60 clinical prevention interventions, the task force also developed seven age-specific periodic health examination regimens: birth to 18 months; age 2 to 6; ages 7 to 12; ages 13 to 18; ages 19 to 39; ages 40 to 64; and age 65 and older. Each age-specific chart lists the leading causes of death, screening services to provide, immunizations and chemoprophylaxis to administer, parent and/or patient counseling topics, and events that the physician should remain alert for as related effects. A time schedule is included indicating how often such service should be provided. Where appropriate, high risk groups are identified.

In addition, there is a chart dealing with pregnant women. Screening recommendations for the first prenatal visit spell out items to inquire about during history taking, advise blood pressure taking during the physical exam, and list appropriate laboratory/diagnostic procedures for the visit. In addition, tests for high risk groups are listed. In the first prenatal visit, the physician should counsel about nutrition, tobacco use, alcohol and other drug use, and the use of safety belts. High risk pregnant women should be counseled about amniocentesis and the risk of HIV infection. For follow-up prenatal visits, the guide provides a time schedule, and suggests screening for blood pressure and urine analysis for bacteriuria and special tests at gestational ages of 14–16 weeks, 24–28 weeks, and at 36 weeks. Counseling covers nutrition, safety belts, and rationales for future tests. High risk groups are counseled about tobacco use and alcohol and other drug use. At all visits by pregnant women, physicians are advised to be alert for signs of physical abuse.

Recommendations for or against preventive interventions were rated by the task force based on the strength and quality of the evidence in terms of study designs. In 13 tables, there are task force ratings of interventions: A (good evidence supporting); B (fair evidence supporting); C (poor evidence supporting); D (fair evidence excluding); and E (good evidence excluding). Similarly, quality ratings about interventions indicate scientific support: I (at least one properly randomized controlled trial); II-1 (well-controlled trials without randomization); II-2 (well-designed cohort or case control analytic studies); II-3 (multiple time series with or without the intervention); and III (opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees).

Within this system, strength of recommendation and quality of evidence ratings are awarded for preventive interventions relative to the following: breast cancer screening, colorectal cancer screening, diabetes mellitus screening, prevention of sexually transmitted diseases including HIV, urinary tract disorders, smoking cessation counseling, physical activity counseling, dietary fat counseling, automobile occupant protection counseling, fall prevention among older persons, prevention of unwanted adolescent pregnancy, preventive dentistry, and immunizations and immunoprophylaxis/chemoprophylaxis to prevent selected infections.

Practitioners may have trouble following all the suggested clinical preventive services recommended for integration into their daily office practice. This *Guide* resolves that problem with a four-page "Quick Reference Guide" which lists about 750 diseases/conditions or aspects of them in alphabetical order with the appropriate chapter indicated. This quick reference system could be of particular use to the nonprimary care specialists who want rapidly to isolate clinical preventive services suitable for their own practices.

The *Guide* indicates that "Although there are sound clinical reasons for emphasizing prevention in medicine, studies have shown that physicians often fail to provide recommended clinical preventive services." Why? A variety of reasons exist including insufficient time, a lack of reimbursement for preventive services, and unfamiliarity with procedures and services. However, even with those barriers resolved, clinicians still do not provide recommended preventive services. Again, why? It appears that clinicians are uncertain as to which services to offer to patients. Recommendations come from multiple sources, frequently the recommendations are different, and confusion and inaction results. Using the latest information, this *Guide to Clinical Preventive Services* applies the therapeutic prescription of a 294-page antidote to the disease and/or condition of "physician uncertainty."

Another area of uncertainty relates to the ability of physicians to counsel and to educate their patients. Medical school education may not have covered those skills in any detail. Based on its investigations, the task force comes up with ten key concepts to enhance the effectiveness of clinicians in getting their patients to change behaviors. Attention to the following principles should allow physicians to integrate the clinical preventive measures into their physician/patient encounters:

1. Develop a therapeutic alliance that is truly a provider-patient partnership to help patients control their own health choices.
2. Counsel all patients, even those who do not ask questions, in educational ways appropriate to their age, sex, socioeconomic status and interpersonal skills.
3. Ensure that patients understand the dynamic relationship between behavior and health, especially their own risk factors inherent in the effects of smoking, lack of exercise, poor nutrition or other lifestyle factors.
4. Work with patients to assess barriers to behavior change such as lack of skills, motivation, resources, and social support. Help your patients to find ways to overcome the barriers.
5. Gain commitment from patients to change by helping them to understand and to agree that their behaviors are signifi-

cantly related to health outcomes. This is critical if counseling and education is to be relevant.

6. Involve patients in selecting priority risk factors to change realizing that too much at once may overwhelm the patient. Take on one problem at a time. Remember that initial success breeds later success.
7. Use a combination of strategies combining individual counseling, group classes, audiovisual aids, written materials and community resources. However, do not substitute slick leaflets or videotapes for your own verbal communication with the patient. Numerous studies demonstrate that individual attention and feedback from the physician is the most useful strategy in changing patient knowledge and behavior.
8. Design a behavior modification plan that can include a written action program, time limited goals and objectives, instructional materials and a reward mechanism. Remember that patients may recall only 50 percent of what is said and that lifestyle recommendations are not as memorable as medication regimens. Also remember that patients feel rewarded when their physician compliments them on their achievements. Recall how school children competed for the teacher's gold star.
9. Monitor progress through follow-up appointments or telephone calls. Reinforce success through positive verbal feedback. If needed, refer patients to community agencies, self-help groups and seek support from the family and friends. Try to progressively transfer responsibility for self care to the patient.
10. Involve your office staff in creating a team approach to patient education. Receptionists can encourage patients to read materials in the reception room. All staff should communicate consistent positive health messages. Consider creating a patient education committee to generate program ideas and to promote staff and patient commitment.

These key concepts to induce behavior change also include a critical caveat for the attending physician, whom the patients see as a role model. A physician who smokes, is overweight, and does not exercise may find it uncomfortable to counsel patients in those areas. "Physicians who both practice and preach prudent health behavior should be better able to foster health within themselves and their patients."

Apparently, there is a great deal of material in the *Guide to Clinical Preventive Services* that instinctively interacts with this reviewer's almost 40 years of working experience in the health care system. Even our proverbs support the contention that prevention is the way to go in life and in medicine: "A stitch in time saves nine," or "An ounce of prevention is worth a pound of cure."

Hopefully, some aspect of this review will stimulate readers' intellectual curiosity to beg, borrow, or steal a copy of this book. Of course, there may be disagreements about some of the specific clinical preventive interventions. That is not the major issue. Those can be corrected easily and there will always be periodic updating of the material.

What is important is the initiative and attention to the broad scope of clinical preventive measures to be integrated into the everyday armamentarium of the practicing physician. If that attitudinal and behavioral change can be effected in practice and in the health care educational system, the US health care system will be moving in the right direction. A system emphasizing clinical preventive services will also contain costs, provide curing and caring, and meet the social and emotional needs of people.

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Breast cancer detection and counseling program

Breast cancer is a major cause of illness and death among women. In New York State alone, nearly 10,000 new cases are diagnosed each year and approximately 4,000 women die from breast cancer annually. A wealth of scientific evidence suggests that increased use of screening mammography would significantly reduce the number of breast cancer deaths. While white women experience higher incidence rates than minority women, black women suffer a similar number of mortalities, with a lower five year survival rate. This disparity is believed to be attributable to social and environmental factors that could be addressed through education, increased access to low cost screening programs, and improved referral and follow-up systems to ensure early diagnosis and prompt treatment.

The New York State Legislature appropriated funding to establish the Breast Cancer Detection and Counselling Program in 1987–88 to support projects in counties with the highest quartile of breast cancer incidence or mortality, or where the population is at least 10% black. Each project provides breast cancer detection and counselling programs including outreach, clinical, mammographic and education services, and referral and follow-up targeted to minority populations with inadequate access to preventive health care. Additionally, the projects helped establish and participated in a statewide service referral network among local cancer providers.

A Request for Application was distributed to hospitals, diagnostic and treatment centers, health maintenance organizations, local health departments, voluntary organizations, and cancer programs sponsored by the American Cancer Society, National Cancer Institute, and the American College of Surgeons in the 23 eligible New York State counties. Of the 46 applications submitted, eight were funded at approximately $100,000 each. The awards were granted to: Roswell Park Memorial Institute (Erie County), Onondaga County Health Department (Onondaga County), Albany Medical Center Hospital/Whitney Young Neighborhood Health Center (Albany County), Westchester County Health Department (Westchester County), St. Luke's-Roosevelt Hospital Center (New York County), Kings County Hospital/Cumberland Neighborhood Family Care Center (Kings County), Queens Hospital (Queens County), and Nassau County Health Department (Nassau County).

Funded projects are managed by the Bureau of Adult and Gerontological Health, evaluated by the Bureau of Cancer Epidemiology, and monitored for mammographic quality assurance by the Bureau of Radiation Protection. A Client Intake Form was developed for all the projects to use in order to ensure uniform reporting. Collected information on demographics, breast health status and service outcome data are then included in a statewide data base. The demographics are consistent with data collected by the New York State Cancer Registry to enable long term follow-up for breast cancer incidence and mortality. Reporting requirements include the completion of quarterly reports that summarize patient contacts, outreach and education activities. Despite a six-month delay from the time funding was awarded and contracts approved, nearly 2800 women were screened for breast cancer by June 30, 1989.

Each project is unique. For example, Nassau County Department of Health has breast screening services including mammography in two of its clinics, while Westchester County Health Department sponsors screening clinics in community-based sites, and refers clients to radiologists who provide free or low-cost mammography. Onondaga County Health Department screens patients through its own Women's Health Outreach clinics, but has negotiated contracts with four providers who perform mammograms for indigent patients.

Hospital based programs also differ. Roswell Park Memorial Institute has a van, the Health Express, that educates and provides clinical breast examination to target neighborhoods, and all radiologic services are provided through Roswell Park's cancer screening program. Queens Hospital's project is based in a multi-service community center, and both Kings County and Albany Medical Center have components at neighborhood health care centers, while the St. Luke's-Roosevelt project is entirely hospital based. Through the projects' different approaches, the Health Department can compare the effectiveness of different strategies to encourage breast screening in minority women.

In addition to the appropriation to project funding, monies were provided to implement a statewide mammography awareness campaign, *A Mammogram Can Be Your Breast Friend*. The campaign began in October 1989, in conjunction with National Breast Cancer Awareness Month, and emphasizes the life-saving benefit of mammography. It provides a toll-free number for additional information. Calls are answered by operators from Roswell Park Memorial Institute's Can Dial Program, who have been trained to give information and referrals.

For more information about the Breast Cancer Detection and Counseling Program, contact the Cancer Prevention Services Program, (518) 474-1222. For a *Mammogram Can Be Your Breast Friend* poster or brochure, contact the Bureau of Health Promotion, (518) 474-5370.

Vital statistics of New York State

The birth rate is rising in New York State for the first time since the early 1970s when post-World War II baby boomers started bearing children (Fig 1). In 1987, the birth rate in New York was 15.2 births per 1,000 population, the highest rate since the 15.4 per 1,000 recorded in 1971 and up 3.2% from 1986. In absolute numbers, there were 271,355 births statewide in 1987, the greatest number since the 1971 total of 238,562.

This trend appears in virtually all areas of the state, although it is most striking in New York City, where the birth rate was 16.7 per 1,000. Rates in New York City ranged from 20.0 in the Bronx, to 18.8 in Brooklyn, to 14.8 in Queens and Staten Island, to 13.9 in Manhattan. Overall, outside the city, the rate was 14.1 per 1,000. Counties in the Lake Ontario basin, from Orleans and Genesee east to Jefferson and Lewis, also contributed to the increased birth rate, virtually all with rates above the state average.

These birth rates are reported in the annual *Vital Statistics of New York State*, a compilation of data from birth, death, marriage and divorce records filed with state and local registrars.

The death toll from AIDS continued to climb in 1987. AIDS became the leading killer of New Yorkers between the ages of 20 and 44. AIDS accounted for 2,723 deaths in this age group, 18% of the total. Seventeen percent of white deaths in this age group resulted from AIDS, as well as 21% of black deaths. Cancer was the second leading cause of death in this group with 1,705 deaths, 11% of the total.

Overall, heart disease continued to be the leading cause of death among all New Yorkers in 1987. However, fewer people died from the disease than in previous years. In 1987, 66,490 New Yorkers died of heart disease which represents a death rate of 372 per 100,000 population, the lowest rate since 1939.

This annual report also provides a snapshot of New York State vital statistics. Figure 2 shows the daily averages for each recorded event. Daily, 743 births and 470 deaths occur, on the average, in New York State. Each day New York can expect 161 teenage pregnancies, 13 accidental deaths and ten deaths as a result of AIDS.

Other statistical highlights include:

- In 1987, the percentage of babies born weighing less than 2,500 grams (low birth weight) rose to 7.6%, the highest level since 1979. This was the second increase in two years and marks the end of the continuous downward trend observed between 1977 and 1985.

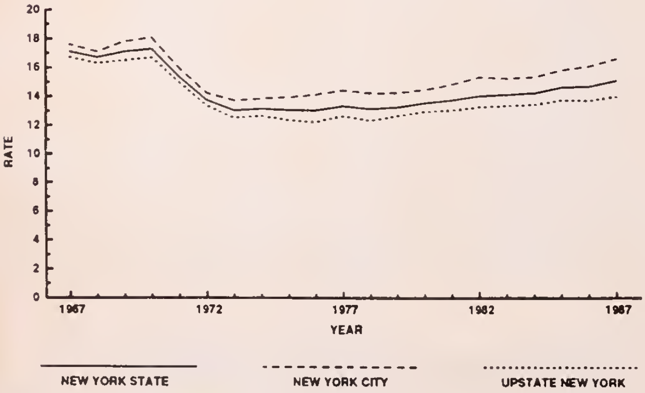

FIGURE 1. Live birth rate: New York State, New York City, Upstate New York, 1967–1987.

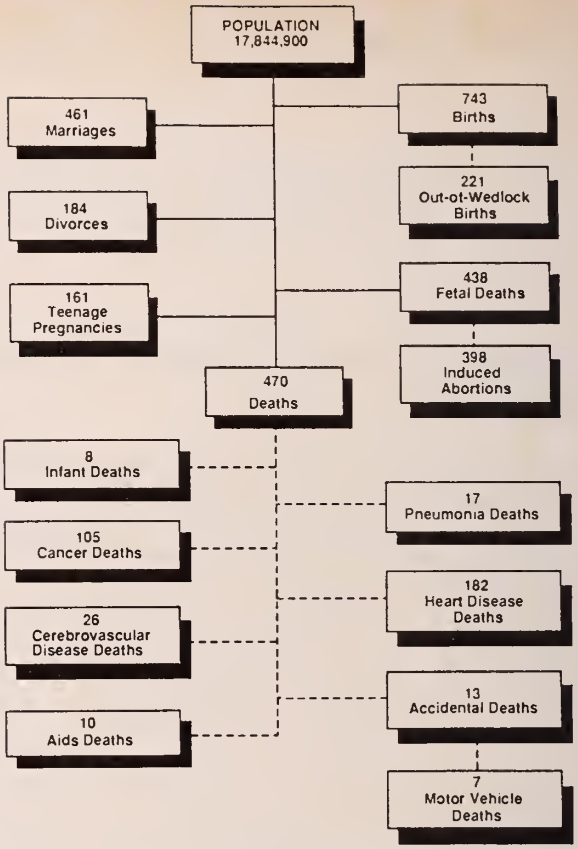

FIGURE 2. Daily average of vital statistics in New York State, 1987.

- After years of decline, the infant mortality rate has flattened out and even begun to increase again. The statewide infant mortality rate (babies per 1,000 births who die before one year) increased from 10.6 in 1986 to 10.7 in 1987. The infant mortality rate in areas outside of New York City actually declined from 9.2 to 9.0, while the rate in New York City increased from 12.4 to 12.7. The infant mortality rate for blacks continues to be about twice that for whites, 17.5 and 7.9, respectively. The national infant mortality rate in 1987 was 10.0.
- The mean age at death for men is decreasing, while the age for women is increasing. The median age of male decedents decreased from 70.6 in 1986 to 70.3 in 1987. The median age of female decedents went from 78.0 to 78.2.
- Whites live significantly longer than blacks. The median age of white decedents was 76.5 compared to 62.4 for blacks. This is true for both men (whites had a median death age of 72.9 compared with 57.4 years for black males) and women (white women had a median death age of 80.0, compared with 67.1 for blacks).
- The median age for first-time brides and grooms rose between 1986 and 1987. For brides, the median age rose from 24.1 to 24.4 years and for grooms from 25.8 to 26.2 years.

- The median length of marriages ending in divorce dropped from 7.3 to 7.2 years between 1986 and 1987, a trend noted since 1984.

Vital Statistics of New York State is available from Production Systems Management, Vital Statistics Section, (518) 474-3069.

Vibrio vulnificus in raw oysters

During 1988, 45 cases of *Vibrio vulnificus* illness were reported to health authorities in the United States. Twenty-five cases were related to known raw oyster consumption and 12 cases involved wound infections. A total of 17 deaths were reported and 11 of these (64%) were related to raw oyster consumption.

Vibrio vulnificus occurs naturally in the marine environment and is considered to be an opportunistic pathogen in individuals having compromised immune systems or medical disorders. *Vibrio vulnificus* illness generally causes a rapid onset of symptoms that include fever, chills, nausea, vomiting and abdominal pain, usually occurring approximately 16 hours after shellfish consumption in healthy individuals. As these symptoms are not unusual for many illnesses, careful questioning of the patient regarding recent shellfish consumption is important.

Cases of *Vibrio vulnificus* have not been reported in New York State, but it is possible that cases may be going undetected. In general, all individuals who consume raw or inadequately cooked oysters are susceptible to vibrio infection. Individuals with liver disease, compromised immune systems and/or those being treated for stomach acid disorders face a higher risk of serious complications from *Vibrio vulnificus*. These individuals should be advised not to eat raw shellfish. Since normal cooking procedures kill the organism, these people may continue to eat cooked shellfish. These high risk groups may develop primary septicemia in association with a *Vibrio vulnificus* infection with a mortality rate of greater than 50%.

Physicians should not wait for confirmation of diagnosis before administering an antibiotic because death of the compromised individual may occur within 48 to 72 hours of contracting the organism. Possibly compromised individuals would include persons with liver disease or other diseases with possible or definite hepatic involvement, such as alcoholism or malignancy; persons with therapeutically or naturally low gastric acid; a compromised immune system. Tetracycline is the antibiotic of choice. Rapid treatment using appropriate antibiotics significantly improves the chance of survival of affected patients. Cases of confirmed *Vibrio vulnificus* illness should be reported to the local health department.

Prevention of disabilities

Current estimates show that about 2.3 million people, 12% of the state's population, have severe or moderate limitations that would classify them as disabled. National estimates indicate that as many as 20–30% of the disabilities could be prevented with the application of current measures. Today's spiraling health costs, coupled with the financial and emotional toll of disabilities on the families of those affected, make it imperative that more efforts be directed toward prevention.

New York is one of nine states to receive funding from the US Centers for Disease Control to establish a statewide program of disability prevention. A Disability Prevention Program has been created within the Department's Office of Public Health (OPH). Categories of disabilities to be addressed include prevention of disabilities as a result of injuries, developmental disabilities, and secondary disabilities.

The program will focus on specific preventable disabilities identified by incidence/prevalence, functional impact and amenability to prevention. Preventive action plans will be developed through a process that will involve agency-wide resources and expertise, representatives of other state agencies and interested organizations and experts outside of the state government. A State Disability Prevention Council is being assembled by the Department of Health to advise on the development of a strategic plan for prevention and to mobilize public, private, and voluntary agencies and organizations to implement disability prevention projects.

The Disability Prevention Program will function within the Center for Community Health, Division of Epidemiology. The Program Director is Susan J. Standfast, MD, MPH. Many units within OPH will participate in this priority initiative through provision of technical support, access to data and assistance with the development of background papers. The visibility and impact of this program will be enhanced through relationships with other state and outside agencies and individuals.

Questions may be directed to Fran Stevens, Disability Prevention Program Coordinator, at (518) 474-2018. The Disability Prevention Program is located in Room 621, Corning Tower, Empire State Plaza, Albany, NY 12237-0608.

THE PEDIATRIC NEUROSURGICAL PATIENT: A COOPERATIVE APPROACH

Edited by L. P. Ivan, MD. 323 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1989. $49.50 (paperback)

The Pediatric Neurosurgical Patient: A Cooperative Approach is edited, and largely written, by L. P. Ivan, MD, former Chairman of Neurosurgery at the University of Ottawa School of Medicine, and H. Hugenholtz, MD, Associate Professor of Neurosurgery at the University of Ottawa School of Medicine. Nine other individuals in different specialty areas also contribute to the 18 chapters which span most of the important areas in pediatric neurosurgery.

Most chapters are succinctly and briefly structured as topic reviews, and include such important subjects as tumors of the central nervous system and vascular lesions of the nervous system. Other topics covered include hydrocephalus, nervous system infections, head trauma, craniosynostosis, and functional neurosurgery. Certain other important topics discussed in this book, but usually omitted from similar works, include special needs of the child, family and staff, pediatric clinical neuropsychology, and pediatric neurosurgical nursing. A number of potentially important subjects are not addressed, including AIDS and the pediatric age group, and bioethical considerations in the treatment of children with malignant and other diseases. Many useful and easy to comprehend tables, radiographs, and scans are included. An addendum section on magnetic resonance imaging scanning has been included, but is flawed by typographical errors, uncommon in the other sections of the book. Limited references are supplied at the conclusion of each chapter. While many of the references are dated, they do serve to direct the reader to other, more comprehensive works.

The book is quite readable and is written in a direct and factual manner, electing to avoid controversial areas which might mislead or confuse some readers. It covers a tremendous amount of useful information in a clear and concise way. The book is most heartily recommended to medical students and nursing students doing clinical rotations on pediatric neurology or neurosurgery services, or house officers in pediatrics, pediatric neurology, and neurosurgery. Pediatric critical care nurses and therapists should also find the book to be of particular interest. However, it simply does not contain the depth and breadth necessary to make it a valuable reference work for practicing neurosurgeons or senior house officers.

Although there are already a number of excellent standard textbooks of pediatric neurosurgery available, including D. D. Matson's *Neurosurgery of Infancy and Childhood* (1969), the section on pediatric neurosurgery of the American Association of Neurological Surgeons' *Surgery of the Developing Nervous System* (1982), and T. H. Milhorat's *Pediatric Neurosurgery* (1978), this book's readable style, light weight, soft cover, and ample illustrations give it a special role. It is a book which should not be filed away in a library for occasional use, but should be readily available for ward consultation, and handed on from student to student. Despite its relatively high cost, it is a good investment for those clinicians and ancillary staff members involved in the care of pediatric neurosurgical patients.

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PEDIATRIC PULMONARY DISEASE

Edited by J. Thomas Stocker, MD. 273 pp, illustrated. New York, Hemisphere Publishing Corporation, 1989. $45.00 (hardcover)

The pathology of neonatal and pediatric pulmonary diseases is given little attention in the modern medical school curriculum or in general textbooks of neonatology, pediatrics, and pathology. Many first-rate medical centers still lack pathologists with experience and interest in pediatric pathology, and clinicians themselves seem to be losing interest in learning from postmortem examinations. For example, death certificates list "prematurity" or "hyaline membrane disease" as the primary cause of death in premature infants despite the fact that dozens of other specific lung disorders, albeit rare, might be detected by autopsy. As a result, today's medical students are exposed to the most advanced concepts of molecular biology, but graduate with the most primitive notions of human anatomy and physiology. Moreover, they are eager to use imprecise diagnostic tools such as new imaging techniques, endoscopy, and lung lavage, and leave history-taking and physical examination to computers and nonphysicians. If these trends continue, modern medicine may become exemplified by a recent comment made by a pediatric resident who claimed that the physical examination of a patient should be deferred until the CT and MRI scans have indicated which part of the body should be examined!

Fortunately, remedial, if vicarious, education is possible for the individual interested in the pathology of pediatric pulmonary disease, and one of the best places to start is to read this slim volume. This seven-chapter multiauthored monograph is devoted exclusively to this topic, and each chapter assembles important information that is unavailable in any other single text.

The first two chapters are devoted to pulmonary embryology and to congenital lung malformations. In both chapters, lung development is described in classic terms put forth by Reid, which focus mainly on epithelial development. One wishes that there were more discussion here about the dynamic interactions between these cells and lung vascular, interstitial, and neuroendocrine maturation as well.

A comprehensive and well-written review of the pulmonary hypoplasia syndromes which challenges traditional tenets is provided in chapter two. We learn, for example, that up to 50% of infants with fatal pulmonary hypoplasia do not have a history of oligohydramnios, and that pulmonary hypoplasia and structural immaturity of the lung are not synonymous.

Three chapters are devoted to acquired neonatal problems and the longest of these is a chapter on hyaline membrane disease and its sequela, bronchopulmonary dysplasia (BPD). In this chapter, the editor offers an innovative and testable conceptual framework with which to view the pathology of BPD. In addition, there is new information about the cardiac and systemic vascular abnormalities seen in these infants.

Another chapter discusses how mechanical ventilation with positive pressure produces interstitial air dissection in premature infants with respiratory failure. The author concludes that, as in adults, alveolar rupture causes this problem. However, data from premature animal models indicate that it is the terminal bronchioles which rupture early and lead to this problem.

Other chapters deal with unusual pulmonary venous and lymphatic diseases, rare tumors of the lung and chest, and the pulmonary consequences of HIV infection. All of the chapters include useful tables and figures, and most contain many photomicrographs which usually appear on the same page as the appropriate text. The illustrations are in black and white, and it is sometimes difficult to see important details in the photographs of gross specimens. Finally, as with other pathology texts, there is little discussion about the clinical or physiological abnormalities that accompany these disorders. Overall, it is a fine review of selected topics in pediatric pulmonary pathology. It should prove useful to the pathologist and to those who practice and teach perinatology, neonatology, and pediatrics.

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BRONCHOPULMONARY DYSPLASIA

Edited by Eduardo Bancalari, MD, and J. Thomas Stocker, MD. 441 pp, illustrated. New York, Hemisphere Publishing Corp, 1988. $79.50 (hardcover)

Bronchopulmonary dysplasia (BPD) is a chronic obstructive pulmonary disease which occurs predominantly in infants of less than 29 weeks' gestation who need treatment for respiratory failure. Because of the increasing survival rates of these premature infants, and because of a high rate of prematurity in the United States, the disorder is now the most frequent chronic pulmonary disorder of infancy. Its pathogenesis and treatment include virtually every known mechanism of lung injury and repair. It is therefore appropriate that the planners of an annual symposium devoted to pediatric pulmonary medicine chose BPD as their inaugural topic in 1986. This multiauthored text is a summary of this first symposium, and because of the multifactorial nature of BPD, not one of the 27 chapters in this book can be said to be inappropriate. Indeed, although the book is entitled "Bronchopulmonary Dysplasia," this ambitious work surveys virtually the entire field of neonatal lung injury and lung development. For the most part, each chapter is written by individuals who continue to make substantial contributions to the field, and the chapters themselves are meticulously referenced and nicely illustrated.

When viewed from the perspective of early 1990, it is clear that the year 1986 was thought to be pivotal for the understanding of BPD. That year, there was new hope that advances in biochemistry, cell biology, and animal modelling would soon lead to a better understanding of the mechanisms of lung injury and repair. Chapters are therefore devoted to thoughtful reviews of the role of oxidant injury, lung barotrauma, specific nutrient deficiencies, inflammatory mediators, and acquired vascular disease. Although much has been learned, the complex interaction between these factors and the immature lung remains poorly understood.

This volume also captures the widespread enthusiasm for new therapies which would prevent or reduce this vexing disease. In 1986, preliminary clinical trials of surfactant replacement and high frequency ventilation suggested that the incidence and severity of BPD could be reduced. Unfortunately, subsequent clinical trials of these therapies have been unable to confirm these results. Indeed, it is likely that surfactant replacement therapy, if widely adopted, will increase the prevalence of BPD as a consequence of increased survival rates for very low birth weight infants.

Similarly, in 1986 there appeared preliminary data which seemed to dispel the prevailing notion that BPD was the inevitable consequence of neonatal survival and lung immaturity. Instead, some argued, differences in BPD incidence existed which might be explained by differences in the clinical care practices of individual nurseries. Although this provocative report prompted clinicians to reexamine their clinical care policies, more recent epidemiologic data have not yet supported this possibility, and the overwhelming predisposing factors to BPD remain low birth weight and lung immaturity.

A special strength of this volume is that it contains a wealth of pathologic studies of acute and chronic BPD which have been artfully assembled by Dr Stocker from material at the Armed Forces Institute of Pathology. These studies indicate that the key morphologic abnormality of BPD is focal squamous metaplasia of small conducting airways, but other abnormalities seen in BPD patients are similar to those seen in patients with congestive heart failure and with chronic asthma. These observations formed the basis for clinical trials of diuretic, bronchodilator, and corticosteroid therapy; the results of these early studies are presented in the text.

In short, despite the passage of time and its cost, this comprehensive work is a welcome and valuable addition to the literature of BPD and to pediatric pulmonary medicine in general.

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TOXICOLOGIC AND PHARMACOLOGIC PRINCIPLES IN PEDIATRICS

Edited by Sam Kacew, MS, PhD, and Simon Lock, MS, PhD. 314 pp, illustrated. New York, Hemisphere Publishing Corp, 1988. $59.50 (hardcover)

This is the first edition of a textbook published by a pharmacologist and toxicologist with the help of 14 contributors. The authors state in the preface that the text is written primarily for the benefit of pharmacologists, toxicologists, and other health scientists, although they state that clinicians, including obstetricians, neonatologists, and pediatricians, may find many areas of the book particularly helpful. This 300-page effort includes 12 chapters which discuss various aspects of pediatric and neonatal pharmacology and toxicology. Included are chapters on in utero drug exposure and its effects on reproductive function, pulmonary surfactant development and its modulation by pharmacologic influences, analgesic embryopathy, and the influence of maternal substance abuse on the developing fetus. There are also three chapters that discuss the environmental toxins lead, chlordecon, and hydrocarbons.

There are many outstanding sections in this textbook. Included among these are many well-outlined tables delineating factors affecting placental transfer of drugs, relationships between drugs and developmental stages, and effects of drugs on the fetus. These tables outline the authors' thoughts and make for a clear presentation. The chapter on analgesic embryopathy and chemically induced cataracts is extremely well written, and provides a balance of clinical and basic science aspects of the study of drug effects on the developing infant.

The textbook appears to have several minor shortcomings. As is often the case with textbooks written by multiple contributors, there is a tendency for an unevenness in the writing, with varying degrees of emphasis on clinical versus basic science and some redundancy in the use of certain concepts. While the tables are clear, one often wishes that the data in the tables were referenced to provide easy access to the original literature on the topic. It is also of note that many references provided in the text are relatively old, having been published in the 1970s. This is true despite the fact that a clear body of literature on these topics has been published more recently. This is particularly true for the chapter on maternal drug dependence. The chapter on lead intoxication only discusses the effect of lead on opiate receptors in the brain, with no discussion of the more important neurodevelopmental consequences of lead intoxication in the fetus and young infant. A discussion of chlordecon (Kepone™) is welcome in the chapter on pesticides. However, one is left wondering why no other pesticides, including 24-D and DDT, are discussed in the book. Finally, many aspects of pediatric toxicology which might be appropriate for a textbook such as this are unmentioned, for example, the influence of radiation hazards such as radon and the specific risks they potentially pose in the young child.

Despite these shortcomings, the textbook is very informative and fills a void in pediatric toxicology. As the authors note, this is a textbook which appears to be designed specifically for nonphysician pharmacologists and toxicologists. Clinical toxicologists may also derive some benefit from this book as an addition to their library.

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GYNECOLOGIC SURGERY

Edited by Luis E. Sanz, MD. 450 pp, illustrated. Oradell, NJ, Medical Economics Books, 1988. $57.95 (hardcover)

The book is intended to provide authoritative and up-to-date coverage of innovative surgical procedures, techniques, and instrumentation in gynecology. The intent has been well accomplished. The editor has brought together the works of distinguished gynecologic surgeons in the field, and the textbook provides a comprehensive, concise, and well-organized presentation of the various operative procedures used in gynecology. This book represents an excellent surgical source for those interested in gynecologic procedures.

The text is organized into six sections which are divided into 44 chapters to allow for easy review of each particular topic. Section I, entitled "General Surgery and Anatomy," includes nine chapters dealing with anatomy of the retroperitoneum, retroperitoneal approaches to pelvic surgery, wound management, application of surgical staples and drains, appendectomy, repair of incidental abdominal wall hernias, intrauterine transfusion, and evaluation of breast lumps.

Section II is entitled "Vaginal Surgery." This section has ten chapters dealing with preventing and treating enterocele, repair of total vaginal prolapse, sacrospinal support after total vaginal prolapse, the Martius graft technique for rectovaginal fistulas (RVF), correcting failed RVFs, indications and techniques for vaginal hysterectomy, vaginectomy, cerclage for incompetent cervix, hysteroscopy, and surgical management of anal incontinence.

Section III, entitled "Gynecologic Urologic Surgery," includes seven chapters on the evolution of surgery for stress urinary incontinence (SUI), procedures for correcting urinary incontinence, treating vesicovaginal fistulas (VVF), the Latzko technique for repairing VVFs, and correcting urethral diverticula.

Section IV is entitled "Infertility Surgery." This section has chapters on implantation to restore tubal patency, laparoscopic surgery for tubal pregnancy, mastering reconstructive tubal microsurgery, managing ectopic pregnancy in young patients, review of techniques for tubal sterilization, metroplasty, and myomectomy.

Section V is entitled "Laser Surgery" and has four chapters dealing with intra-abdominal laser applications, hysteroscopic laser surgery, laser laparoscopy, and endometrial ablation with the Nd:YAG laser.

Section VI is entitled "Managing Complications of Gynecologic Surgery." This section has seven chapters dealing with postpartum hemorrhage and techniques for hypogastric artery ligation, uterine artery ligation for hemorrhage, recognizing injuries to the urinary tract, repairing the bladder during gynecologic surgery, management of ureteral laceration, repair of bowel injuries, and geriatric gynecologic surgery.

Overall the writing style is simple, straightforward, and uniform throughout. The chapters are very well organized and referenced. In general, the illustrations are simple, clear, and to the point (germane). The text is clearly written and the print is easy to read with double columns on each page and graphs and tables that complement the written material. The chapters on the anatomy of the retroperitoneum, wound management, preventing and treating enterocele, and sacrospinal support after total vaginal prolapse are excellent, with ample illustrations to enhance coverage and current references. The chapter on laser surgery puts this current modality in proper perspective with potential uses, advantages, and disadvantages well outlined. The rest of the chapters are adequate in terms of content, illustrations, and bibliography.

The chapters on intrauterine transfusion, cerclage for incompetent cervix, postpartum hemorrhage, techniques for hypogastric artery ligation, and uterine artery ligation belong in a textbook on operative obstetrics rather than in a textbook on gynecologic surgery. The chapter on geriatric gynecologic surgery is very relevant but does not belong within the section, "Managing Complications of Gynecologic Surgery."

Despite these partisan complaints, this is a well-conceived, well-executed textbook and ranks among the best in the field. It is readable and thorough and has a timely bibliography. It will be invaluable as a textbook and reference work. If one's interest lies in gynecologic surgery, one would do well to find room on the shelf for this commendable and valuable work.

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Frank John Amodio, MD, Syracuse. Died October 13, 1989; age 75. Dr Amodio was a 1942 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Fellow of the American College of Surgeons. His memberships included the New York State Surgical Society, the New York Academy of Medicine, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Lowyd Ballantyne, Jr, MD, McLean, Virginia. Died October 4, 1989; age 66. Dr Ballantyne was a 1949 graduate of Columbia University College of Physicians and Surgeons. He was a Diplomate of the American Board of Plastic Surgery and a Fellow of the American College of Surgeons. He was a member of the American Society of Plastic and Reconstructive Surgery, the Pan American Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Irving Rosen Block, MD, Patchogue. Died October 27, 1989; age 74. Dr Block was a 1940 graduate of Howard University College of Medicine, Washington, DC. He was a Diplomate of the American Board of Surgery and of the American Board of Colon and Rectal Surgery, and a Fellow of the American College of Surgeons. He was a member of the American Society of Colon and Rectal Surgery, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Lester Blum, MD, New York. Died August 30, 1989; age 83. Dr Blum was a 1930 graduate of Columbia University College of Physicians and Surgeons. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the New York State Surgical Society, the New York Society for Cardiovascular Surgery, the American Association for the Surgery of Trauma, the New York County Medical Society, and the Medical Society of the State of New York.

Dominic Anthony Bovino, MD, Brooklyn. Died September 15, 1989; age 80. Dr Bovino was a 1936 graduate of the New York Medical College. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Winfield L. Butsch, MD, Buffalo. Died October 24, 1989; age 83. Dr Butsch was a 1930 graduate of the State University of New York at Buffalo School of Medicine. He was a Diplomate of the American Board of Thoracic Surgery and the American Board of Surgery and a Fellow of the American College of Surgeons. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Murray Cohen, MD, Manhasset. Died October 27, 1989; age 56. Dr Cohen was a 1959 graduate of the New York University School of Medicine. He was a Diplomate of the American Board of Internal Medicine and a member of the Nassau County Medical Society and the Medical Society of the State of New York.

John E. Cunningham, MD, Glens Falls. Died May 4, 1989; age 86. Dr Cunningham was a 1930 graduate of the Albany Medical College of Union University. He was a member of the New York Academy of Medicine, the Medical Society of the County of Warren, and the Medical Society of the State of New York.

Michael DiGiacoma, MD, Bronxville. Died October 28, 1989; age 75. Dr DiGiacoma was a 1940 graduate of the New York Medical College. He was a Fellow of the American Academy of Family Physicians. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Peter Dineen, MD, New York. Died October 17, 1989; age 64. Dr Dineen was a 1947 graduate of Cornell University Medical College. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. His memberships included the New York State Surgical Society, the American Geriatrics Society, the American Surgical Association, the American Thoracic Society, the New York County Medical Society, and the Medical Society of the State of New York.

Martin Dollin, MD, Babylon. Died November 6, 1989; age 78. Dr Dollin was a 1937 graduate of Rush Medical College, Chicago. He was a Diplomate of the American Board of Psychiatry and Neurology and a Fellow of the American Psychiatric Association. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Bernard Drexler, MD, Marcellus. Died November 4, 1989; age 78. Dr Drexler was a 1935 graduate of the State University of New York at Buffalo School of Medicine. He was a member of the New York Academy of Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Myron John Dybich, MD, Schenectady. Died October 16, 1989; age 87. Dr Dybich was a 1930 graduate of the State University of New York at Buffalo School of Medicine. He was a member of the Medical Society of the County of Schenectady and the Medical Society of the State of New York.

Walter Davis Edwards, MD, Rochester. Died September 15, 1989; age 99. Dr Edwards was a 1915 graduate of Harvard Medical School. He was a member of the New York Academy of Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Ruth Selke Eissler, MD, New York. Died October 7, 1989; age 83. Dr Eissler was a 1932 graduate of the Medizinische Fakultaet der Universitaet Heidelberg, Heidelberg, Baden-Wurtemberg, Germany. She was a Diplomate of the American Board of Psychiatry and Neurology. She was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Edward Henry Eppers, MD, Buffalo. Died May 1, 1989; age 73. Dr Eppers was a 1940 graduate of the State University of New York at Buffalo School of Medicine. He was a member of the New York Academy of Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Aldo Rocco Faga, MD, Scarsdale. Died May 24, 1989; age 58. Dr Faga was a 1958 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Roma, Rome, Italy. He was a Diplomate of the American Board of Allergy and Immunology and a Fellow of the American College of Allergists. He was a member of the American Academy of Allergy, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Abraham Isidor Feldman, MD, Kingston. Died November 11, 1989; age 65. Dr Feldman was a 1948 graduate of the Chicago Medical School. He was a Fellow of the American College of Obstetricians and Gynecologists. He was a member of the Medical Society of the County of Ulster and the Medical Society of the State of New York.

Martin Friedland, MD, Amherst. Died October 21, 1989; age 88. Dr Friedland was a 1926 graduate of the State University of New York at Buffalo School of Medicine. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Rudolph C. Giannattasio, MD, Massapequa Park. Died October 8, 1989; age 69. Dr Giannattasio was a 1949 graduate of Loyola University Stritch School of Medicine, Maywood, Illinois. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Ramon Garcia Gochuico, MD, Brooklyn. Died October 25, 1989; age 52. Dr Gochuico was a 1962 graduate of the Faculty of Medicine and Surgery University of Santo Tomas, Manila, Philippines. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Lester B. Goldstein, MD, Whitestone. Died February 6, 1989; age 80. Dr Goldstein was a 1936 graduate of the Faculte de Medecine de l'Universite de Lausanne, Lausanne, Switzerland. He was a member of the American Academy of Family Physicians, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Jack Jason Goliger, MD, Lawrenceville, New Jersey. Died November 5, 1989; age 81. Dr Goliger was a 1934 graduate of the University of St Andrews School of Medicine, Dundee, Scotland. He was a Fellow of the American College of Chest Physicians. He was a member of the New York Cardiological Society, the American Thoracic Society, the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Phyllis Greenacre, MD, Garrison. Died October 24, 1989; age 95. Dr Garrison was a 1916 graduate of Rush Medical College, Chicago, Illinois. She was a Diplomate of the American Board of Psychiatry and Neurology. She was a member of the New York Academy of Medicine, the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Herman Jack Grobstein, MD, Kent, England. Died October 15, 1989; age 78. Dr Grobstein was a 1936 graduate of the Faculte de Medecine de l'Universite de Lausanne, Lausanne, Switzerland. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Stephen L. Gumport, MD, New York. Died November 12, 1989; age 76. Dr Gumport was a 1938 graduate of Cornell University Medical College. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. He was a member of the New York Academy of Medicine, the New York State Surgical Society, the American Association for Cancer Research, the Society of Surgical Oncology, the New York County Medical Society, and the Medical Society of the State of New York.

Arthur Harold Hartley, MD, Cranbury, New Jersey. Died September 11, 1989; age 79. Dr Hartley was a 1933 graduate of Columbia University College of Physicians and Surgeons. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Leslie L. Hayos, MD, Valley Stream. Died November 8, 1989; age 79. Dr Hayos was a 1934 graduate of the Orvosi Fakultas Tudomanyegyetem, Budapest, Hungary. He was a Fellow of the American College of Cardiology. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Edward E. Helwith, MD, Rockaway Point. Died October 20, 1989; age 76. Dr Helwith was a 1952 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Thomas P. Jacobs, MD, New Rochelle. Died August 24, 1989; age 74. Dr Jacobs was a 1940 graduate of Rush Medical College, Chicago. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the American Society of Internal Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Arnold Jacobsen, MD, Teaneck, New Jersey. Died October 15, 1989; age 91. Dr Jacobsen was a 1922 graduate of the Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Germany. He was a Qualified Fellow of the International College of Surgeons. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

J. Harry Katz, MD, Naples, Florida. Died October 11, 1989; age 77. Dr Katz was a 1936 graduate of Hahnemann Medical College of Philadelphia. He was a Diplomate of the American Board of Dermatology, Inc. His memberships included the New York Academy of Medicine, the American Academy of Dermatology, the Society for Investigative Dermatology, the New York County Medical Society, and the Medical Society of the State of New York.

Andrew D. Kerr, MD, New York. Died November 23, 1989; age 67. Dr Kerr was a 1945 graduate of New York University School of Medicine. He was a Diplomate of the American Board of Anesthesiology and a Fellow of the American College of Anesthesiologists. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the New York County Medical Society, and the Medical Society of the State of New York.

Kathryn Kirby, MD, South Nyack. Died May 23, 1989; age 66. Dr Kirby was a 1947 graduate of the University of Iowa College of Medicine. She was a Diplomate of the American Board of Psychiatry and Neurology. Her memberships included the American Psychiatric Association, the American Medical Women's Association, the New York County Medical Society, and the Medical Society of the State of New York.

William W. Kleinhandler, MD, Schenectady. Died September 6, 1989; age 62. Dr Kleinhandler was a 1954 graduate of Tufts University School of Medicine, Boston, Massachusetts. He was a Diplomate of the American Board of Ophthalmology. He was a member of the American Academy of Ophthalmology and Otolaryngology, the Medical Society of the County of Schenectady, and the Medical Society of the State of New York.

Marcus D. Kogel, MD, Croton-on-Hudson. Died November 28, 1989; age 86. Dr Kogel was a 1927 graduate of New York Medical College. He was a Diplomate of the American Board of Preventive Medicine, Inc, and a Fellow of the American College of Preventive Medicine. His memberships included the New York Academy of Medicine, the American Public Health Association, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Howard J. Kopins, MD, New Rochelle. Died September 30, 1989; age 80. Dr Kopins was a 1933 graduate of Georgetown University College of Medicine. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

David John Kreis, Jr, MD, Stony Brook. Died October 8, 1989; age 38. Dr Kreis was a 1977 graduate of Yale University School of Medicine. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Beatrice Kresky, MD, Jamaica. Died October 12, 1989; age 74. Dr Kresky was a 1945 graduate of the New York University School of Medicine. Her memberships included the New York Academy of Medicine, the American Public Health Association, the American Academy of Family Physicians, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

J. Walter Landsberg, MD, Locust Valley. Died October 4, 1989; age 82. Dr Landsberg was a 1943 graduate of Columbia University College of Physicians and Surgeons. He was a member of the New York Academy of Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Jessie L. Gillespie Merin, MD, Rochester. Died October 28, 1989; age 89. Dr Merin was a 1927 graduate of Cornell University Medical College. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Philip Vincent Messina, MD, Copiague. Died November 23, 1989; age 83. Dr Messina was a 1932 graduate of Indiana University School of Medicine. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Virginia Connolly Mitty, MD, New York. Died November 9, 1989; age 70. Dr Mitty was a 1946 graduate of New York University School of Medicine. She was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Walter Charles Mott, MD, Ipswich, Massachusetts. Died December 14, 1988; age 92. Dr Mott was a 1925 graduate of Albany Medical College of Union University. He was a Diplomate of the American Board of Ophthalmology. He was a member of the Medical Society of the County of Albany and the Medical Society of the State of New York.

Paul Fordham Nugent, MD, Sarasota, Florida. Died October 21, 1989; age 92. Dr Nugent was a 1922 graduate of Cornell University Medical College. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

James J. O'Brien, MD, Binghamton. Died September 23, 1989; age 67. Dr O'Brien was a 1950 graduate of Georgetown University School of Medicine, Washington, DC. His memberships included the New York State Society of Internal Medicine, the New York Academy of Medicine, the American Society of Internal Medicine, the Broome County Medical Society, and the Medical Society of the State of New York.

Charles Kirby Planer, MD, Syracuse. Died November 18, 1989; age 71. Dr Planer was a 1945 graduate of Hahnemann Medical College of Philadelphia. He was a member of the Onondaga County Medical Society and the Medical Society of the State of New York.

Rudolf Radna, MD, Bronx. Died September 28, 1989; age 84. Dr Radna was a 1929 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Germany. His memberships included the New York Cardiological Society, the New York Society of Tropical Medicine, the Bronx County Medical Society, and the Medical Society of the State of New York.

William Henry Raymond, MD, Gloversville. Died October 7, 1989; age 84. Dr Raymond was a 1934 graduate of the Albany Medical College of Union University. He was a member of the American Society of Abdominal Surgeons, the Medical Society of the County of Fulton, and the Medical Society of the State of New York.

Howard A. Rusk, MD, New York. Died November 5, 1989; age 88. Dr Rusk was a 1925 graduate of the University of Pennsylvania School of Medicine. He was a Diplomate of the American Board of Physical Medicine and Rehabilitation and the American Board of Internal Medicine and a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the American Academy of Physical Medicine and Rehabilitation, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Sydney Samet, MD, Rochelle Park, New Jersey. Died September 11, 1989; age 86. Dr Samet was a 1937 graduate of the Universitaet Leipzig Medizinische Fakultaet, Saxony, Germany. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Rosario P. San Filippo, MD, Brooklyn. Died October 27, 1989; age 90. Dr San Filippo was a 1923 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Surgeons. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Abraham N. Saperstein, MD, Great Neck. Died November 20, 1989; age 89. Dr Saperstein was a 1930 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Samson A. Seley, MD, North Miami Beach, Florida. Died September 8, 1989; age 92. Dr Seley was a 1921 graduate of the Cornell University Medical College. He was a Fellow of the American College of Gastroenterology. He was a member of the International Academy of Proctology, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Shirley R. Snow, MD, Rochester. Died October 4, 1989; age 92. Dr Snow was a 1923 graduate of Columbia University College of Physicians and Surgeons. She was a Diplomate of the American Board of Obstetrics and Gynecology and a Fellow of the American College of Surgeons. She was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Harry Spiegelman, MD, Williamsville. Died August 24, 1989; age 84. Dr Spiegelman was a 1928 graduate of the State University of New York at Buffalo School of Medicine. He was a member of the New York Academy of Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Harry Strongin, MD, New York. Died October 15, 1989; age 80. Dr Strongin was a 1934 graduate of Cornell University Medical College. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Max Thaler, MD, Parishville. Died September 9, 1989; age 80. Dr Thaler was a 1935 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a Diplomate of the American Board of Family Practice and a Fellow of the American Academy of Family Physicians. He was a member of the Medical Society of the County of St Lawrence and the Medical Society of the State of New York.

William George Toll, MD, San Diego, California. Died November 3, 1989; age 79. Dr Toll was a 1935 graduate of the Jefferson Medical College of Thomas Jefferson University, Philadelphia. He was a Diplomate of the American Board of Ophthalmology. His memberships included the New York Society for Clinical Ophthalmology, the American Academy of Ophthalmology and Otolaryngology, the Pan American Association of Ophthalmology, the New York County Medical Society, and the Medical Society of the State of New York.

Sylvia Gould Traube, MD, New York. Died September 26, 1989; age 80. Dr Traube was a 1950 graduate of Boston University School of Medicine. She was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Robert Tuby, MD, Great Neck. Died September 30, 1989; age 82. Dr Tuby was a 1930 graduate of the New York University School of Medicine. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

David Wainapel, MD, Ellenville. Died December 23, 1988; age 81. Dr Wainapel was a 1931 graduate of the Akademia Medyczna Wydzial Lekarski, Warszawa, Poland. He was a member of the Medical Society of the County of Ulster and the Medical Society of the State of New York.

Sue B. Wallis, MD, Pittsburgh, Pennsylvania. Died October 29, 1989; age 79. Dr Wallis was a 1935 graduate of the Medizinische Fakultaet der Universitaet Bern, Bern, Switzerland. She was a Fellow of the American College of Obstetricians and Gynecologists. She was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

Schedule of Sessions 95
Officers, Councilors, and Trustees 97
Members of the House 100
Reference Committees 104
Nominating Committee 105
Report of the House Committee on Bylaws 106

HOUSE OF DELEGATES

March 29 through April 1, 1990
New York Hilton

SCHEDULE OF SESSIONS

Thursday, March 29, 1990
Opening Session .... 1:00 p.m.

Friday, March 30, 1990
Reference Committee Hearings ... 8:30 a.m.–5:00 p.m.

Saturday, March 31, 1990
Second Session .... 9:00 a.m.
Third Session .... 1:30 p.m.

Sunday, April 1, 1990
Closing Session .... 9:00 a.m.

A Guide to the 1990 Annual Meeting

Policy-making in the House of Delegates

This section of the New York State Journal of Medicine is a call to the officers, delegates, and members of the Medical Society of the State of New York to attend the 184th Annual Meeting of the Society and the meeting of its House of Delegates; 292 delegates representing 61 county medical societies, 9 district branches, specialty societies, the hospital medical staff section, 12 medical schools, the resident and medical student membership will meet at the Hilton Hotel in New York City on March 29 to April 1, 1990.

The officers, members of the Council and the Board of Trustees, past presidents of the MSSNY, the Commissioner of the New York State Department of Health, the Past President of the American Medical Association from New York, Gerald D. Dorman, M.D., and Past Executive Vice-President of the Medical Society of the State of New York, George J. Lawrence, Jr., M.D., and Past Deputy Executive Vice-President of the Medical Society of the State of New York, Robert J. O'Connor, M.D., are also official representatives to the House.

Mandated by the Bylaws of the Society, this section lists the names of elected representatives, delegates who are serving on reference committees, and selections of the Nominating Committee for the election of Officers, Councilors, Trustees and AMA Delegates. Also included are the amendments to the Bylaws which will be considered by the House of Delegates.

Since the House of Delegates is the legislative body of the Society, charged with the general management of the State Society and its affairs, members of the Society are urged to use this information to consult with and advise their representatives. Resolutions introduced by the representatives bring members' concerns to the attention of the entire Society. Representatives not only have the responsibility to present the views of their constituents, but also to inform them of the actions taken by this governing body. The House of Delegates also formulates policy for the Society. No officer, councilor, board, commission, committee, or employee may initiate any policy unless such policy has been expressly approved by the House of Delegates, or by the Council of the Society.

If one were to choose the most important work of the House of Delegates, it would have to be that of the reference committees. Any member of the Medical Society of the State of New York has the right to appear before any reference committee and participate in its deliberations. It is also in the reference committees that people outside of the State Society are asked to give their views. Nonmember physicians, guests or interested observers who wish to express their opinions may, with permission, come before the reference committee and speak to the business of the committee. I strongly urge members to use this forum to express their views.

Seymour R. Stall, M.D., Speaker
House of Delegates

John H. Carter, M.D., Vice-Speaker
House of Delegates

MEDICAL SOCIETY OF THE STATE OF NEW YORK

ORGANIZED 1807

Officers 1989–1990

David M. Benford, MD

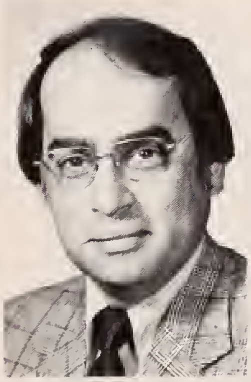
New York
President

Robert A. Mayers, MD

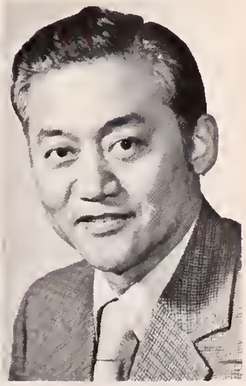
Westchester
President-Elect

Charles D. Sherman, Jr., MD

Monroe
Past-President


Charles N. Aswad, MD

Broome
Vice-President


George Lim, MD

Oneida
Secretary

Joyce M. McChesney, MD

Monroe
Assistant Secretary

Morton Kurtz, MD

Queens
Treasurer

James H. Cosgriff, Jr., MD

Erie
Assistant Treasurer

Seymour R. Stall, MD

Dutchess
Speaker

John H. Carter, MD

Albany
Vice-Speaker

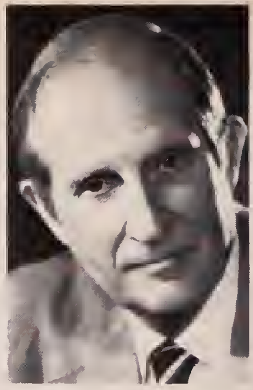
Board of Trustees 1989–1990

Bernard J. Pisani, MD

New York
Chairman

Daniel F. O'Keeffe, MD

Warren


Allison B. Landolt, MD

Westchester

John A. Finkbeiner, MD

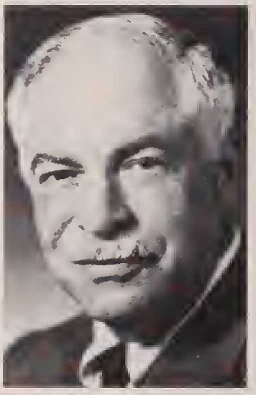
New York

James M. Flanagan, MD

Wayne

Samuel M. Gelfand, MD

Nassau


M. Theodore Tanenhaus, MD

Kings

Councilors 1989–1990

Duane M. Cady, MD

Onondaga

William A. Dolan, MD

Monroe

Iraj Iraj, MD

Kings

Stuart I. Orsher, MD

New York

Franklin A. DePeters, MD

Warren

Sidney Mishkin, MD

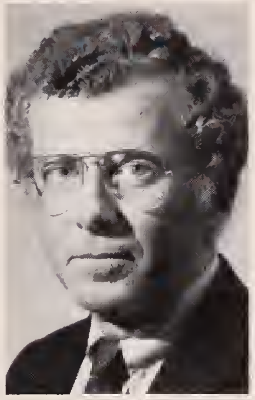
Nassau

Jeffrey A. Ribner, MD

Broome

Anthony P. Santomauro, MD

Erie



Robert E. Fear, MD

Suffolk

Stanley L. Grossman, MD

Orange

Thomas D. Pemrick, MD

Rensselaer

Ralph E. Schlossman, MD

Queens

John F. Piper, MD

Onondaga

Tanya Lumpkins

New York

1990 HOUSE OF DELEGATES MEMBERS

The following is a list of members of the 1990 House of Delegates of the Medical Society of the State of New York.

Officers 1989–1990
President—David M. Benford, New York
President-Elect—Robert A. Mayers, Westchester
Vice-President—Charles N. Aswad, Broome
Secretary—George Lim, Oneida
Assistant Secretary—Joyce M. McChesney, Monroe
Treasurer—Morton Kurtz, Queens
Assistant Treasurer—James H. Cosgriff, Erie
Speaker—Seymour R. Stall, Dutchess
Vice-Speaker—John H. Carter, Albany

Councilors
Term Expires 1990
Duane M. Cady, Onondaga
William A. Dolan, Monroe
Iraj Iraj, Kings
Stuart I. Orsher, New York

Term Expires 1991
Franklin A. DePeters, Warren
Sidney Mishkin, Nassau
Jeffrey A. Ribner, Broome
Anthony P. Santomauro, Erie

Term Expires 1992
Robert E. Fear, Suffolk
Stanley L. Grossman, Orange
Thomas D. Pemrick, Rensselaer
Ralph E. Schlossman, Queens

Student Councilor
Term Expires 1990
Tanya Lumpkins, New York

Resident Councilor
Term Expires 1990
John F. Piper, Onondaga

Trustees
Bernard J. Pisani, New York, *Chairman*
M. Theodore Tanenhaus, Kings
Daniel F. O'Keeffe, Warren
Allison B. Landolt, Westchester
John A. Finkbeiner, New York
James M. Flanagan, Wayne
Samuel M. Gelfand, Nassau

Past Presidents
1960–1961—Norman S. Moore, Tompkins
1966–1967—James M. Blake, Schenectady
1970–1971—Walter Scott Walls, Erie
1971–1972—George Himler, New York
1972–1973—Edward Siegel, Clinton
1974–1975—Lynn R. Callin, Monroe
1975–1976—Ralph S. Emerson, Nassau
1976–1977—George L. Collins, Jr., Erie
1977–1978—Carl Goldmark, Jr., New York
1978–1979—George T. C. Way, Dutchess
1980–1981—Ralph M. Schwartz, Kings
1982–1983—Richard D. Eberle, Onondaga
1983–1984—Bernard J. Pisani, New York
1984–1985—Allison B. Landolt, Westchester
1985–1986—Daniel F. O'Keeffe, Warren
1986–1987—John A. Finkbeiner, New York
1987–1988—Samuel M. Gelfand, Nassau
1988–1989—Charles D. Sherman, Monroe

Past Executive Vice-President
George J. Lawrence, Jr., Queens

Past Deputy Executive Vice-President
Robert J. O'Connor, Richmond

Past President of the American Medical Association
Gerald D. Dorman, New York

Speaker of the American Medical Association
John L. Clowe, Schenectady

Commissioner, New York State Department of Health
District Delegates
(*Elected Delegates of District Branches*)
First—Roger D. Billig, Richmond
Second—A. Philip Fontanetta, Nassau
Third—Albert M. Ellman, Albany
Fourth—Michael J. Lynch, Washington
Fifth—Frederic A. Stone, Jefferson
Sixth—Anton A. Vreede, Broome
Seventh—Warren Glaser, Monroe
Eighth—Victorino Anllo, Erie
Ninth—John A. Ramsdell, Westchester

Specialty Society Delegates
New York State Society of Allergy and Immunology
New York State Society of Anesthesiology—Edward C. Sinnott, Nassau
New York State Society of Dermatology—David Sibulkin, Nassau
New York Chapter American College of Emergency Physicians—Jeffrey L. Margulies, Suffolk
New York State Academy of Family Physicians—James F. Wright, Warren

New York State Society of Internal Medicine—Robert M. Kohn, Erie
New York State Neurosurgical Society, Inc.—Kalmon D. Post, New York
American College of Obstetricians and Gynecologists—District II—Leonard Weiss, Orange
New York Occupational Medical Association, Inc.—
New York State Ophthalmological Society—Frank J. Piper, Onondaga
New York State Society of Orthopaedic Surgeons, Inc.—Leo Arthur Green, Queens
New York State Society of Otolaryngology—Head and Neck Surgery—Lyon M. Greenberg, Albany
New York State Society of Pathologists—Edward Zaino, Nassau
American Academy of Pediatrics—District II—Marvin Blumberg, Queens
New York Society of Physical Medicine and Rehabilitation, Inc.—David Ernstoff, Queens
New York Regional Society of Plastic and Reconstructive Surgery—
New York State Psychiatric Association—George L. Ginsberg, New York
The New York State Radiological Society, Inc. (A Chapter of the American College of Radiology)—Jerome Zwanger, Nassau
New York State Society of Surgeons, Inc.—Theodore C. Max, Oneida
New York State Urological Society, Inc.—Marcus L. Shoobe, Albany

Delegates from Component Medical Societies

Albany (*5*)
John W. Abbuhl, Albany
James H. Puleo, Albany
Nathan P. Reed, Albany
Demostene Romanucci, Latham

Allegany (*1*)
M. Keith Sale, Wellsville

Bronx (*10*)
John P. Albanese, Bronx
Sana Bloch, Bronx
Dattatraya G. Lanjewar, Pelham Manor
Charles LaVerdi, Bronx
Stanley Luftschein, Rye
Hugo M. Morales, Bronx
Narayan Pai, Bronx
Venkatachala Pathy, Bronx
Raul Pohorille, New Rochelle
Juana Toporovsky, Bronx

Broome (*4*)
Louis W. Giordano, Endicott
Antonio F. LaSorte, Binghamton
Theodore R. Poritz, Binghamton
Charles Rubin, Johnson City

Cattaraugus (*1*)
Joel Levine, Olean

Cayuga (*1*)
Richard J. Freeman, Auburn

Chautauqua (*2*)
Bert W. Rappole, Jamestown
Nadia Geleil, Jamestown

Chemung (*2*)
Donald L. Brooks, Elmira
John Forrest, Elmira

Chenango (*1*)
Norman C. Lyster, Norwich

Clinton (*2*)
Soham S. Patel, Plattsburgh

Columbia (*2*)
Neil Baillargeon, Valatie
Jeffrey I. Monkash, Hudson

Cortland (*1*)
S. Robert Paik, Cortland

Delaware (*1*)
Glen Joshpe, Stamford

Dutchess (*4*)
Stephen R. Chernay, Fishkill
Eleanor C. Kane, Poughkeepsie
Florian M. Cortese, Hyde Park
Pinaki Ray, Poughkeepsie

Erie (*9*)
Thomas W. Bradley, Kenmore
Amy P. Early, Buffalo
Evan J. Evans, Buffalo
Edmond J. Gicewicz, Buffalo
Allen L. Lesswing, Buffalo
William K. Major, Jr., Buffalo
M. Luther Musselman, Buffalo
Nancy H. Nielson, Orchard Park
Paul H. Wierzbieniec, Snyder

Essex (*1*)
Glen D. Chapman, Ticonderoga

Franklin (*1*)

Fulton (*1*)

Genesee (*1*)
David A. Parker, Batavia

Greene (*1*)
Karuvath Enu, Hudson

Herkimer (*1*)
Krishna K. Vadlamudi, Ilion

Jefferson (*2*)
W. Bruce Carter, Watertown
David O. VanEenenaam, Watertown

Kings (*19*)
Judithann Anderson, Brooklyn
Norman S. Blackman, Brooklyn

Randall D. Bloomfield, Brooklyn
Joseph R. Brennan, Brooklyn
George W. Calvosa, Brooklyn
Vernal G. Cave, Brooklyn
Duncan W. Clark, Brooklyn
Clifford Cohen, Brooklyn
Paul Finkelstein, Brooklyn
Ladislav P. Hinterbuchner, Rye
Julius Holtzman, Brooklyn
Monte Malach, Brooklyn
Martin Markowitz, Brooklyn
A. W. Martin Marino, Jr., Brooklyn
Bentley D. Merrim, Brooklyn
Hubert S. Pearlman, Brooklyn
Abdul Rehman, Staten Island
Veronica Santilli, Brooklyn
Marcus Wiener, Brooklyn

Lewis (*1*)
John C. Herrman, Lowville

Livingston (*1*)
Sheppard Arluck, Geneseo

Madison (*1*)
Leonard A. Argentine, Oneida

Monroe (*7*)
Marilyn R. Brown, Rochester
Arthur J. Florack, Rochester
William A. Kern, Jr., Rochester
Mario B. LoMonaco, Rochester
Joyce M. McChesney, Rochester
Alexander L. Strasser, Rochester
Edward C. Tanner, Rochester

Montgomery (*2*)
Charles J. Bertuch, Amsterdam
Feori F. Pipito, Amsterdam

Nassau (*11*)
Robert Bruce Bergmann, Massapequa
Lee S. Binder, Valley Stream
Samuel M. Gelfand, Rockville Centre
Paul A. Hamlin, Great Neck
Jeffrey T. Kessler, Great Neck
Arnold D. Lurie, Baldwin
Sidney Mishkin, Fort Salonga
Felix A. Monaco, Rockville Centre
John A. Ostuni, Freeport
E. John Pesiri, Massapequa
Arthur J. Wise, Jr., Great Neck

New York (*24*)
Arnold L. Bachman, New York
Melvin H. Becker, New York
Robert S. Bernstein, New York
Michael B. Brodin, New York
Joseph B. Cleary, New York
Anthony A. Clemendor, New York
Irwin J. Cohen, New York
Hugh Clark Davidson, New York
Sally Faith Dorfman, New York
Jonas M. Goldstone, New York
Wilbur J. Gould, New York
Michael H. Grieco, New York
George Hyams, New York
Virginia Kanick, New York
Vance Lauderdale, Jr., New York
Samuel H. Madell, New York
Aaron G. Meislin, New York
Alfred Moldovan, New York
Donna B. O'Hare, New York
Stuart Orsher, New York
Richard N. Pierson, Jr., New York
William B. Rawls, Pleasant Valley
William B. Rosenblatt, New York
Clifford L. Spingarn, New York

Niagara (*3*)
Mary Ann Z. Bishara, Lewiston
Claus M. Fichte, Niagara Falls
James J. Kropelin, Niagara Falls

Oneida (*4*)
Gregory B. Brown, Utica
Robert M. George, Utica
Neville W. Harper, Rome
Sheldon Waldman, Rome

Onondaga (*7*)
Armand J. Cincotta, Liverpool
Daniel L. Dombroski, Syracuse
John A. Hoepner, Syracuse
Richard A. Konys, Syracuse
Frank J. Piper, Syracuse
Jeffrey Sneider, Baldwinsville
George P. Tilley, Syracuse

Ontario (*2*)
Verne M. Marshall, Geneva
Kenneth T. Steadman, Geneva

Orange (*4*)
Jacob J. Barie, Middletown
Stanley F. Brunn, Port Jervis
Michael A. Parmer, Port Jervis
Seymour J. Weiner, Port Jervis

Orleans (*1*)
David B. L. Meza III, Albion

Oswego (*1*)
Corliss Varnum, Oswego

Otsego (*3*)
Rodman D. Carter, Cooperstown
John O. Lusins, Oneonta
Jonathan Sastic, Oneonta

Putnam (*1*)

Queens (*16*)
Marvin L. Blumberg, Jamaica
Daniel Chansky, Forest Hills
John W. V. Cordice, Jr., St. Albans
Daniel G. Deckler, Richmond Hill
Alexander M. De le Garza, Flushing
Fred S. Fensterer, Glendale
Fred N. Flatau, Flushing
Lorraine M. Giordano, Jamaica Estates
Victor Guarneri, Bayside
Nicetas H. Kuo, Great Neck
Maxwell J. Marder, Forest Hills
Frank Ratner, Flushing

Hilda Ratner, Beechurst
Sheldon D. Sax, Whitestone
Ezra A. Wolff, Manhasset
Bernard M. Zazula, Kew Gardens

Rensselaer (*2*)
Arsenio G. Agopovich, Watervliet
William A. Whyland, Troy

Richmond (*4*)
Albert B. Accettola Jr., Staten Island
Robert Bonvino, Staten Island
Tano S. Carbonaro, Staten Island
Gerald J. Lustig, Staten Island

Rockland (*4*)
Herbert Z. Geller, New City
Roy H. Lieberman, Nanuet
Michael Kalvert, New City
Stanley H. Oransky, Bardonia

St. Lawrence (*1*)
Brian McMurray, Canton

Saratoga (*1*)
Allen T. Jones, Saratoga Springs

Schenectady (*3*)
George J. Giokas, Schenectady
Richard H. Lange, Schenectady
Robert J. Pletman, Schenectady

Schoharie (*1*)
Frederick J. Michel, Cobleskill

Schuyler (*1*)
L. Titus Parker, Watkins Glen

Seneca (*1*)
Donald W. Bidwell, Seneca Falls

Steuben (*2*)
Jack D. O'Neil, Corning
C. Susan Rainwater, Hornell

Suffolk (*11*)
William H. Bloom, Bay Shore
Stephen I. Braitman, Islip Terrace
Clive D. Caplan, Islip Terrace
Frank R. Collier, Port Jefferson
John G. Egner, West Islip
Robert A. Held, Islip Terrace
Kenneth B. Levites, West Sayville
Philip A. Mantia, Smithtown
Gerard F. Ryan, Patchogue
Stanley A. Steckler, Smithtown
William H. Weir, Jr., Riverhead

Sullivan (*1*)
Paul D. Salzberg, Callicoon

Tioga (*1*)

Tompkins (*2*)
Robert A. Hesson, Ithaca
Elliot Rubinstein, Ithaca

Ulster (*2*)

Warren (*3*)
Richard Hughes, Sr., Glens Falls
Robert A. Reid, Glens Falls
Thomas A. Quaresisma, Glens Falls

Washington (*1*)
Nadav Sharon, Cambridge

Wayne (*3*)
William J. Braell, Palmyra
David T. Hannan, Newark
Daniel J. Koretz, Ontario

Westchester (*9*)
Ann C. Cea, Rye Brook
Mark L. Fox, Bronxville
Sanford A. Kaplan, Bronxville
Thomas A. Lansen, Valhalla
Stanley M. Mendelowitz, Tarrytown
Robert C. Mickatavage, Rye
Toni G. Novick, New Rochelle
Pellegrino J. Tozzo, New Rochelle
William J. Walsh, Jr., White Plains

Wyoming (*1*)
Reginald Ishman, Warsaw

Yates (*1*)
Michael J. Boquard, Penn Yan

Medical School Delegates (12)
Albany Medical—Henry Pohl, Albany
Albert Einstein College of Medicine of Yeshiva University of New York—Melvin N. Zelefsky, Bronx
Columbia University College of Physicians and Surgeons—Linda D. Lewis, New York
Cornell University Medical College—
Mount Sinai School of Medicine of the City University of New York—
New York Medical College—Marilyn Pearl, Great Neck
New York University of Medicine—Stanford Wessler, New York
State University of New York Health Science Center at Brooklyn—
State University of New York at Buffalo School of Medicine—Robert L. Scheig, Erie
State University of New York at Stony Brook School of Medicine—Dorothy S. Lane, Suffolk
State University of New York Health Science Center at Syracuse—John Bernard Henry, Syracuse
University of Rochester School of Medicine and Dentistry—Philip P. Bonanni, Rochester

Medical Student Delegates (3)
Scot Glasberg, New York
Howard Pomeranz, New York
Lloyd Roberts, Westchester

Resident Membership Delegate (1)
Paul Pipia, Richmond

Hospital Medical Staff Delegate (1)
Vicki Lynn Seltzer, Nassau

REFERENCE COMMITTEES

Reports of Officers and Administrative Matters

William B. Rosenblatt, New York, *Chairman*
Paul A. Hamlin, Nassau
Edmond Gicewicz, Erie
Verne M. Marshall, Ontario
Marilyn Pearl, New York Medical College
Jeffrey L. Margulies, New York Chapter, American College of Emergency Physicians (Observer)

Bylaws

Vernal G. Cave, Kings, *Chairman*
Daniel Chansky, Queens
Robert M. Kohn, New York State Society of Internal Medicine
Samuel H. Madell, New York
Anton A. Vreede, Sixth District Branch
James F. Wright, New York State Academy of Family Physicians (Observer)

Governmental Affairs and Legal Matters

Louis W. Giordano, Broome, *Chairman*
Anthony Clemendor, New York
Robert A. Hesson, Tompkins
Pinaki Ray, Dutchess
Nathan P. Reed, Albany
Mark L. Fox, Westchester (Observer)

Socio-Medical Economics

Veronica C. Santilli, Kings, *Chairwoman*
Kenneth B. Levites, Suffolk
David B. L. Meza, III, Orleans
John A. Ramsdell, Ninth District Branch
James J. Kropelein, Niagara
David T. Hannan, Wayne (Observer)

Scientific and Educational Activities

Stanley H. Oransky, Rockland, *Chairman*
Howard Pomeranz, Medical Student Membership
Hugh Clark Davidson, Jr., New York
Nancy H. Nielsen, Erie
Henry S. Pohl, Albany Medical College
Michael A. Parmer, Orange (Observer)

Hospital and Medical Facilities

John C. Herrman, Lewis, *Chairman*
Daniel L. Dombroski, Onondaga
Robert A. Held, Suffolk
Richard H. Lange, Schenectady
Frank Ratner, Queens
Eleanor C. Kane, Dutchess (Observer)

CONVENTION COMMITTEES

Credentials

E. John Pesiri, Nassau, *Chairman*
James Puleo, Albany
Clive D. Caplan, Suffolk
Antonio F. LaSorte, Broome
Seymour J. Wiener, Orange
Paul Pipia, Richmond (Observer)

Sergeant at Arms

Arthur J. Florack, Monroe, *Chairman*
Kalman Post, New York State Neurosurgical Society, Inc.
Sharon C. Mead, Nassau
Hugo M. Morales, The Bronx
Feori F. Pipito, Montgomery
Lloyd Roberts, Medical Student Membership (Observer)

Rules and Order of Business

Richard Hughes, Sr., Warren, *Chairman*
Fred N. Flatau, Queens
Roy H. Lieberman, Rockland
Gerald J. Lustig, Richmond
Venkatachala Pathy, The Bronx
John Forrest, Chemung (Observer)

Tellers

Albert Accettola, Richmond, *Chairman*
Ann Cea, Westchester
Neville W. Harper, Oneida
Claus Fichte, Niagara
David Van Eenenaam, Jefferson
Richard J. Freeman, Cayuga (Observer)

NOMINATING COMMITTEE

To the House of Delegates, Ladies and Gentlemen:

In accordance with Article XI, Section 2 of the Bylaws of the Medical Society of the State of New York, the Nominating Committee met at the Society's headquarters, 420 Lakeville Road, Lake Success, New York, on Wednesday, November 8, 1989 at 11:00 a.m.

Members of the Committee present were:

Member-at-Large—**Charles D. Sherman, Jr., M.D.**, Monroe, *Chairman*
First District Branch—**John A. Finkbeiner, M.D.**, New York
Second District Branch—**Robert B. Bergmann, M.D.**, Nassau
Third District Branch—**John H. Carter, M.D.**, Albany
Fourth District Branch—**David M. Benford, M.D.**, New York
Fifth District Branch—**David O. Van Eenenaam, M.D.**, Jefferson
Sixth District Branch—**Antonio F. LaSorte, M.D.**, Broome
Seventh District Branch—**William A. Dolan, M.D.**, Monroe
Eighth District Branch—**Nancy H. Nielsen, M.D.**, Ph.D., Erie
Ninth District Branch—**John A. Ramsdell, M.D.**, Westchester
Member-at-Large—**Seymour R. Stall, M.D.**, Dutchess

Excused were: Arsenio G. Agopovich, M.D., Third District Branch, and Richard Hughes, Sr., M.D., Fourth District Branch. President David M. Benford, M.D., designated John H. Carter, M.D., to represent the interest of the Third District Branch and David M. Benford, M.D., to represent the interest of the Fourth District Branch.

After careful consideration of the recommendations submitted by the district branches, the county medical societies, and the resident and student sections in response to a memorandum, dated July 7, 1989, your Committee respectfully nominated the following candidates for election on April 1, 1990:

President—**Robert A. Mayers, M.D.**, Westchester
President-Elect—**Charles N. Aswad, M.D.**, Broome
Vice-President—**Morton Kurtz, M.D.**, Queens
Secretary—**George Lim, M.D.**, Oneida
Assistant Secretary—**Joyce M. McChesney, M.D.**, Monroe
Treasurer—**James H. Cosgriff, Jr., M.D.**, Erie
Assistant Treasurer—**Antonio F. LaSorte, M.D.**, Broome
Speaker—**Seymour R. Stall, M.D.**, Dutchess
Vice-Speaker—**John H. Carter, M.D.**, Albany
Councilors (*four for three years*):—**Randall D. Bloomfield, M.D.**, Kings
—**William A. Dolan, M.D.**, Monroe
—**Robert M. George, M.D.**, Oneida
—**Stuart I. Orsher, M.D.**, New York
Resident Councilor (*one for one year*):—**Malcolm Reid, M.D.**, New York
Student Councilor (*one for one year*):—* **Howard Pomeranz**, New York
Trustee (*one for five years*):—**Charles D. Sherman, Jr., M.D.**, Monroe

Delegates to the American Medical Association for two years commencing January 1, 1991:

Charles N. Aswad, M.D., Broome
Randall D. Bloomfield, M.D., Kings
John L. Clowe, M.D., Schenectady
Hugh Clark Davidson, Jr., M.D., New York
William A. Dolan, Jr., M.D., Monroe
Richard D. Eberle, M.D., Onondaga
Robert E. Fear, M.D., Suffolk
Louis W. Giordano, M.D., Broome
Robert E. Gordon, M.D., Kings
Stanley L. Grossman, M.D., Orange
James Kropelin, M.D., Niagara
Dattatraya G. Lanjewar, M.D., The Bronx
Samuel H. Madell, M.D., New York
Robert A. Mayers, M.D., Westchester
Joyce M. McChesney, M.D., Monroe
Sidney Mishkin, M.D., Nassau
Donna B. O'Hare, M.D., New York
Richard N. Pierson, Jr., M.D., New York
Paul A. Pipia, M.D., (Resident), Richmond
William B. Rosenblatt, M.D., New York
Anthony P. Santomauro, M.D., Erie
Ralph E. Schlossman, M.D., Queens
Ralph Schwartz, M.D., New York
* **Tara Solomon**, The Bronx
Stanley A. Steckler, M.D., Suffolk
George T. C. Way, M.D., Dutchess

The twelve nominees receiving the largest number of votes will be delegates; the second twelve will be alternates.

Respectfully submitted:
Charles D. Sherman, Jr., M.D., *Chairman*

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* Medical Student

HOUSE COMMITTEE ON BYLAWS

To the House of Delegates, Ladies and Gentlemen:

The members of the House Committee on Bylaws are as follows:

George Lim, M.D., Chairman	Oneida
Marilyn R. Brown, M.D.	Monroe
Stanley F. Brunn, M.D.	Orange
Rodman D. Carter, M.D.	Otsego
Robert M. Kohn, M.D.	Erie
Sidney Mishkin, M.D.	Nassau
Donna B. O'Hare, M.D.	New York
Hilda Ratner, M.D.	Queens
Stanley A. Steckler, M.D.	Suffolk
David M. Benford, M.D., President, ex officio	New York
Seymour R. Stall, M.D., Speaker, ex officio	Dutchess
Donald F. Foy	Executive Vice President, ex officio
William R. Egan	Deputy Executive Vice President, ex officio
Donald R. Moy	General Counsel, ex officio

A meeting of the Reference Committee on Bylaws was held at the Holiday Inn Hotel in Rochester, New York on Friday, April 7, 1989. The Reference Committee prepared a transcript of testimony and a summary of its recommendations to the House Committee on Bylaws. The input received from the meeting of the Reference Committee on Bylaws proved to be of great assistance to the House Committee on Bylaws when it met on October 26, 1989.

Eleven resolutions asking for amendments to the Bylaws were referred to the Committee for consideration. Resolution 88-9 was referred back to the Council for further evaluation. In all, twelve resolutions are under consideration.

Resolution 89-5, Councilor Representing the Hospital Medical Staff Section

Introduced by Medical Society of the County of Lewis

Whereas, Many of the regulatory issues confronting the medical profession today relate to medical practice in the hospital environments; and

Whereas, It is important to have a strong communications link between the MSSNY Council and the Society's hospital medical staff section; and

Whereas, This can best be accomplished through formal hospital medical staff section representation on the Council, and state medical societies have been encouraged by the AMA to provide for such representation; and

Whereas, The medical student and resident physician sections of the MSSNY are represented on the Council; therefore be it

Resolved, That the Bylaws of the Medical Society of the State of New York be amended to provide that the Chairperson of the Hospital Medical Staff Section be a voting member of the MSSNY Council; and be it further

Resolved, That Article XII-A, paragraph 1 of the Bylaws of the Medical Society of the State of New York be amended to read as follows (additions italicized):

"The hospital medical staff section is authorized to be represented in the House of Delegates by one delegate, *and on the MSSNY Council by one councilor, who shall be the individual elected by the section as its chairperson.* The hospital medical staff section shall represent physicians on the medical staffs of acute care hospitals located in New York State; of psychiatric inpatient facilities maintained by the State of New York or its political subdivisions; and of hospitals with inpatient programs certified by the New York State Office of Mental Health."

and be it further

Resolved, That Article XI, Section 2, paragraph 2 under the heading "Nominating Committee" be amended to read as follows (additions italicized, deletions in brackets):

"In the event that a member of the Nominating Committee who has been selected as the representative of a district branch (or his alternate) shall be unable to attend the meeting of this committee, the president shall appoint an at large member, from the district branch whose representative is absent, to represent the interests of that district on the Nominating Committee. It shall be the duty of this committee to propose and nominate members of the Medical Society of the State of New York for all vacancies to be filled at the ensuing annual meeting of the House of Delegates, *with the exception of the councilor representing the hospital medical staff section, who shall be a member of the Medical Society of the State of New York nominated by the hospital medical staff section.* [These] *Nominating Committee* recommendations shall be made to the House of Delegates in the same manner as prescribed in Article X, Section 1, paragraph 2."

and be it further

Resolved, That Article IV, Section 1, paragraphs 1, 2, 3, and 6 be amended to read as follows (additions italicized, deletions in brackets):

"The Council shall be composed of the president, president-elect, vice-president, immediate past-president, secretary, assistant secretary, treasurer, assistant treasurer, speaker, vice-speaker, chairman of the Board of Trustees, and [14] *15* councilors elected by the House of Delegates.

The trustees, the executive vice-president, the deputy executive vice-president, and the general counsel of the Medical Society of the State of New York shall attend all meetings of the Council with voice but without vote. The councilors from the *hospital medical staff section and* the resident physician section shall attend all meetings of the Council with voice and with vote. The councilor from the medical student section shall attend all meetings with voice but without vote.

Four councilors shall be elected annually by the House of Delegates, each for a term of three years. Two councilors, one councilor representing the medical student section and one councilor representing the resident physician section to the Medical Society of the State of New York, shall be elected every year by the House of Delegates, each for a term of one year. *One councilor, representing the hospital medical staff section, shall be elected every other year by the House of Delegates, for a term of two years.* Article IV, Section 1, paragraph 4 is not applicable to the term of office of a resident or student councilor, *or a councilor representing the hospital medical staff section.* In the event of a vacancy, a councilor shall be elected by the Council to serve until the next meeting of the House of Delegates, at which time the House of Delegates shall elect a councilor to fill the unexpired term.

Councilors, other than the [councilor representing the student membership and the councilor representing the resident membership,] *councilors representing the medical student and resident physician sections and the hospital medical staff section,* shall be assigned to specific county societies as liaison for the Council in accordance with the provisions of Article V, Section 2. Councilors shall be required to disseminate information of Council activity as well as returning information to the Council, and shall report regularly to the Council on their activity. The councilor representing the medical student section must be a student member of the Medical Society of the State of New York; the councilor representing the resident physician section must be a resident member of the Medical Society of the State of New York.

and

Resolution 89-100, Councilor Representing the Hospital Medical Staff Section

Introduced by Hospital Medical Staff Section

Whereas, Many of the regulatory issues confronting the medical profession today relate to medical practice in the hospital environment; and

Whereas, It is important to have a strong communications link between the MSSNY Council and the Society's hospital medical staff section; and

Whereas, This can best be accomplished through formal hospital medical staff section representation on the Council, and state medical societies have been encouraged by the AMA to provide for such representation; and

Whereas, The medical student and resident physician sections of the MSSNY are represented on the Council; therefore be it

Resolved, That the Bylaws of the Medical Society of the State of New York be amended to provide that the Chairperson of the Hospital Medical Staff Section be a voting member of the MSSNY Council; and be it further

Resolved, That Article XII-A, paragraph 1 of the Bylaws of the Medical Society of the State of New York be amended to read as follows (additions italicized):

"The hospital medical staff section is authorized to be represented in the House of Delegates by one delegate, *and on the MSSNY Council by one councilor, who shall be the individual elected by the section as its chairperson.* The hospital medical staff section shall represent physicians on the medical staffs of acute care hospitals located in New York State; of psychiatric inpatient facilities maintained by the State of New York or its political subdivisions; and of hospitals with inpatient programs certified by the New York State Office of Mental Health."

and be it further

Resolved, That Article XI, Section 2, paragraph 2 under the heading "Nominating Committee" be amended to read as follows (additions italicized, deletions in brackets):

"In the event that a member of the Nominating Committee who has been selected as the representative of a district branch (or his alternate) shall be unable to attend the meeting of this committee, the president shall appoint an at large member, from the district branch whose representative is absent, to represent the interests of that district on the Nominating Committee. It shall be the duty of this committee to propose and nominate members of the Medical Society of the State of New York for all vacancies to be filled at the ensuing annual meeting of the House of Delegates, *with the exception of the councilor representing the hospital medical staff section, who shall be a member of the Medical Society of the State of New York nominated by the hospital medical staff section.* [These] *Nominating Committee* recommendations shall be made to the House of Delegates in the same manner as prescribed in Article X, Section 1, paragraph 2."

and be it further

Resolved, That Article IV, Section 1 be amended to read as follows (additions italicized, deletions in brackets):

"The Council shall be composed of the president, president-elect, vice-president, immediate past-president, secretary, assistant secretary, treasurer, assistant treasurer, speaker, vice-speaker, chairman of the Board of Trustees and [14] *15* councilors elected by the House of Delegates.

The trustees, the executive vice-president, the deputy executive vice-president, and the general counsel of the Medical Society of the State of New York shall attend all meetings of the Council with voice but without vote. The councilors from *the hospital medical staff section and* the resident physician section shall attend all meetings with voice and with vote. The Councilor from the medical student section shall attend all meetings of the Council with voice but without vote.

Four councilors shall be elected annually by the House of Delegates, each for a term of three years. Two councilors, one councilor representing the medical student section and one councilor representing the resident physician section to the Medical Society of the State of New York, shall be elected every year by the House of Delegates, each for a term of one year. *One councilor, representing the hospital medical staff section, shall be elected every other year by the House of Delegates, for a term of two years.* Article IV, Section 1, paragraph 4 is not applicable to the term of office of a resident or student councilor, *or a councilor representing the hospital medical staff section.* In the event of a vacancy, a councilor shall be elected by the Council to serve until the next meeting of the House of Delegates, at which time the House of Delegates shall elect a councilor to fill the unexpired term.

No person shall serve, consecutively, more than two terms as councilor. An unexpired term shall not be construed as a term of office.

The councilors shall assume office on election and shall hold office until their successors are duly elected and qualified.

Councilors, other than the [councilor representing the student membership and the councilor representing the resident membership,] *councilors representing the medical student and resident physician sections and the hospital medical staff section*, shall be assigned to specific county societies as liaison for the Council in accordance with the provisions of Article V, Section 2. Councilors shall be required to disseminate information of Council activity as well as returning information to the Council, and shall report regularly to the Council on their activity.

The councilor representing the medical student section must be a student member of the Medical Society of the State of New York; the councilor representing the resident physician section must be a resident member of the Medical Society of the State of New York.

A councilor shall practice or maintain a residence or an office in the State of New York. Otherwise his position in the Medical Society of the State of New York shall be declared vacant by the Council.

If a councilor is absent from three consecutive regular meetings of the Council without proper excuse, the Council shall declare his position vacant and elect a successor for the unexpired term.

Because of the similarity of content, Resolutions 89-5 and 89-100 were considered at the same time.

The Committee believes that the interests of the hospital medical staff section are well represented in the meetings of the Medical Society of the State of New York. The hospital medical staff section of the Medical Society of the State of New York has a strong voice in the House of Delegates. Resolutions submitted by the hospital medical staff section are fully evaluated by the House of Delegates. Recognizing that the hospital medical staff section usually holds its annual meeting in conjunction with the House of Delegates, the House of Delegates has accepted late resolutions submitted by the hospital medical staff section. As a result of the adoption of Resolution 88-83 last year, late resolutions submitted by the hospital medical staff section, when it meets in conjunction with the House of Delegates, are automatically accepted by the House of Delegates.

In addition, the interests of the medical staff are represented in meetings of specialty medical societies, county medical societies and the State Medical Society since many, if not most, physicians are members of hospital medical staffs.

Since there are many avenues through which issues of interest to hospital medical staffs may be raised and fully explored, the committee does not believe that it is necessary to amend the Bylaws to provide for a councilor representing the hospital medical staff section.

Accordingly, the Committee recommends that Resolutions 89-5 and 89-100 **NOT BE ADOPTED.**

Resolution 89-33, Affiliate Membership Category for Physicians Residing Outside the State of New York

Introduced by the Council

Whereas, Increasing numbers of physicians are leaving New York State due to socioeconomic factors and disincentives to practice medicine here; and

Whereas, Some of these physicians have expressed an interest in maintaining membership in the Medical Society of the State of New York, yet the current Bylaws require that all but student members, life members and honorary members must reside or practice medicine within the State of New York; and

Whereas, Various state medical societies, including those in Connecticut, Indiana, Ohio, Pennsylvania and Texas, allow out-of-state physicians to remain or become members provided the physician is also a member of the county and state medical societies where he or she resides or practices; therefore be it

Resolved, That Article II, Section 1, paragraph 1 of the Bylaws of the Medical Society of the State of New York be amended to read as follows (additions italicized, deletions in brackets):

The membership of the Medical Society of the State of New York shall be divided into [five] *six* classes: (a) active, (b) life, (c) honorary, (d) resident, [and] (e) student, *and (f) affiliate.*

and be it further

Resolved, That Article II, Section 1 be amended by the addition of a new paragraph, to read as follows (additions italicized):

Affiliate members shall be those physicians, other than life or honorary members, who neither reside nor practice medicine in New York State, yet wish to be affiliated with the Medical Society of the State of New York. The affiliate members shall be active members in good standing of the county and state medical societies in the state where they reside or practice medicine.

and be it further

Resolved, That Article II, Section 2 be amended by the addition of a new paragraph, to read as follows (additions italicized):

Affiliate members shall be entitled to receive the New York State Journal of Medicine, the News of New York, and the Medical Directory of New York State, and to attend meetings of the Medical Society of the State of New York.

and be it further

Resolved, That Article XV, paragraph 3 be amended to read as follows (additions italicized):

The dues and assessments of those active members who have been graduated from medical or osteopathic college not more than five calendar years, not counting active service in the armed forces of the United States or in the United States Public Health Service, shall be one third the amount levied on all other active members. The dues and assessments of those active members who have not completed their continuous residency training shall be one tenth the amount levied on all other active members. In no case may an active physician continue to pay such reduced dues more than seven years after the date of graduation from medical or osteopathic school. The dues and assessments of all active members entering practice for the first time, who are not otherwise eligible for reduced dues as set forth in prior provisions in this paragraph, shall be one half the amount levied on all other active members for the first two years of practice. Once an active member pays full dues, he shall no longer qualify for half-dues status *as a beginning practitioner. The dues and assessments of affiliate members shall be one half the amount levied on full dues paying active members.*

After a thorough deliberation, the Committee recommends the following Substitute Resolution:

Resolved, That Article II, Section 1, paragraph 1 of the Bylaws of the Medical Society of the State of New York be amended to read as follows (additions italicized, deletions in brackets):

The membership of the Medical Society of the State of New York shall be divided into [five] *six* classes: (a) active, (b) life, (c) honorary, (d) resident, [and] (e) student, *and (f) affiliate.*

and be it further

Resolved, That Article II, Section 1 be amended by adding new paragraphs 12, 13 and 14 to read as follows (additions italicized):

Affiliate members shall be those physicians other than life or honorary members who were previously members in good standing of a component county medical society and the Medical Society of the State of New York who no longer reside or practice medicine in New York State, yet wish to be affiliated with the Medical Society of the State of New York. The affiliate members shall be active members in good standing of county and state medical societies in the state where they reside or practice medicine.

In order to qualify for affiliate membership, the physician must have been a member in good standing of a component county medical society and the Medical Society of the State of New York at the time the physician terminated the residence and/or practice of medicine in New York State. An affiliate member who reestablishes either residence or the practice of medicine in New York State shall no longer qualify for affiliate membership but must apply for an appropriate class of membership. Any dues paid for affiliate membership in the year of reapplication shall be credited towards the dues required for the applicable class of membership.

In the event that an affiliate member resumes the status as an active member of a component county medical society and the Medical Society of the State of New York following the reestablishment of residence and/or the practice of medicine in the State of New York, the duration of time in which the individual held the status as an affiliate member in good standing shall not be considered as an interruption in active membership for the purpose of determining eligibility for life membership in accordance with paragraph 6 of this section.

and be it further

Resolved, That Article II, Section 2 be amended by a new paragraph 7 to read (additions italicized):

Affiliate members shall be accorded all the rights and privileges of active membership in the Medical Society of the State of New York but shall not vote and hold office, and shall be entitled to receive such publications of the Medical Society of the State of New York as are approved by the Council on an annual basis.

and be it further

Resolved, That Article XV of the Bylaws be amended by the addition of a new paragraph 5 to read (additions italicized):

The dues and assessments of affiliate members shall be one half the amount levied on full dues paying active members.

Accordingly, the House Committee on Bylaws unanimously recommends that the Substitute Resolution for Resolution 89-33 be **ADOPTED.**

Resolution 89-34, Amnesty Periods for Reinstatement Without Penalty

Introduced by the Council

Whereas, Each year some 400 to 550 physicians are dropped from the rolls of membership of the Medical Society of the State of New York for nonpayment of dues; and

Whereas, In order to be reinstated as members, such physicians must pay dues for the year in which membership was dropped in addition to the current year's dues; and

Whereas, This reinstatement penalty does not apply to those who resigned from membership, yet members may not be aware of the need to resign in order to be exempted from the reinstatement penalty; and

Whereas, During the period from June 6, 1985 to April 1, 1986, when the reinstatement penalty was temporarily waived by an action of the House of Delegates, the number of reinstated members nearly doubled as compared to previous periods when the penalty was required; therefore be it

Resolved, That Article II, Section 2, paragraph 15 of the Bylaws of the Medical Society of the State of New York be amended to read as follows (additions italicized, deletions in brackets):

The provision in Article II, Section 2, paragraph 14 that no member dropped for nonpayment of dues and assessments shall be reinstated until he has, in addition, paid his dues and assessments for the year in which he was dropped, shall not be in effect [for the period beginning June 6, 1985 through April 1, 1986] *during amnesty periods as declared by the House of Delegates or the Council.*

and be it further

Resolved, That Article XV, paragraph 9 be amended to read as follows (additions italicized, deletions in brackets):

The provision in Article XV, paragraph 8 that no member dropped for nonpayment of dues and assessments shall be reinstated until he has, in addition, paid his dues and assessments for the year in which he was dropped, shall not be in effect [for the period beginning June 6, 1985 through April 1, 1986] *during amnesty periods as declared by the House of Delegates or the Council.*

PAST HOUSE ACTION: Resolution 87-72

This resolution would authorize the House of Delegates or the Council to declare amnesty periods to the reinstatement penalty contained in Article XV, paragraph 8 of the Bylaws. The Committee does not believe that the reinstatement penalty is overly severe. The Committee believes that forgiving the reinstatement penalty may only encourage non-payment of dues.

Accordingly, the Committee unanimously recommends that Resolution 89-34 **NOT BE ADOPTED.**

Resolution 89-39, Unified Membership With the AMA

Introduced by Nassau County Medical Society

Whereas, Federal government legislation and regulations are increasingly interfering with the physicians' autonomy to practice medicine; and

Whereas, This interference is now invading the clinical decision-making process; and

Whereas, This is affecting the physician/patient relationship; and

Whereas, This is inhibiting the maintenance of an accessible, quality health care system; and

Whereas, The medical profession's ability to correct present inequities and prevent further inequities is firmly dependent upon a strong unified voice in Washington, D.C.; therefore be it

Resolved, That New York State become a unified membership state requiring membership in the county medical society, the Medical Society of the State of New York and the American Medical Association to belong to any of the three; and be it further

Resolved, That Article II, Section 1, paragraph 3 of the Bylaws of the Medical Society of the State of New York be amended to read as follows (additions italicized):

The active members shall be all active members in good standing of the component county medical societies *and the American Medical Association*. A copy of the roster of such members, certified to be correct by the respective secretary of each component county medical society, shall be evidence of the right of the members whose names appear therein to membership in the Medical Society of the State of New York.*

Many vital issues affecting health care are pending at the national level. For this reason, it is important that the medical profession be strong at the national, state and local levels. The Committee believes that membership in the AMA should be strongly encouraged, but does not believe that membership in the AMA should be required as a condition for membership in the State and component county medical societies. In addition, last year, the House of Delegates approved Resolution 88-12 which amended Article XVIII, Section 2, paragraph 1 of the Bylaws of the Medical Society of the State of New York to allow each component county medical society to decide the question of unified membership with the AMA. The Committee believes that the impact of resolution 88-12 should be carefully evaluated before mandatory AMA membership is considered on a statewide basis.

Accordingly, the Committee recommends that Resolution 89-39 **NOT BE ADOPTED.**

Resolution 89-42, Conversion to Resident Member Status for Graduating Student Members of MSSNY

Introduced by the Resident Physician Section

Whereas, Approximately 250 student members of the Medical Society of the State of New York have graduated from medical school in each of the last several years; and

Whereas, Student members who wish to continue their affiliation with the MSSNY during residency training are required to re-apply for membership through their local county medical society; and

Whereas, Only 14 individuals who graduated from medical school in 1987 were elected to membership as residents in their first year of training; and

Whereas, Of the students who graduated in 1988, 59 have been elected to resident membership in the first eight months of training, with all but four joining in those counties which adopted simplified application procedures, indicating that a major barrier to membership for residents is the application process itself; and

Whereas, It should not be necessary to again scrutinize such members who have been screened so recently for admission to residency training programs; therefore be it

Resolved, That former student members of the Medical Society of the State of New York in their first year of an ACGME-approved residency training program should be allowed to convert to resident member status without having to re-apply and be re-elected to membership; and be it further

Resolved, That Article XVIII, Section 2, paragraph 1 of the Bylaws of the Medical Society of the State of New York be amended to read as follows (additions italicized):

Eligibility for membership in a component county medical society shall be determined by that society, *except that a former student member of the Medical Society of the State of New York shall be permitted to convert to resident member status through his local county medical society during the first post-graduate year in an ACGME-approved residency training program upon payment of county and state society dues and provision of an updated information form.* Except by the approval of the Council no physician shall be an active member in a component county medical society other than that of the county in which he practices or maintains legal residence.

The Committee agreed that Resolution 89-42 represents a fair measure to expedite the application process.

Accordingly, the Committee unanimously recommends that Resolution 89-42 be **ADOPTED.**

Resolution 89-43, One Year Terms for Student and Resident Delegates to the AMA House of Delegates

Introduced by MSSNY-Medical Student Section

Whereas, Current MSSNY and AMA Bylaws require that Delegates to the AMA House of Delegates be elected

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** If Resolution 89-39 is adopted, various sections of the Bylaws will have to be revised to conform with unified membership requirements.*

for terms of two years and that they be elected in the calendar year preceding the meeting of the AMA House of Delegates to which they are elected; and

Whereas, These requirements present great difficulty for both the student and resident sections as the time from nomination until the delegate's tenure expires often extends beyond an individual's membership in the student or resident category; and

Whereas, The AMA is expected to amend its Bylaws in June 1989 to allow states the option to elect medical students and resident physicians to serve either one-year or two-year terms as delegates or alternate delegates to the AMA (Resolution 124, I-88); therefore be it

Resolved, That Article VIII, paragraph 3 of the Bylaws of the Medical Society of the State of New York be amended to read as follows (additions italicized):

The delegates to the American Medical Association shall be elected in the calendar year preceding the meeting of the House of Delegates of the American Medical Association to which they are elected. *Other than medical student or resident physician delegates or alternate delegates who shall serve for a term of one year*, one half of the allotted number of delegates and alternate delegates shall be elected each year.

and be it further

Resolved, That Article VIII, Section 1, paragraph 6 of the Bylaws be amended to read as follows (additions italicized):

The delegates and alternate delegates shall take office at the beginning of the calendar year following their election and shall serve for a term of two years, *except in the case of medical student or resident physician delegates or alternate delegates, who shall serve for a term of one year.*

The Committee agreed that Resolution 89-43 is necessary to address the problems encountered by the medical student and resident physician sections.

Accordingly, the Committee unanimously recommends that Resolution 89-43 be **ADOPTED.**

Resolution 89-47, Increase in the Number of Council Members

Introduced by Medical Society of the County of Westchester, Inc.

Whereas, An Ad Hoc Committee of the House of Delegates in 1975 recommended that the MSSNY Council be increased to 16 members; and

Whereas, The House recommended that the allocations be as follows:

District 1—New York and Richmond 3, Kings 1, Queens 1 Bronx 1
Districts 3–8—1 councilor each
Districts 2 & 9—Combined 4 councilors

and

Whereas, The reasoning behind the call for increasing the size of the MSSNY Council is as valid today as it was in 1975; therefore be it

Resolved, That the Medical Society of the State of New York amend Article IV, Section 1, paragraph 1 of the Bylaws to read as follows (additions italicized, deletions in brackets):

The Council shall be composed of the president, president-elect, vice president, immediate past president, secretary, assistant secretary, treasurer, assistant treasurer, speaker, vice-speaker, chairman of the Board of Trustees, and [14] *16* councilors elected by the House of Delegates.

and be it further

Resolved, That the MSSNY amend Article IV, Section 1, paragraph 3 of the Bylaws to read as follows (additions italicized, deletions in brackets):

[Four councilors shall be elected annually by the House of Delegates, each for a term of three years.] *Five councilors shall be elected by the House of Delegates two years in succession and four councilors shall be elected, all for a term of three years every third year.* Two councilors, one councilor representing the medical student section and one councilor representing the resident physician section to the Medical Society of the State of New York, shall be elected every year by the House of Delegates, each for a term of one year. Article IV, Section 1, paragraph 4 is not applicable to the term of office of a resident or student councilor. In the event of a vacancy, a councilor shall be elected by the Council to serve until the next meeting of the House of Delegates, at which time the House of Delegates shall elect a councilor to fill the unexpired term.

Similar resolutions have been introduced in recent years. The Committee believes that the present number of councilors is adequate to provide for a full representation of views. Councilors are representatives of the State Medical Society and are assigned to specific county medical societies as liaison for the Council. The Committee believes that the councilors are carrying out their duties as representatives of the State Medical Society in a responsible and conscientious manner and, therefore, it is not necessary to increase the number of councilors.

Accordingly, the Committee recommends that Resolution 89-47 **NOT BE ADOPTED.**

Resolution 89-48, Dues Structure for Dual Physician Families

Introduced by Medical Society of the County of Westchester, Inc.

Whereas, With increasing numbers of women in the medical profession today, there are increasing numbers of physicians married to physicians and practicing in the same county; and

Whereas, To avoid duplication of benefits and to save on professional expenses, many married physician couples decide that only one spouse will join the medical society; and

Whereas, The Medical Society of the State of New York wishes to have all qualified physicians as members; therefore be it

Resolved, That Article XV, paragraph 3 of the Bylaws of the Medical Society of the State of New York be amended to read as follows (additions italicized):

The dues and assessments of those active members who have been graduated from medical or osteopathic college not more than five calendar years, not counting active service in the armed forces of the United States or in the United States Public Health Service, shall be one third the amount levied on all other active members. The dues and assessments of those active members who have not completed their continuous residency training shall be one tenth the amount levied on all other active members. In no case may an active physician continue to pay such reduced dues more than seven years after the date of graduation from medical or osteopathic school. The dues and assessments of all active members entering practice for the first time, who are not otherwise eligible for reduced dues as set forth in prior provisions in this paragraph, shall be one half the amount levied on all other active members for the first two years of practice; *and if married to a full dues paying member the one half amount levied on all other active members shall be for the first five years of practice*; full dues shall be paid after the first two years of practice. Once an active member pays full dues, he shall no longer qualify for half-dues status.

The Committee does not believe that there is any reliable data showing that dual physician married couples experience financial hardship. The Bylaws of the Medical Society of the State of New York provide adequate remedies in specific cases where hardship is demonstrated.

Accordingly the Committee unanimously recommends that Resolution 89-48 **NOT BE ADOPTED.**

Resolution 89-51, Establishment of a Young Physician Section of the Medical Society of the State of New York

Introduced by Suffolk County Medical Society
Nassau County Medical Society
Fifth and Sixth District Branches of the Medical Society of the State of New York

Whereas, There are nearly 14,000 young physicians (those out of residency training and under the age of 40) in the State of New York; and

Whereas, Of these young physicians, only about 3,800 (28%) are members of the Medical Society of the State of New York; and

Whereas, This level of involvement compares very unfavorably with that for physicians age 40 and over (of whom 56% are members); and

Whereas, The recognition of similar trends at the national level has led the American Medical Association, after much study, to create a Young Physician Section to help identify and serve the needs of young physicians and to give them a voice in AMA policy-making; and

Whereas, The representational mechanism of the AMA Young Physician Section (YPS) calls for election of young physician delegates by each state medical society; and

Whereas, The AMA YPS has encouraged state medical societies to create young physician sections of their own to elect their delegates and to foster greater involvement by young physicians in state and county medical societies; therefore be it

Resolved, That the Medical Society of the State of New York amend its Bylaws to include a new Article XII-B to provide a Young Physician Section to read as follows (additions italicized):

The young physician section is authorized to be represented in the House of Delegates by one delegate. The young physician section shall represent physicians who have completed residency training and are under the age of 40 and physicians, regardless of age, who have completed residency training within the past five years. The method of electing the delegate representing the young physician section shall be determined by the Council.

and be it further

Resolved, That Article III, Section 1, paragraph 1 be amended to read as follows (additions italicized):

SECTION 1. Composition

The House of Delegates shall be composed of: (a) duly designated delegates from the component county medical societies; (b) officers of the Medical Society of the State of New York, councilors, and trustees; (c) a duly designated delegate from each district branch; (d) a duly designated delegate from each recognized specialty society; (e) duly designated delegates from the medical student section; (f) the Commissioner of Health of the State of New York, or a deputy designated by him, provided that any representative shall be a member of the State Society; (g) past-presidents of the State Society and any past president of the American Medical Association, provided that individual is a member of the Medical Society of the State of New York who shall be members for life; (h) any past executive vice-president of the State Society, who shall be a member for life, provided that individual is a member of the Medical Society of the State of New York, resides in the State of New York and is not otherwise a member of the House of Delegates for life in accordance with this section; (i) any past deputy executive vice-president of the State Society who has served a minimum of three years as deputy executive vice-president, who shall be a member for life, provided that the individual is a member of the State Society, resides in the State of New York, is not otherwise a member of the House of Delegates for life in accordance with this section, and is elected as a member for life by a majority of the members of the House of Delegates present and voting; (j) a representative from each of the medical schools in New York State, provided said representative is a member of the Medical Society of the State of New York; (k) a delegate representing the resident physician section; (l) a delegate representing the hospital medical staff section; (m) *a delegate representing the young physician section; and* (*n*) elected officers, trustees, and speakers of the American Medical Association, provided those individuals are members of the Medical Society of the State of New York.

Fiscal Note: $55,000

The Committee believes that the future of the organized medical profession rests upon its ability to recruit young physicians, and to encourage young physicians to take an active role in shaping policies affecting health care. The Committee does not believe, however, that the creation of a separate section based upon age criteria would be effective or desirable since it could have the unintended effect of separating physicians on the basis of age. Young physicians should be encouraged to participate in the mainstream of organized medicine—and not set aside in a separate category.

The Committee, therefore, unanimously recommends that Resolution 89-51 **NOT BE ADOPTED.**

Resolution 89-57, To Increase the Length of Term of the President and President-Elect of the Medical Society of the State of New York

Introduced by Medical Society of the County of Albany

Whereas, The President of the Medical Society of the State of New York is significant in terms of setting a policy agenda for organized medicine in the State; and

Whereas, As prescribed by Article V, Section 1 of the Bylaws of the Medical Society of the State of New York, the term of the presidency shall not exceed one year in duration; and

Whereas, One year is insufficient time for the president to adequately initiate and enact a policy agenda; therefore be it

Resolved, That Article V, paragraph 1 of the Bylaws of the Medical Society of the State of New York be amended to read as follows (additions italicized):

The officers of the Medical Society of the State of New York shall be a president, a president-elect, a vice-president, a secretary, an assistant secretary, a treasurer, an assistant treasurer, a speaker, and a vice-speaker of the House of Delegates. They shall take office at the termination of the annual meeting at which they are elected and shall serve for one year or until their successors have been duly chosen, *with the exception of the president and president-elect who shall serve for two years.*

Before an individual assumes the responsibilities of the president, the individual has served a full year as president-elect and, usually, has been active for many years in a leadership position in a component county medical society and the State Medical Society. The Committee, therefore, does not believe that Resolution 89-57 is necessary. Any individual who serves as president-elect and then as president must devote two years of his career to the Medical Society. Resolution 89-57 would require individuals to devote two additional years. This may have the effect of deterring qualified individuals from serving.

Accordingly, the Committee unanimously recommends that Resolution 89-57 **NOT BE ADOPTED.**

Resolution 88-9, Mission Statement of the Medical Society of the State of New York

Introduced by the Council

Whereas, The Medical Society of the State of New York has developed a Strategic Plan which reflects the Society's realization that the organization must focus its activities and resources on those areas which are of most importance to its membership; and

Whereas, The Strategic Plan recognized the necessity of updating the mission statement of the Society to be more responsive to the current environment in which medical practice is being conducted, and the commitment of the Society to serve as a strong advocate for physician interests; therefore be it

Resolved, That Article I of the Bylaws of the Medical Society of the State of New York be amended to read as follows (deletions in brackets, additions italicized):

The name and title of the State Society shall be the Medical Society of the State of New York. The purposes of the State Society shall be: [to federate into one organization the medical profession of the State of New York; to extend medical knowledge and advance the science and art of medicine; to establish the Principles of Professional Conduct which shall be binding on the members of the Medical Society of the State of New York, the members of the component county medical societies and the members of the component district branches, in all matters in their relation to each other and to the public; to enhance the delivery of medical care of high quality to all people in the most economical manner; to promote the betterment of community health; and to enlighten and direct public opinion in regard to the problems of medicine and health for the best interests of the people of the State.]

1. to contribute to the professional and personal development of member physicians by representing the profession as a whole and advocating health-related rights, responsibilities and issues. These actions are designed to promote a favorable environment for the practice of medicine and improvement of the health of the residents of New York State.

2. to serve as a resource for its members and assist them in addressing the many issues and needs which they face in providing health care to their patients.

3. to seek to educate the public regarding important health care issues, including identification and discussion of their implications for patients and physicians in the current medical practice environment.

4. to act to promote and maintain high standards in medical education and in the practice of medicine in an effort to ensure that quality medical care is available to the public.

5. to seek to attain these goals by acting, to the fullest extent possible, in concert with organizations and agencies which have similar aims.

This resolution was referred back to the Council last year. In referring the resolution back to the Council, the Council was requested to consider amending the resolution by retaining the statement "to enhance the delivery of medical care of high quality to all people in the most economical manner", which is currently included in Article I of the Bylaws. It was requested that the Council consolidate the above statement with the statement reading, "to act to promote and maintain high standards in medical education and in the practice of medicine in an effort to ensure that quality medical care is available to the public." The Council was also requested to consider amending the resolution by the statement "to federate into one organization the medical profession of the State of New York."

The Council acted upon these recommendations and referred the Resolution to the House Committee on Bylaws for further evaluation. As acted upon by the Council and House Committee on Bylaws, Resolution 88-9, as amended, now reads:

Whereas, The Medical Society of the State of New York has developed a Strategic Plan which reflects the Society's realization that the organization must focus its activities and resources on those areas which are of most importance to its membership; and

Whereas, The Strategic Plan recognized the necessity of updating the mission statement of the Society to be more responsive to the current environment in which medical practice is being conducted, and the commitment of the Society to serve as a strong advocate for physician interests; therefore be it

Resolved, That Article I of the Bylaws of the Medical Society of the State of New York be amended to read as follows (deletions in brackets, additions italicized):

The name and title [of the State Society] shall be the Medical Society of the State of New York. The purposes of the [State Society] *Medical Society of the State of New York* shall be: [to federate into one organization the medical profession of the State of New York; to extend medical knowledge and advance the science and art of medicine; to establish the Principles of Professional Conduct which shall be binding on the members of the Medical Society of the State of New York, the members of the component county medical societies and the members of the component district branches, in all matters in their relation to each other and to the public; to enhance the delivery of medical care of high quality to all people in the most economical manner; to promote the betterment of community health; and to enlighten and direct public opinion in regard to the problems of medicine and health for the best interests of the people of the State.]

1. to federate into one organization the medical profession of the State of New York.

2. to contribute to the professional and personal development of member physicians by representing the profession as a whole and advocating health-related rights, responsibilities and issues. These actions are designed to promote a favorable environment for the practice of medicine and improvement of the health of the residents of New York State.

3. to serve as a resource for its members and assist them in addressing the many issues and needs which they face in providing health care to their patients.

4. to enhance the delivery of medical care of high quality to all people in the most economical manner, and to act to promote and maintain high standards in medical education and in the practice of medicine in an effort to ensure that quality medical care is available to the public.

5. to seek to educate the public regarding important health care issues, including identification and discussion of their implications for patients and physicians in the current medical practice environment.

6. to seek to attain these goals by acting, to the fullest extent possible, in concert with organizations and agencies which have similar aims.

The Committee unanimously recommends that Resolution 88-9 be **ADOPTED AS AMENDED.**

Your Chairman would like to thank, in addition to the members of the Committee and the physicians who testified before it, those members of staff who helped this Committee; Donald R. Moy, Esq., Jay Silverman, Esq. and Kathleen Wellborn for her secretarial assistance.

Respectfully submitted,
George Lim, M.D., *Chairman*

ACQUIRED IMMUNODEFICIENCY SYNDROME

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Edited by Pascal James Imperato, MD

With a foreword by
George J. Lawrence, Jr., MD, Chairman
AIDS Task Force
Medical Society of the State of New York

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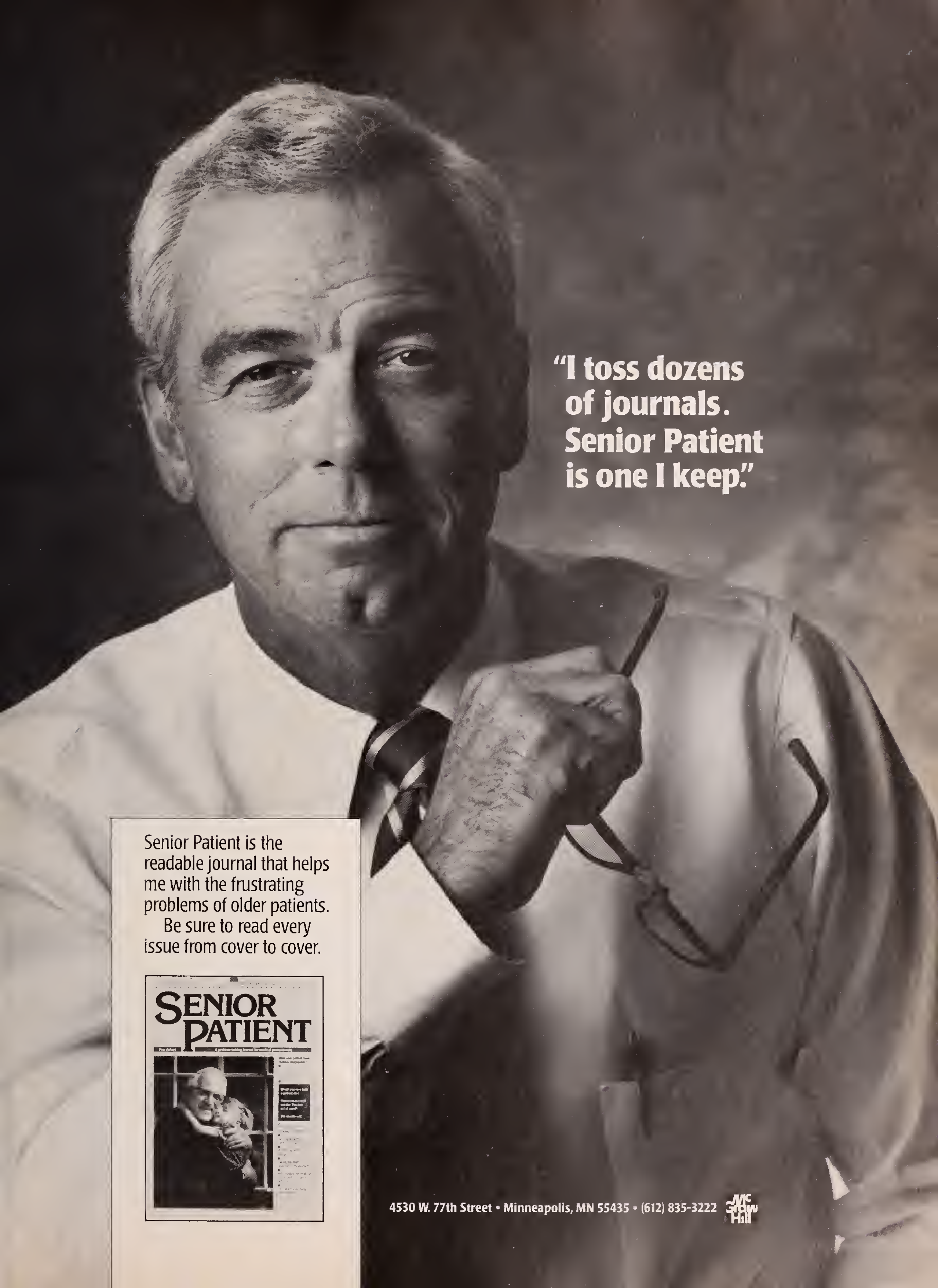

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References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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its. NYU Medical Center. *Mar 25–30.* **Anesthesiology Comprehensive Review X.** 44 Cat I Credits. Waldorf Astoria Hotel. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Mar 2. **Current Concepts in Asthma, Allergy and Pulmonary Disease.** Beth Israel Medical Center. Contact: Nora Pisano, Medical Education, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003. Tel: (212) 420-2000.

Mar 2–3. **The Masters of Ophthalmic Plastic Surgery-Lacrimal Eyelid and Orbital Surgery.** Manhattan Eye, Ear & Throat Hospital. *March 31.* **The New York Society for Clinical Ophthalmology Spring Meeting.** Waldorf Astoria. Contact: Francine Leinhardt, Course Coordinator, Manhattan Eye, Ear & Throat Hospital, 210 E 64th St, New York, NY 10021. Tel: (212) 838-9200, ext 2776.

Mar 13–15. **Sixth Cooley's Anemia Symposium.** Sheraton Centre. Contact: Conference Director, The New York Academy of Sciences, 2 E 63rd St, New York, NY 10021. Tel: (212) 838-0230.

Mar 15–17. **Common Problems in Obstetrics and Gynecology.** 16 Cat I Credits. New York. Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024. Tel: (202) 628-5577.

Mar 22–25. **17th Annual Conference and Medical Research Symposium on Dyslexia.** Penta Hotel. Contact: New York Branch of the Orton Dyslexia Society, 80 Fifth Ave, New York, NY 10011. Tel: (212) 691-1930.

Mar 24. **Modern Management of Malignant Melanoma.** Mt Sinai School of Medicine. Contact: Virginia Harrison, Post-Graduate School, Box 1193, Mt Sinai School of Medicine, 1 Gustave L. Levy Pl, New York, NY 10021. Tel: (212) 241-6737.

Mar 24–25. **Cochlear Implants in Children: Rehabilitation Techniques.** 13½ Cat I Credits. NYU Medical Center. *Mar 26–30.* **Seminar in Advanced Rheumatology.** *March 26–30.* **Temporal Bone Dissection.** NYU Medical Cen-

(*continued on p 16A*)

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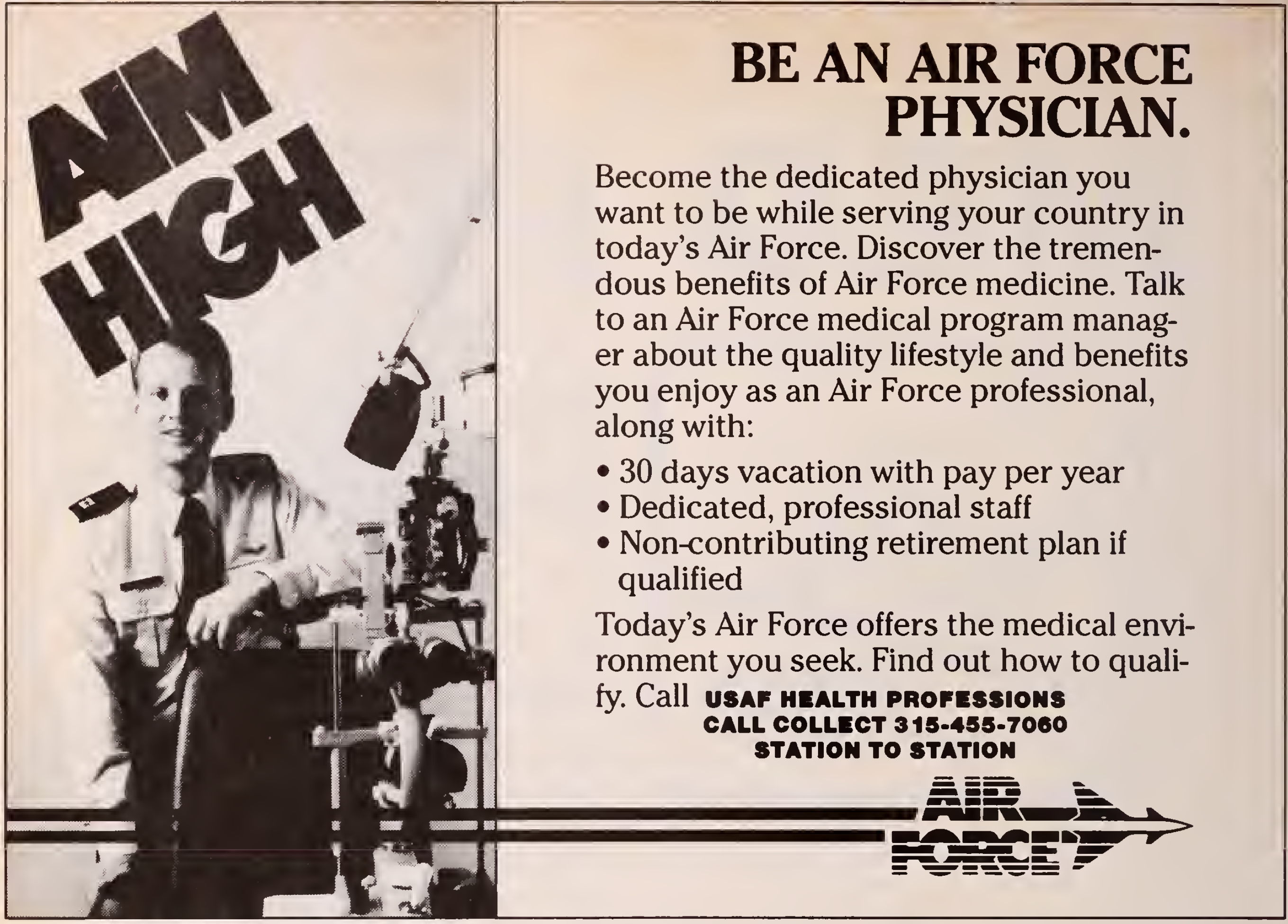

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(*continued from p 12A*)

ter. Contact: NYU Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

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CALIFORNIA

Mar 2–4. **Advanced Seminar in Diagnostic Imaging.** 18 Cat I Credits. Ritz-Carlton Resort Hotel, Laguna Niguel. *Mar 5–9.* **Fifth Annual San Diego Postgraduate Magnetic Resonance Imaging.** Hotel del Coronado, Coronado (San Diego). Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

Mar 4–9. **11th Annual Mammoth Mountain Emergency Medicine Ski Conference.** 18 Cat I Credits. Mammoth Lakes. Contact: Medical Conferences, Inc, PO Box 52-B, Newport Beach, CA 92662. Tel: (714) 650-4156.

Mar 16–18. **Ob/Gyn and Abdominal Sonography: Update 1990.** Hyatt Regency, San Francisco. *Mar 19–23.* **Diagnostic Radiology.** Fairmont Hotel, San Francisco. Contact: Radiology Postgraduate Education, University of California, Room C324, San Francisco, CA 94143-0628. Tel: (415) 476-5731.

COLORADO

Mar 4–9. **Tenth Annual Keystone ENT Ski Conference.** Keystone. Contact: Brenda C. Ram, Continuing Education, University of Nebraska Medical Center, 42nd & Dewey Ave, Omaha, NE 68105. Tel: (402) 559-4152.

DISTRICT OF COLUMBIA

Mar 17–18. **Hyperbaric Center Awareness Course.** Washington, DC. Contact: Armed Forces Institute of Pathology, 14th St and Alaska Ave, NW, Washington, DC 20306-6000. Tel: (202) 576-2868.

HAWAII

Mar 26–30. **Diagnostic Imaging: 1990.** Kauai. Contact: Radiology Postgraduate Education, University of California, Room C324, San Francisco, CA 94143-0628. Tel: (415) 476-5731.

NEBRASKA

Mar 26–Apr 6. **Family Practice Review.** Omaha. Contact: Brenda C. Ram, Continuing Education, University of Nebraska Medical Center, 42nd & Dewey Ave, Omaha, NE 68105. Tel: (402) 559-4152.

NEVADA

Mar 1–3. **Advanced Colposcopy.** 16 Cat I Credits. Las Vegas. Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024. Tel: (202) 628-5577.

OKLAHOMA

Mar 4–9. **17th Annual Critical Care Medicine Course.** Holiday Inn Airport West, Oklahoma City. Contact: Critical Care Medicine Course, University of Oklahoma Health Sciences Center, Room 3 SP 400, PO Box 26901, Oklahoma City, OK 73190.

PENNSYLVANIA

Mar 23–25. **Medical Acupuncture 1990. Applications in Pain Management and Substance Abuse.** Hershey Philadelphia Hotel, Philadelphia. Contact: Medical Acupuncture 1990, 5275 Lee Highway #101, Arlington, VA 22207. Tel: (703) 237-3105.

VERMONT

Mar 14–16. **Primary Care Rheumatology for the '90's.** 9 Cat I Credits. Sugarbush Inn/Warren. Contact: Barbara Post, Sunnyview Hospital and Rehabilitation Center, 1270 Belmont Ave, Schenectady, NY 12308. Tel: (518) 382-4595.

AROUND THE WORLD

PORTUGAL

Mar 4–10. **19th Annual Pediatric Postgraduate Update.** 20 Cat I Credits. Lisbon. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Schneider Children's Hospital of Long Island Jewish Medical Center, New Hyde Park, NY 10042. Tel: (718) 470-8650. □

Index to Advertisers

Blanksteen Companies 15A
CIGNA 5A
Classified Advertising 6A, 10A, 14A, 15A
Eli Lilly and Company .... 2nd Cover
Marine Midland Bank 7A
McGraw Hill Publications 8A, 11A
Med Plus 10A
Med Pro 13A
Palisades Pharmaceuticals 12A
Roche Laboratories .... 3A, 4A, 3rd & 4th Cover
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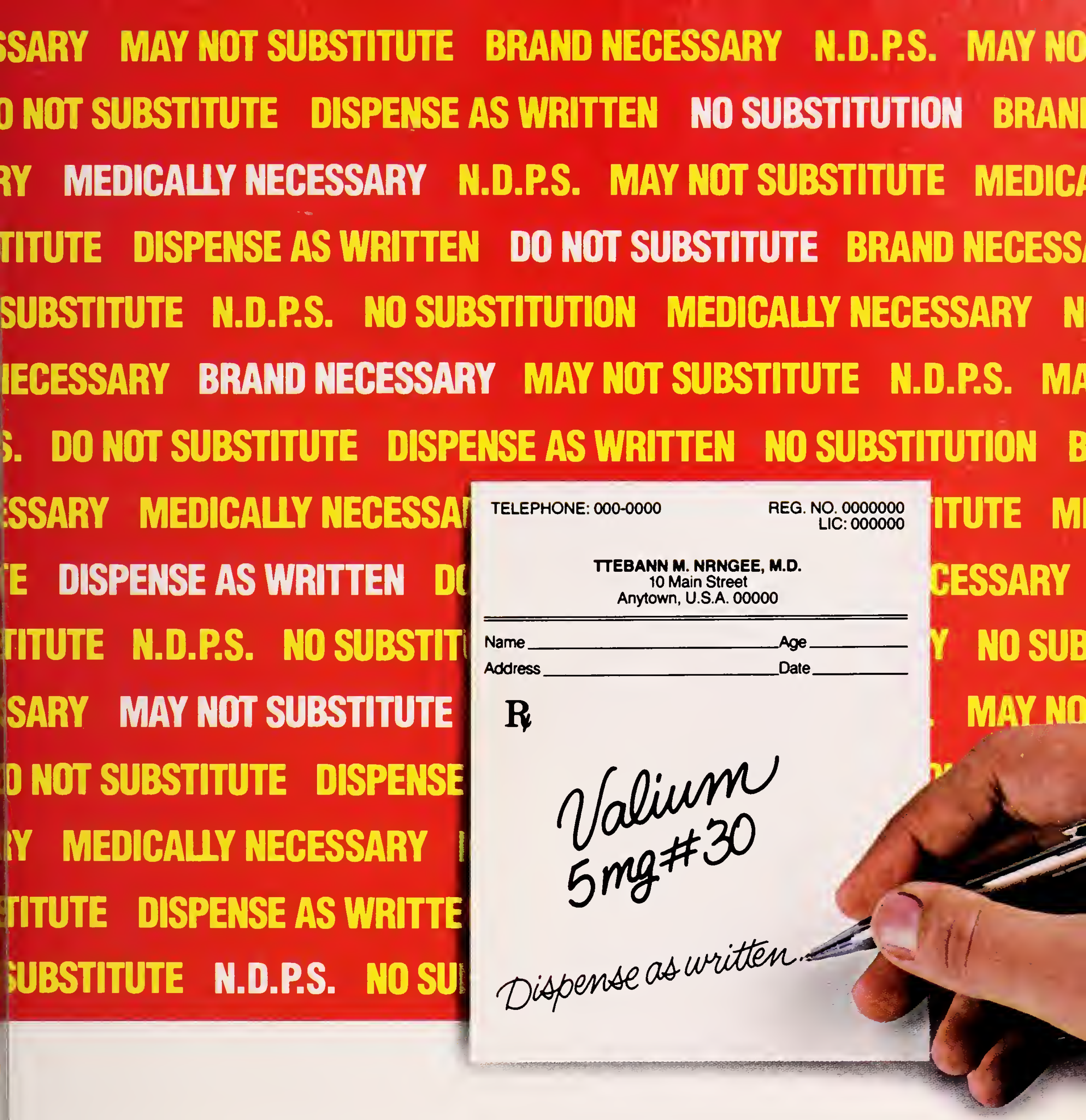
U.S. Army Reserves 1A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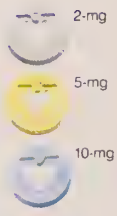

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ACQUIRED IMMUNODEFICIENCY SYNDROME

COMMENTARIES

Acquired immunodeficiency syndrome: The agenda for the 1990s 115
PASCAL JAMES IMPERATO, MD

Expanding the focus of human immunodeficiency virus prevention in the 1990s 116
NICHOLAS A. RANGO, MD, MICHAEL RAMPOLLA

The increasing role of primary care in the management of HIV-infected patients 119
JACK A. DeHOVITZ, MD

Dissemination of *Pneumocystis* 121
VERNON A. PILON, MD

RESEARCH PAPERS

Changes in AIDS risk behavior among intravenous drug abusers in New York City 123
RANDALL E. HARRIS, PhD, MD; JOHN LANGROD, PhD; JAMES R. HEBERT, ScD; JOYCE LOWINSON, MD; EDITH ZANG, PhD; ERNST L. WYNDER, MD

Assessment of AIDS knowledge in selected New York State sexually transmitted disease clinics 126
HWA-GAN H. CHANG, MA; DENNIS MURPHY, MA; GEORGE T. DIFERDINANDO, JR, MD, MPH; DALE L. MORSE, MD

AIDS education: Evaluation of school and worksite based presentations 129
AVIMA M. RUDER, PhD; ROBIN FLAM, DrPH; DAVID FLATTO, CSW; ANITA S. CURRAN, MD, MPH

AIDS behind bars: Epidemiology of New York State prison inmate cases, 1980–1988 133
DALE L. MORSE, MD; BENEDICT I. TRUMAN, MD, MPH; JOHN P. HANRAHAN, MD, MPH; JAROMIR MIKL, MPH; RAYMOND K. BROADDUS, PhD; BARBARA H. MAGUIRE, RN; JOHN C. GRABAU, PhD, MPH; SUE KAIN-HYDE; YANGSOOK HAN; CHARLES E. LAWRENCE, PhD

A seroprevalence survey for human immunodeficiency virus antibody in mentally retarded adults 139
SUSAN H. PINCUS, MD; ELLIE E. SCHOENBAUM, MD; MAYRIS WEBBER, MPH

REVIEW ARTICLE

Lessons from New York City on HIV/AIDS in adolescents 143
KAREN HEIN, MD

THE CPC

Thoracic lymphadenopathy and disseminated *Pneumocystis carinii* in AIDS 146

SPECIAL ARTICLE

A medical student's dilemma 154
BARRY A. SOLOMON

CASE REPORTS

Disseminated *Pneumocystis carinii* infection in the acquired immunodeficiency syndrome 155
STEFANO RAVALLI, MD; ROGER L. GARCIA, MD; RAF A. VINCENT, MD; RUVAN SHEIN, MD

Erosive arthritis, atypical psoriasis, and *Pneumocystis carinii* pneumonia in an HIV-positive man 157
KENNETH M. STEIN, MD; GEORGE A. MENSAH, MD; R. GORDON DOUGLAS, JR, MD

Deep venous thrombosis in patients with AIDS 159
JON R. COHEN, MD; RUDY LACKNER, MD; PETER WENIG, MD; GEORGE PILLARI, MD

LETTERS TO THE EDITOR

Resolution of cavitary *Pneumocystis carinii* pneumonia 162
RICK CONETTA, MD, RAJINDER K. CHITKARA, MD

Multifocal human papillomavirus infections of the genital tract in HIV seropositive women 162
PATRICK CAUBEL, MD; HENRI FOULQUES, MD; CHRISTINE KATLAMA, MD

Physicians needed to work with AIDS patients 163
DONALD N GROFF, MD

AIDS legislation needed 163
RAYMOND A. SMEGO, JR, MD, MPH; ELIZABETH A BARRANCO, MD

LEADS FROM EPIDEMIOLOGY NOTES 164
NEWS BRIEFS 168
GUIDELINES FOR AUTHORS 170
MEDICAL MEETINGS AND LECTURES 4A

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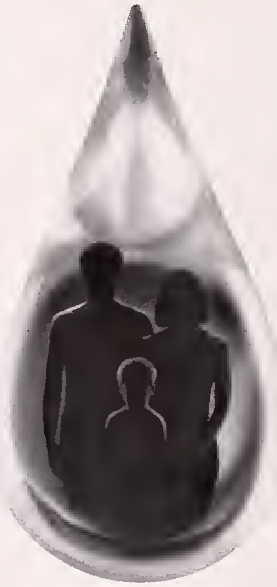

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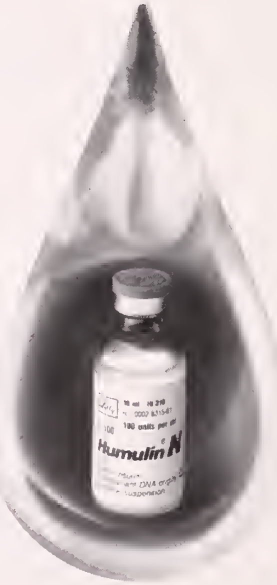

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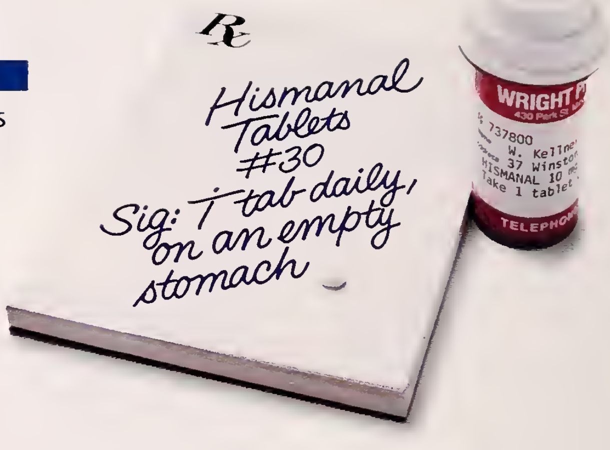

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DESCRIPTION
HISMANAL® (astemizole) is a histamine H_1-receptor antagonist available in scored white tablets for oral use.

CONTRAINDICATIONS
HISMANAL is contraindicated in patients with known hypersensitivity to astemizole or any of the inactive ingredients.

PRECAUTIONS
General: Caution should be given to potential anticholinergic (drying) effects in patients with lower airway diseases, including asthma.

Caution should be used in patients with cirrhosis or other liver diseases (See Clinical Pharmacology section).

HISMANAL does not appear to be dialyzable.

Caution should also be used when treating patients with renal impairment.

Information for Patients: Patients taking HISMANAL should receive the following information and instructions. Antihistamines are prescribed to reduce allergic symptoms. Patients should be questioned about pregnancy or lactation before starting HISMANAL therapy, since the drug should be used in pregnancy or lactation only if the potential benefit justifies the potential risk to fetus or baby (see Pregnancy subsection). Patients should be instructed 1) to take HISMANAL only as needed, 2) not to exceed the prescribed dose, and 3) to take HISMANAL on an empty stomach, e.g., at least 2 hours after a meal. No additional food should be taken for at least 1 hour post-dosing. Patients should also be instructed to store this medication in a tightly closed container in a cool, dry place, away from heat or direct sunlight, and away from children.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Carcinogenic potential has not been revealed in rats given 260x the recommended human dose of astemizole for 24 months, or in mice given 400x the recommended human dose for 18 months. Micronucleus, dominant lethal, sister chromatid exchange and Ames tests of astemizole have not revealed mutagenic activity.

Impairment of fertility was not observed in male or female rats given 200x the recommended human dose.

Pregnancy: Pregnancy Category C: Teratogenic effects were not observed in rats administered 200x the recommended human dose or in rabbits given 200x the recommended human dose. Maternal toxicity was seen in rabbits administered 200x the recommended human dose. Embryocidal effects accompanied by maternal toxicity were observed at 100x the recommended human dose in rats. Embryotoxicity or maternal toxicity was not observed in rats or rabbits administered 50x the recommended human dose. There are no adequate and well controlled studies in pregnant women. HISMANAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. Metabolites may remain in the body for as long as 4 months after the end of dosing, calculated on the basis of 6 times the terminal half-life (See Clinical Pharmacology section).

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because certain drugs are known to be excreted in human milk, caution should be exercised when HISMANAL is administered to a nursing woman. HISMANAL is excreted in the milk of dogs.

Pediatric Use: Safety and efficacy in children under 12 years of age has not been demonstrated.

ADVERSE REACTIONS
The reported incidences of adverse reactions listed in the following table are derived from controlled clinical studies in adults. In these studies the usual maintenance dose of HISMANAL® (astemizole) was 10 mg once daily

	Percent of Patients Reporting		
	Controlled Studies*		
ADVERSE EVENT	Hismanal (N = 1630) %	Placebo (N = 1109) %	Classical** (N = 304) %
Central Nervous System			
Drowsiness	7.1	6.4	22.0
Headache	6.7	9.2	3.3
Fatigue	4.2	1.6	11.8
Appetite increase	3.9	1.4	0.0
Weight increase	3.6	0.7	1.0
Nervousness	2.1	1.2	0.3
Dizzy	2.0	1.8	1.0
Gastrointestinal System			
Nausea	2.5	2.9	1.3
Diarrhea	1.8	2.0	0.7
Abdominal pain	1.4	1.2	0.7
Eye, Ear, Nose, and Throat			
Mouth dry	5.2	3.8	7.9
Pharyngitis	1.7	2.3	0.3
Conjunctivitis	1.2	1.2	0.7
Other			
Arthralgia	1.2	1.6	0.0

*Duration of treatment in Controlled Studies ranged from 7 to 182 Days

**Classical Drugs: Clemastine (N = 137); Chlorpheniramine (N = 100); Pheniramine Maleate (N = 47); d-Chlorpheniramine (N = 20)

Adverse reaction information has been obtained from more than 7500 patients in all clinical trials. Weight gain has been reported in 3.6% of astemizole treated patients involved in controlled studies, with an average treatment duration of 53 days. In 46 of the 59 patients for whom actual weight gain data was available, the average weight gain was 3.2 kg.

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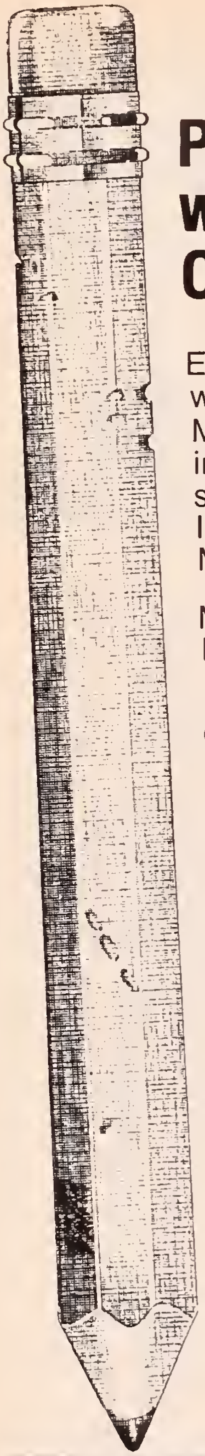

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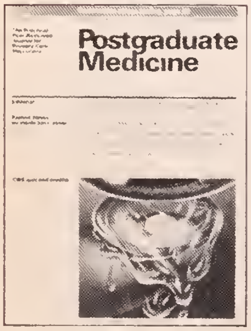

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APRIL 1990

AROUND THE STATE

ALBANY

Apr 27. **1990 Sports Medicine Update.** Albany Marriott Hotel. Contact: Diane M. Martin, Conference Coordinator, Albany Medical College, Office of CME, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

MANHATTAN

Apr 2–7. **Comprehensive Review of Physical Medicine and Rehabilitation.** 55 Cat I Credits. New York University Medical Center. *Apr 3–6*. **Post Graduate Course in Neuroradiology Including Head and Neck.** 28 Cat I Credits. Grand Hyatt Hotel. *Apr 6–8*. **Post-Graduate Course in Bone and Joint Radiology.** 16 Cat I Credits. Grand Hyatt Hotel. *Apr 26–28*. **Advances in Dermatology.** 17 Cat I Credits. New York University Medical Center. *Apr 28*. **Fluid Resuscitation of the Critically Ill Patient.** $6\frac{1}{2}$ Cat I Credits. New York University Medical Center. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

(*continued on p 10A*)

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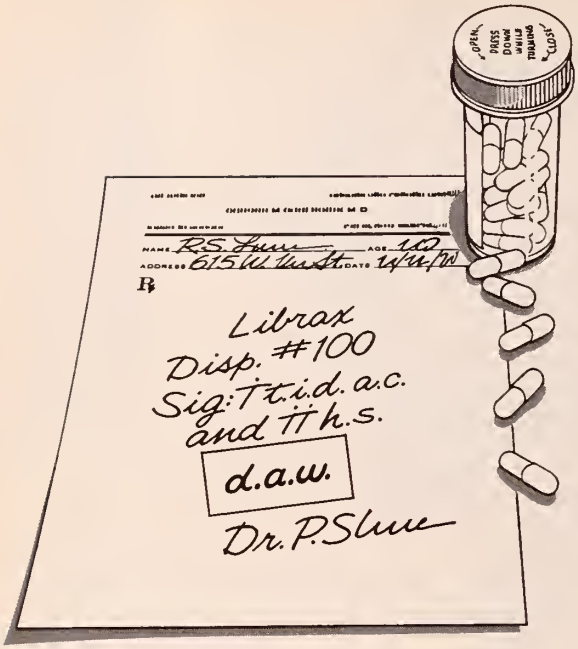

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Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.

As with all anticholinergics, inhibition of lactation may occur.

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

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Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. After extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

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(*continued from p 4A*)

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Apr 5–7. **AIDS, HPV, and Other Viral Infections.** 16 Cat I Credits. Washington. Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024. Tel: (202) 638-5577.

FLORIDA

Apr 2–4. **Contemporary Colorectal Surgery.** 11 Cat I Credits. West Palm Beach. Contact: Laura J. Lyons, CME Coordinator, Good Samaritan Hospital, PO Box 3166, West Palm Beach, FL 33402. Tel: (407) 650-6236.

Apr 27–30. **The Spine: Current Concepts of Diagnosis and Treatment.** Hyatt Regency Grand Cypress, Orlando. Contact: The Spine Study Group, Pat Curl, Conference Coordinator, PO Box 33185, San Diego, CA 92103. Tel: (619) 692-9115.

HAWAII

Apr 7–14. **Cardiopulmonary Emergencies.** Waiohai & Poipu Beach Hotels, Kauai. Contact: Edith S. Bookstein, Conference Management Associates, PO Box 2586, La Jolla, CA 92038. Tel: (619) 454-3212.

MISSOURI

Apr 5–6. **17th Annual Symposium on Obstetrics and Gynecology.** Washington University Medical Center, St Louis. Contact: Cathy Caruso, Office of Continuing Medical Education, Washington University School of Medicine, 660 S Euclid, Box 8063, St Louis, MO 63110. Tel: (314) 362-6893.

TEXAS

Apr 18–22. **Sixth Annual Family Medicine Review.** 40 Cat I Credits. Stouffer Hotel, Austin. *Apr 23–24.* **Advanced Cardiac Life Support (ACLS) Provider Course.** 13 Cat I Credits. Temple. Contact: Department of Continuing Medical Education, Scott and White, 2401 South 31st Street, Temple, TX 76508. Tel: (817) 774-4073.

(*continued on p 12A*)

NEW YORK STATE
JOURNAL OF MEDICINE

March 1990 • Volume 90, Number 3

COMMENTARIES

Acquired immunodeficiency syndrome: The agenda for the 1990s

The acquired immunodeficiency syndrome (AIDS) epidemic is now in its tenth documented year in the United States. During the last decade, the disease and its causative agent, the human immunodeficiency virus (HIV), have been closely studied, resulting in an inexorable advance in knowledge. The epidemiology of AIDS fully emerged during the 1980s. It is largely characterized now by person-to-person spread through sexual contact and through the sharing of contaminated intravenous (IV) injection equipment and by transplacental transmission. Transmission via contaminated blood and blood products, once an important mode of transmission earlier in the epidemic, is still a concern. However, more effective screening efforts have greatly reduced its importance.

The transplacental spread of AIDS to the children of female IV drug abusers and the female sexual partners of male IV drug abusers will continue to be a serious problem in the 1990s. This is especially true in inner city areas among population groups that so far have been refractory to health education efforts aimed at prevention. The desire of male IV drug abusers and their female sexual partners, who may or may not be IV drug abusers themselves, to have children poses a significant obstacle to implementing effective strategies for preventing transplacental transmission. Needle exchange programs have so far not proven to be a viable approach for reducing primary transmission among IV drug abusers and subsequent transplacental transmission.

Changes in current substance abuse patterns and the modes of administering drugs could have a future impact on the incidence and prevalence of transplacental spread in the 1990s. However, it is more likely that new cases of transplacental AIDS in children will continue to occur with significant frequency during the 1990s, particularly among inner city parturients.

During the 1990s, greater focus will be required on certain groups of teenagers who, because of early age of first intercourse, choice of sexual partners, and failure to use appropriate preventive measures, are at high risk for acquiring HIV infections. Intense public health activities for these teenagers should be high on the agenda for the 1990s.

Public health education concerning the risks of transmission through male homosexual sexual contacts have been partially successful, as evidenced by current national statistics. However, continued public health efforts aimed at preventing HIV transmission among homosexual and bisexual men will be required in the next decade.

During the 1990s, the concept of AIDS as a chronic disease will become more widely accepted. Advances in the prevention and management of opportunistic infections not only among those patients with clinical AIDS, but also among those with earlier stages of HIV infection, translate into greater longevity for many patients. These patients, as well as those who benefit from antiviral agents such as zidovudine, will increasingly require much longer periods of ongoing care than was the case for AIDS patients in the early 1980s. Acceptance of AIDS as a chronic disease will require some shifts in our current medical and social approaches. Primary care physicians will have to assume an increasingly larger role in following HIV infected patients, monitoring them for progression of their disease and initiating appropriate therapeutic interventions, as required.[1] Laboratory tests such as the CD4 (T4) lymphocyte count and/or the CD4 percent as well as the HIV p24 antigen and beta 2-microglobulin are now being used as effective markers to gauge the progress of HIV infection.[2] The wide availability of these tests and the knowledge that zidovudine use at certain CD4 levels can retard progression of the disease speak not only for a great advance in the management of HIV infection, but also for a role for primary care physicians. Such physicians should also assume a critical role in the ambulatory prevention of *Pneumocystis carinii* pneumonia (PCP), which eventually infects 80% of those with AIDS.[3] This infection can now be effectively prevented in a large percentage of AIDS patients through the use of aerosolized pentamidine and trimethoprim-sulfmethoxazole. If primary care physicians assume a greater responsibility for the management

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The opinions expressed in this editorial are those of the author alone and do not constitute an expression of official opinion of the Medical Society of the State of New York.

of HIV-infected patients, then the 1990s may witness a marked shift in care from the inpatient to the ambulatory setting with resulting social and cost benefits.

It is well known that residency training programs situated in medical centers where the high proportion of the beds are occupied by AIDS patients have difficulty attracting candidates.[4] Centers of this kind in geographic areas where the incidence of AIDS is high will continue to have difficulty in attracting residents in the absence of innovative initiatives. Formulating these initiatives will not be an easy task. However, in their absence, the present inability to recruit quality house staff will only worsen.

Many of the social, health care, and ethical issues that were at the center of concern during the 1980s will remain in place throughout the 1990s. Aggregate inpatient care costs for AIDS patients will continue to rise if current medical care approaches prevail. Likewise, the presence of large numbers of AIDS patients on inpatient services will continue to add to the scarcity of general medical beds, particularly in places such as New York City. A greater shift to the ambulatory and hospice approach could ameliorate this problem.

The 1990s receive an AIDS legacy from the 1980s of unresolved and highly visible social, public health, ethical, and medical care issues. However, this legacy also includes much progress in the prevention and clinical management of HIV infection, the identification of the human immunodeficiency virus, and basic science efforts that may eventually lead to the development of vaccines and more effective therapeutic agents. It is on these less visible basic science efforts that society has pinned its hopes for the 1990s. The development and subsequent widespread use of protective vaccines and/or curative antiviral agents would dramatically alter the AIDS epidemic and all of the medical care and social structures that have been elaborated during the past decade to deal with it. The likelihood of these breakthroughs occurring in the early 1990s seems remote at present. However, scientific progress on these fronts is steady and continuous, raising the possibility that these breakthroughs may become a reality at the end of this decade. Meanwhile, the medical profession and society as a whole must achieve the best with the limited modalities at hand. Later in this decade, these may appear in retrospect as cumbersome and ineffective, much as early efforts to manage tuberculosis do in the era of antituberculosis drugs.

The problems posed by AIDS, and medicine's success in dealing with some of them, have been well documented in the *Journal* over the past several years. Since 1987, four thematic issues of the *Journal* have been devoted to AIDS (May 1987, May 1988, December 1988, and this current issue) and the contents of the first two issues have been published in book form.[5] This current issue chronicles not only the complexity and scope of the AIDS epidemic, but also its ever-unfolding nature. It is this latter characteristic that introduces some uncertainty into the nature of the AIDS agenda for the 1990s.

As the decade opens, it is obvious that many familiar issues from the 1980s will prominently figure in the 1990s AIDS agenda. Foremost among these are a commitment to develop effective vaccines and better therapeutic agents, and to strengthen preventive efforts begun in the previous decade. However, because of the epidemic's unfolding nature, newer and even surprising items will be added to the agenda as the decade matures.

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Expanding the focus of human immunodeficiency virus prevention in the 1990s

The conceptualization and implementation of an effective human immunodeficiency virus (HIV) prevention strategy for New York State rely on epidemiologic data to identify populations and areas at increased risk and to describe trends. Until mid-1985, when HIV antibody testing became widely available and provided an additional, and more timely, source of intelligence, acquired immunodeficiency syndrome (AIDS) case reporting provided virtually the sole basis for defining the AIDS epidemic in New York State.

From the outset of the epidemic through mid-1985, approximately 62% of the state's nearly 4,500 AIDS cases involved homosexual or bisexual men.[1] During this period, only approximately 35% of all reported cases were among intravenous (IV) drug users. Approximately 400 women had been reported with AIDS, with only 15% of them classified as having no risk factors other than sexual contact with an at-risk male.[1]

During the second half of the 1980s, the epidemic took a much heavier toll. An additional 21,000 New Yorkers were diagnosed with Centers for Disease Control (CDC)-defined AIDS.[2] Homosexual and bisexual men continued to bear the brunt, with the average monthly AIDS case count among this risk behavior group increasing during the period. Yet the increase in the number of reported AIDS cases among intravenous drug users, their heterosexual contacts, and their offspring was even more dramatic. By the fall of 1987, the number of newly reported cases among intravenous drug users had exceeded that among homosexuals/bisexuals and became the most common category of reported risk.[3(p54)] Intravenous drug use has remained the predominant risk factor. This significant expansion and acceleration of AIDS diagnoses through the second half of the 1980s signalled an equally important shift in the demographics of AIDS. By the end of the 1980s, fewer new cases of AIDS were being reported among whites in New York City than among either blacks or Hispanics. It was not that AIDS among whites or homosexual men had declined, it was simply that the rate of increase among persons of color and among drug users was advancing on a much steeper upward slope. Furthermore, knowledge is increasing about the ways in which the clinical manifestations of HIV infection among drug users differ from those among gay men. We are becoming more acutely aware that these trends in CDC-defined AIDS represent a significant underestimation of HIV-related illness among IV drug users.

More than three thousand women have been reported with AIDS since mid-1985 and the proportion of them who were reportedly infected by heterosexual contact with an at-risk male increased by 50% during the period. Of the more than 600 pediatric AIDS cases reported in the 1980s, the vast majority were diagnosed in the latter half of the decade. Most were born to intravenous drug using women or to partners of drug users; and more than 90% were black or Hispanic.[1,2]

The surveillance of HIV in New York State has been conducted through blinded research studies and through an accumulation of data from confidential antibody testing activity. The New York State Department of Health (DOH) has been conducting studies in settings such as sexually transmitted disease clinics, sentinel family planning clinics, drug treatment sites, state prison intake facilities, and other locations. The most broadly based study has been ongoing since December 1987 and has involved the anonymous testing of heel-stick blood collected from virtually every baby born in New York State. HIV test data from this study have provided information about the infection status of women giving birth.

During the first year of the study, more than 276,000 babies were tested. Statewide, 0.66% of the newborns, or one in 150, tested HIV positive. The seropositivity rate among Hispanic babies has been ten times greater than among whites and the seropositivity rate among black babies has been 14 times greater than among whites. Stated differently, one in 770 white babies was born to an infected mother, while one in 75 Hispanic babies and one in 55 black babies were born to an HIV infected mother in New York State. Analysis of the residential zip codes of HIV infected women giving birth in New York City revealed a correlation between the geographic distribution of drug use, as measured by drug-related hospitalizations, and HIV prevalence.[3(p10)]

An anonymous study of clients entering 28 methadone maintenance treatment clinics in New York City indicated a 45.9% HIV positivity rate.[3(p18)] An anonymous study of male prisoners entering the state prison system through the Downstate Correctional Facility revealed an HIV positivity rate of 17.4%; of those entrants with a history of intravenous drug use, 44.1% were seropositive.[3(p20)] Similar results were observed in an anonymous study of the HIV status of female prisoners entering the Bedford Hills Correctional Facility. Of those tested, 18.8% were positive; of those entrants with a history of intravenous drug use, 45.6% were HIV seropositive.[3(p22)] The newborn, prison, and drug treatment HIV seroprevalence studies have confirmed that drug use is the driving force behind the HIV epidemic within the heterosexual population.

It is estimated that 200,000 to 400,000 New Yorkers may currently be infected with HIV. If the current trend of AIDS incidence continues, an additional 60,000 New Yorkers, more than half intravenous drug users, will be diagnosed during the first five years of the 1990s. An increasing portion of newly diagnosed cases will be black or Hispanic.

All sexually active New Yorkers, and those who share drug injection paraphernalia, are at risk for HIV. Widespread awareness of this reality leading to behavior modification is the foundation of HIV prevention. However, the epidemiologic evidence clearly indicates that HIV is far from evenly distributed throughout the state, either geographically or demographically. In some areas of New York City, as many as one in 33 women giving birth is infected.[3(p11)] Outside of the city, newborn seroprevalence data indicates a clustering of a majority of positive cases in a relatively small number of communities. Thus, although all sexually active New Yorkers are at risk, gradations of risk apply not only to risk behavior, but also to geography. As a result, HIV prevention efforts are appropriately both statewide in scope and intensified in communities which are at demonstrably higher risk.

Although risk categorization by personal behavior or residential community can serve as a valuable targeting tool, it can also engender denial and inaction by individuals, clinicians, public health officials, and society at large, or, worse, prejudice against those at risk for HIV can become the implicit basis for rationalizing a feeble public policy response to the epidemic. It is important for HIV prevention in the 1990s that clinical practice as well as community-wide behavioral norms reflect awareness that, although HIV is not evenly distributed throughout the state, HIV risk is ubiquitous. There is no biologic or epidemiologic basis for assuming that HIV cannot be introduced in any community or, once introduced, that it will not spread. Complacency in areas of relatively low seroprevalence, therefore, must be dispelled. Conversely, prevention efforts in high seroprevalence areas must confront a different sort of complacency, one born of a sense of defeatism.

In many affected communities in the state, HIV is but the latest in a series of "environmental insults." Areas heavily affected by drug use-associated HIV are likely also to be coinfected with a high prevalence of low birthweight babies, infant mortality, teenage pregnancy, syphilis, and other maladies which have been resistant to public health intervention. Within these communities, beneath the clinical symptoms of HIV disease, lies a social morbidity characterized by low educational attainment, poverty, inadequate housing, dysfunctional families, and violence, all of which indirectly feed the epidemic. HIV prevention forces us to attend to these social sores. However, our efforts should not be vitiated by a false sense that transmission cannot be curbed in these communities unless underlying structural problems are eliminated.

Although drug use is now the major force driving the AIDS epidemic, the epidemiology of drug use itself is changing in ways likely to further influence the spread of HIV infection. The recent epidemic spread of syphilis, the doubling of the number of reported cases statewide between 1986 and 1988, has been associated with the emergence of "crack" cocaine.[3(p74)] Although not injected, crack can facilitate HIV transmission, as it has syphilis, by its connection with sexual disinhibition and prostitution.

Despite the established causal connection between drug use and a host of undesirable social sequellae, we lack a basic understanding of the social context of drug use. Compounding our ignorance of the sociology of drug abuse is the paucity of proven therapies aimed at curtailing its use. From the start, we must acknowledge that treatment opportunities are in relatively short supply. Moreover, methadone, the primary treatment for heroin addiction, often serves as a supplemental, rather than a substitute drug. How this situation has come about remains the subject of controversy. No effective therapy has yet been identified for crack users. Unfortunately, HIV prevention in the 1990s will be weakened by such limitations in current drug abuse prevention and treatment efforts.

On a more positive note, the campaign to inform the public about modes of transmission of HIV and approaches to prevention has generally been effective. A state health department developed survey, conducted statewide in 1988 of more than 4,000 adults, demonstrated that more than 90% were aware of the risk of transmission of HIV by sexual intercourse and the sharing of drug works. A similar percentage also understood that the use of condoms can reduce transmission risk.[4] Although such knowledge is not uniform in every age, geographic, or socioeconomic group, the primary barrier to prevention no longer appears to be inadequate community awareness. Street surveys of active drug users confirm that even among this difficult to reach population, awareness levels are high. Dispelling myths of their disregard for personal health and well-being, drug users have also often adjusted their behavior to reduce, if not to eliminate, their risk.

AIDS education, however, is hardly the "magic bullet" of HIV prevention. The success of AIDS educational programs must be weighed against their limitations. Awareness of risk does not necessarily translate to risk avoidance, especially on a consistent, long term basis. Street surveys indicate greater usage of condoms and clean needles by drug users, but absolute behavioral changes are not common. Even within the well-informed and well-organized gay male community, the consistent practice of safer sex is problematic.

The high level of community awareness about HIV transmission and prevention merely provides a starting point for risk reduction action. Prevention in the 1990s needs to extend beyond imparting information to affecting individual behavior through influencing social norms and developing personal risk avoidance skills. A major preventive goal for the 1990s should be to establish as the social norm the use of condoms in sexual encounters in which the potential for HIV transmission cannot be ruled out, a sexual behavior equivalent of "universal precautions" in clinical practice.

Individual behavior modification often requires more than individual choice or personal determination; influencing normative social behavior often requires more than effective advertising. Prevention messages without prevention support will be of limited value. A substantial reduction in drug addictive behavior will require external interventions and supports. HIV prevention will require that effective addiction treatment services be available for all those in need. Ongoing supportive counseling for those at risk for infection as a result of their addiction or for those already infected must be available to assist individuals to maintain low-risk or no-risk lifestyles. To optimize their impact, preventive messages and services require the involvement of affected communities in their design and implementation. Promoting the active participation of groups, organizations and communities in closest contact with at-risk individuals must remain a cornerstone of state and local public health policy.

Innovation, local participation, and broad program saturation must characterize HIV prevention tactics within New York State in the 1990s. Already underway are efforts to maximize public health settings as opportunities for intervention; to incorporate HIV counseling and the encouragement of testing in routine clinical practice; to develop and expand community-based outreach education; and to target intensified interventions at populations and communities at increased risk. These and other elements of HIV prevention require careful design and implementation, as well as rigorous oversight and evaluation. We expect that these tactics will help reduce the spread of HIV. However, in the absence of a vaccine or curative therapy for what may be an invariably fatal communicable disease, these preventive interventions are likely to be unacceptably incomplete.

Communities at greatest risk for HIV which are already suffering from a myriad of clinical and social ills are essentially "public health disaster areas," warranting a coordinated and comprehensive governmental response appropriate to the severity of their condition. Moreover, it is imprudent to assume that through the 1990s HIV will not spread beyond these communities.

In the 1990s, HIV prevention needs to be broadly defined, reflecting the social as well as the clinical etiology of AIDS. The public health approach to disease prevention properly considers agent, host, and environmental influences on transmission and seeks to break the chain wherever possible. Clinical and laboratory research pursues intervention at the point of the agent through vaccine and chemotherapy development. Educational interventions are the primary means of reducing host susceptibility. Dealing with the environmental dimension will require that drug abuse and the underlying social morbidity be vigorously pursued. Responsibility for the prevention of

HIV cannot be passed on solely to the at-risk individual or to the community through the dissemination of preventive health information—not when the connection between HIV and addictive behavior is evident; nor when the connection between drug abuse and the social milieu is also apparent; and not when the persistence of other public health problems within these communities, despite a variety of traditional public health interventions, including community education, is so clearly visible.

Government at all levels—federal, state, and local—must meet its responsibility to promote and protect the public health and welfare. Compartmentalized, disjointed governmental responses need to be replaced with intergovernmental, interagency, and governmental-community participation in strategic planning to address the full spectrum of needs in the highest risk communities. One step the federal government should explore with high prevalence states is to establish a pilot program of public health disaster relief grants for communities which qualify on the basis of the extent of measurable social and clinical morbidity. Such grants could require a comprehensive assessment of community need, an integrated approach to planning and project oversight, and a program which has an explicit agenda and timetable for each of its interconnected elements.

AIDS is forcing us to reexamine how we approach public health problems, particularly when we lack a vaccine or cure. AIDS will likely dramatically affect the public health agenda in the 1990s, most notably bringing substance abuse to the fore as a top public health priority. The manner in which public health is practiced is also likely to undergo an accelerated evolution to community-oriented models of prevention and service delivery as we enter the 21st century. One of the few benefits which society may derive from the devastation of AIDS may be a fuller understanding of the interaction between social morbidity and health outcomes within a community and the implications of both conditions on the health of society at large. Out of such awareness may emerge more effective public health approaches to long-standing problems and a firmer societal resolve to demand strategies to correct both the underlying social ills as well as their clinical manifestations.

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The increasing role of primary care in the management of HIV-infected patients

Since the identification of the etiologic agent of the acquired immunodeficiency syndrome (AIDS), much has been learned about the natural history of the disease. Until recently, medical education and the clinical literature have focused on patients with Centers for Disease Control (CDC)-defined AIDS. Clinically, the extraordinary complexity of these patients' illnesses and the multisystem involvement by both opportunistic infections and opportunistic malignancies have necessitated intervention by teams of medical specialists.

However, this emphasis on the end stage of infection with the human immunodeficiency virus (HIV) is now changing. Recognition of the chronic and progressive nature of HIV infection as well as the observation that most HIV-infected individuals are asymptomatic have focused attention on this population.[1,2] Advances in a number of areas including identification of clinical markers for progression of HIV disease,[3] new indications for the prophylaxis of opportunistic infections,[4] as well as broadened indications for zidovudine (formerly known as azidothymidine, or AZT)[5] will allow the clinician to provide multiple interventions earlier in the course of the disease. As the concept of HIV disease changes from that of a rapidly progressive fatal disease to a chronic illness requiring multiple laboratory, therapeutic, and social interventions, the appropriate clinician contact is changing from the infectious disease specialist, oncologist, and general internist who specialize in HIV care to the primary care practitioner. This changing locus of care is the result of dramatic progress in the understanding of the natural history of HIV disease and the prevention of the complications of HIV-induced immunosuppression.

Among the most important advances has been the identification of a number of specific clinical markers that have been associated with progression towards CDC-defined AIDS.[3,6] Perhaps the most useful of these is the CD4 (T4) lymphocyte count and/or CD4 percent. The use of such a marker is consistent with what is known regarding the immunologic defect in HIV disease. HIV infection results in the eventual destruction of CD4 cells, contributing partly to the immunosuppression characteristic of the disease. By following the CD4 count or percent periodically, clinicians can identify specific points at which to employ appropriate prophylactic measures to prevent the more common opportunistic infections. Several studies have now demonstrated that patients with a CD4 count of less than 200 to 250 cells/mm^3 or those patients whose CD4 cells are less than 20% of total lymphocytes are at markedly higher risk of opportunistic infections than are those with higher circulating CD4 counts.[3,4] CD4 enumeration is now widely available, and with the use of this marker as well as others (HIV p24 antigen, beta2-microglobulin), HIV-infected patients at highest risk of developing opportunistic infections can be identified.

Yet the development of such markers would be clinically useless in the absence of effective prophylactic measures against opportunistic infections. This is no longer the case with regard to *Pneumocystis carinii* pneumonia (PCP). This opportunistic pneumonia develops in 80% of patients with AIDS, and 60% acquire it as their initial AIDS-related opportunistic infection.[7] Trimethoprim-sulfamethoxazole[8] and aerosolized pentamidine[9] have now been shown to be effective agents in the prevention of PCP. As a result, in June 1989 the CDC recommended that PCP prophylaxis be initiated using one of the above medications in the setting of a CD4 count of less than 200/mm^3 or a CD4 percentage of less than 20% of total lymphocytes.[4] Thus the use of primary prophylaxis for an opportunistic infection is now linked to serial studies of CD4 cells such that an appropriate intervention strategy can be designed to prevent the most common serious opportunistic pathogen in HIV-infected patients.

Monitoring HIV-infected asymptomatic patients may also play a role in the therapy of HIV infection itself. Zidovudine (AZT) was originally employed in HIV infected patients with AIDS or severe AIDS-related complex (ARC) (CD4 counts < 200). In this population it was found to decrease both mortality and frequency of opportunistic infections.[10] The indications for zidovudine now appear to be extended. In August 1989, the National Institute of Allergy and Infectious Diseases AIDS Clinical Trial Group (ACTG) announced the results of studies of AZT used in asymptomatic HIV-infected persons as well as in patients with early ARC (CD4 counts of 200–800).[5] Both of these studies significantly extended the use of zidovudine by demonstrating that HIV-infected asymptomatic persons with less than 500 CD4 cells as well as persons with symptomatic HIV infection with less than 500 CD4 cells will benefit from zidovudine therapy with minimal side effects. Patients in the treatment arms of both studies had a highly significant decreased rate of progression to AIDS or severe ARC than those receiving placebo.

The results of these studies have clearly provided a rational clinical base for early intervention in HIV disease. Concrete protocols for initial evaluation and immunologic follow-up of infected individuals can now be developed. The components of such a clinical program would include periodic monitoring of immune function, prophylaxis against selected opportunistic pathogens, the initiation and management of anti-HIV therapy where indicated, the identification of HIV-associated infections such as tuberculosis and syphilis, as well as the diagnosis and treatment (through the use of specialty consultants if necessary) of opportunistic infections. Finally, such a program would include unique services for women including referrals for abortions, and insuring access for pediatric HIV testing.

The provision of social and emotional support must be linked to clinical and immunologic follow-up. Depression and anxiety on being informed one is HIV-positive is a frequent and not inappropriate response. However, through counseling and, if necessary, referral, the serious consequences of these psychologic reactions may be minimized. Legal and social issues surrounding HIV infection must also be addressed, and include do not resuscitate orders, durable power of attorney, and issues of confidentiality involving friends, family, employers, and insurers. Contact notification, a critical issue in the control of a disease transmitted by sexual contact or sharing injection equipment, must also be addressed. HIV testing (or referral) for these potentially infected contacts must be provided. Finally, behavior intervention to reduce subsequent transmission of HIV is critical. In the same manner that one collects information for clinical decisions, risk assessment must be conducted to identify transmission prone behaviors. The employment of appropriate risk reduction programs can then be utilized to reduce the risk of subsequent HIV transmission.

The primary care practitioner appears to be well positioned for coordinating the comprehensive care these HIV-infected patients require as well as for providing continuity of care.[11] As do other patients with lifelong chronic diseases (ie, diabetes mellitus, hypertension), HIV-infected patients require the services of primary care practitioners who are capable of providing the guidance and support that such long term diseases necessitate as well as clinical support. It is unlikely, however, that the primary care practitioner can provide all of these components alone. Rather, a more appropriate role might be to coordinate the various clinicians necessary to provide care to HIV-infected individuals. In larger programs such a staff might consist of social workers, health educators, and nurses while in smaller practices, it might involve an identified resource person who has become familiar with HIV-related educational interventions as well as community support services.

While the rationale for increased involvement of the primary care practitioner in the management of HIV disease may be clear, so are the impediments. These include financial and educational constraints as well as the need to overcome many of the prejudices that accompany this disease.

The costs of large scale early intervention programs are formidable. Arno et al[1] recently estimated the costs of providing care to an individual HIV-infected patient (requiring PCP prophylaxis and zidovudine) at over $9,000 per year. In New York City, making the assumption that 50% of HIV infected individuals are identified and placed in care, this translates to slightly over $800 million per year in additional costs. The coordination of state, federal, and other payers in addressing this problem is clearly important.

Primary care physicians need to develop an adequate knowledge base in order to provide ambulatory care to patients with HIV infection. Most HIV-related medical education is provided in training institutions and concentrates on the end stage HIV-infected patient with AIDS. The rapidly expanding knowledge base regarding the management of the asymptomatic HIV-infected patient needs to be efficiently transmitted to primary care practitioners so that they can provide outpatient management of their patient's HIV disease.

Primary care physicians may avoid treatment of patients with HIV infection because of concern regarding retention of their nonaffected patient pool should it become public knowledge that they are providing care to HIV-infected patients. This concern may have been demonstrated in a random study of non-institutionalized adults in the United States in which 25% of those who responded indicated they would change clinicians if they knew their physician were treating someone who was HIV infected.[12] The path for the leadership in medicine here is clear. The medical profession at both the organizational and individual level must increase its efforts to educate patients regarding the absence of personal risk in seeking care from physicians who care for HIV-infected patients.

The impediments to expanded early treatment of HIV disease are significant. However, we are at a crucial point in the history of this disease. Can the interventions defined in clinical studies be readily transferred to the affected patient population? Through early intervention in the natural history of this viral infection we now have the opportunity to change both the perception and the reality of HIV infection from that of a rapidly fatal disease to a chronic illness. Through the use of the primary care model, we approach the real possibility of preventing transmission of HIV and the rapid progression of disease in those already infected.

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Dissemination of *Pneumocystis*

Since the beginning of the acquired immunodeficiency syndrome (AIDS) epidemic, *Pneumocystis* has been and continues to be the major pathogen that infects these patients. Even so, relatively little is known about the biology of this organism and debate continues on its taxonomy. The majority of patients with AIDS will experience *Pneumocystis carinii* pneumonia (PCP), many will have repeated episodes, and a few will develop extrapulmonary disseminated disease.

A variety of opportunistic organisms have been known to disseminate widely in patients with immunodeficiency. That *Pneumocystis* can widely disseminate in patients with AIDS has now been clearly shown by the report of Ravalli et al[1] in this *Journal*. Other case reports have also shown *Pneumocystis* to be present in extrapulmonary sites in AIDS patients. However, very few have shown such wide dissemination as this case report. Virtually no organ examined was left uninvolved. Yet, dissemination of *Pneumocystis* must be a rare event, given the large numbers of AIDS patients with documented PCP, and the growing number of AIDS autopsy series that have failed to document cases with such widespread disease.[2]

The ability of an organism to disseminate requires that it be able to gain access to the vascular system and overcome the many obstacles of an immune system that is intact. How *Pneumocystis* gains access to the vascular system and disseminates is not known. *Pneumocystis* is typically observed within the alveolar spaces and only rarely has it been reported to be present in vascular spaces, and then usually in cases in which it has disseminated. Perhaps *Pneumocystis* gains access by secondary spread after tissue destruction by another organism. It is not unusual to find more than one organism in the lungs of patients with AIDS. It is also possible that reactivation of *Pneumocystis* could, on rare occasions, occur in extrapulmonary locations. Recent identification of antibodies in human sera specific for *Pneumocystis carinii* has added further evidence that most of the adult population has been exposed to *Pneumocystis*, and thus infection is presumed to be from reactivation.[3] Another plausible explanation is that enough immune activity is present in most patients with AIDS to inhibit either vascular invasion or extrapulmonary foci from becoming established. Nevertheless, in some, immunity could diminish to such a degree as to allow spread beyond the lungs. This latter hypothesis would imply a stepwise loss of immunity in some patients that eventually allows such a normally indolent organism not only to establish an infection in the air spaces, but also to disseminate.

The study by Kovacs et al[4] comparing PCP in patients with AIDS versus patients with other types of immunodeficiency has shown that PCP has a more indolent course in AIDS patients. This would imply that there are specific defects in the immune system that allow for the level of aggressiveness of *Pneumocystis*. The immune defects of AIDS are only beginning to become known, and they involve both cellular and humoral immunity. A recent study at the National Institute of Health[5] has shown a correlation between the occurrence of PCP in AIDS patients with absolute CD4(T4) levels of 200 cells/mm^3 or less. It would be interesting to know if such a correlation between dissemination of *Pneumocystis* and numbers of peripheral CD4 cells exists. Use of recently developed monoclonal antibodies to *Pneumocystis*[6] or perhaps recombinant DNA studies might also help in determining if *Pneumocystis* organisms that disseminate are different from those that stay localized. In addition, monoclonal antibodies to *Pneumocystis* might someday prove useful for in vivo diagnosis.

Animal studies[7] have implicated both cellular and humoral immunity in the pathogenesis of *Pneumocystis*, and natural killer cell activity may also be involved.[8] The role of macrophages is not known. It is interesting to note that phagocytosis without destruction is prominent with many of the opportunistic infections in AIDS patients. *Pneumocystis* organisms, however, seem to remain chiefly outside the phagocytic cells, within the characteristic foamy exudate that accompanies the infection regardless of the organ involved.

A better understanding of the parts of the immune system responsible for defense against *Pneumocystis* may ultimately lead to better methods of control. Until more effective treatments for PCP become available, awareness of the possibility of dissemination of *Pneumocystis* in patients with AIDS may have an impact on the usefulness of current treatments, including aerosol prophylaxis.

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Changes in AIDS risk behavior among intravenous drug abusers in New York City

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ABSTRACT. **Forty newly admitted patients to a methadone treatment clinic in New York City were interviewed to assess their patterns of intravenous (IV) drug abuse, sexual activity, and other risk behaviors for the acquired immunodeficiency syndrome (AIDS). The study revealed a marked decline in needle sharing since 1984 (46% to 14%), despite a concurrent doubling of the number of weekly IV drug injections per subject (10 to 21). High rates of promiscuous heterosexual activity were noted among men (80% said they were currently active with more than one woman), while the rates of condom use were low, as reported by both men and women (<30%). Recent reductions in weight were reported by 40% of subjects, and the average body mass of all individuals was subnormal. Thus, despite an apparent decline in needle sharing, high risk sexual behavior is apparently persisting, drug use is increasing, and nutritional deficits are prevalent among IV drug abusers. Synergism of these factors should be considered in the manifestation of HIV infection among IV drug abusers.**

(NY State J Med 1990; 90:123–126)

Acquired immunodeficiency syndrome (AIDS) presents new and challenging dimensions for epidemiologic investigation. The clandestine nature of the dominant risk factors in AIDS, sexual activity and intravenous (IV) drug abuse, makes ascertainment of accurate information particularly difficult. Most of the studies of the risk factors for AIDS have focused on the type and frequency of sexual activity among homosexual and bisexual men.[1,2] In the New York City area, however, the majority of AIDS patients appear to be IV drug abusers.[3] Individuals of this risk group are not well characterized with respect to changes that have occurred over time in their sexual activity and drug use. For this reason, we have begun systematic interviews of patients newly admitted to the Methadone Treatment Clinic of the Bronx Psychiatric Institute of the Albert Einstein School of Medicine. Our purpose is to determine changes that have occurred in IV drug abuse and sexual activity in a sample of individuals representative of the large population of IV drug abusers in the New York City area.

PATIENTS AND METHODS

Under guarantee of anonymity, newly admitted patients to the Methadone Treatment Clinic of the Bronx Psychiatric Institute of the Albert Einstein School of Medicine in New York City were intensively interviewed to assess their patterns of IV drug abuse, sexual activity, and other risk behaviors for AIDS and other sexually transmitted diseases. For this purpose, a structured questionnaire was utilized, designed to ascertain information about the frequency, type, and temporal pattern of heterosexual, bisexual, and homosexual activity, illicit drug use, alcohol and tobacco utilization, and other related behaviors such as needle sharing, condom use, and promiscuity. A total of 40 randomly selected subjects were interviewed—ten females and 30 males. None of the subjects selected refused to participate. Nine of the ten females presented for interview with a male companion (either spouse or sexual partner), thereby facilitating paired analysis and validation of certain shared exposures. Subjects entering the clinic were not routinely tested for HIV infection; however, an initial physical examination was performed to rule out AIDS or AIDS-related complex prior to treatment.

Retrospective time series data were examined by linear regression techniques to delineate temporal changes in reported activities. Adjustment for age was made utilizing analysis of covariance. Paired data were examined by correlation analysis to provide validation and misclassification estimates for heterosexual activities within couples.[4,5]

RESULTS

Table I provides demographic and other descriptive data for the 40 subjects interviewed. Subjects were predominantly black or Hispanic (66%), between 25 and 45 years of age (82%), of Catholic religious preference (79%), and many were immigrants to the New York City area from Latin America (32%). These individuals were typically of low socioeconomic status (95% currently unemployed) with subpar educational levels (50% did not complete high school). The majority (55%) were raised in single parent homes and most were never married, separated, or divorced (89%). In our experience, the demographic profile of this sample is highly representative of all newly admitted patients to the methadone treatment clinic.

Among all subjects, there was a definite tendency toward poor nutrition. Twelve men and four women (40% overall) reported weight loss in the past year; the mean weight loss was 11 lb (range, 2–23 lb). Based on the quetelet index, $QI = wt(kg)/ht^2(cm)$, the average body mass was 22.7 for males and 22.3 for females, both of which are about one standard deviation below the normal adult values for this age group.[6]

In Figures 1 and 2, the average number of different sexual partners per year and the frequency of intercourse per week are plotted over time to reflect temporal patterns of heterosexual activity. For each year examined, both measures of heterosexual activity were two- to fourfold higher in men compared to women. Overall, the

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TABLE I. Descriptive Features of IV Drug Abusers in New York City

		Age (yr)		Race (%)			Religion (%)		Max Education (%)			Foreign	Single*	Unemployed	Ht	Wt	
Sex	N	Mean	Range	Wht	Blk	Hisp	Cath	Prot	Elementary	High School	College	(%)	(%)	(%)	(in)	(lb)	QI†
Males	30	39	23–58	38	14	48	86	10	52	19	29	33	90	93	73	159	22.7
Females	10	33	24–42	14	43	43	57	43	43	43	14	29	86	90	64	129	22.3
Total	40	38	23–58	32	21	46	79	18	50	25	25	32	89	92	—	—	—

* Single denotes never married, separated, or divorced subjects.

† Quetelet index = $wt(kg)/ht^2(cm)$.

average number of different heterosexual partners per year was 3.8 for men versus 1.5 for women ($p < 0.01$), and the frequency of intercourse per week was 2.8 for men versus 1.1 for women ($p < 0.05$).

Condom use over time is plotted in Figure 3. While the frequency of condom use has increased somewhat in recent years, the overall utilization has remained relatively low, with fewer than 30% of individuals reporting that they or their partners ever used condoms in 1988.

Temporal trends in IV drug abuse are summarized in Figures 4 and 5 as the age-adjusted frequency of heroin or cocaine injections and the age-adjusted frequency of needle sharing. Notably, while the number of injections of heroin or cocaine increased markedly since 1984 (10 to 21, $p < 0.01$), the frequency of needle sharing dropped dramatically during the same time period (46% to 14%, $p < 0.01$).

The frequency of individuals' "ever sharing needles" during their lifetime was dependent on when they started using IV drugs, ie, 32 of 36 (89%) subjects who started drug use before 1984 reported ever sharing needles, whereas only one of four who started later reported ever sharing needles.

Levels of sexual activity and IV drug abuse are given by age and sex in Table II. Heterosexual activity of both men and women declined with age based on the frequency of intercourse and number of sexual partners. However, clear differences are evident between the reported activity of men and women, with men reporting two- to sevenfold higher frequencies of intercourse per week and two- to fourfold greater number of different partners per year than women of corresponding age. Eight of the men (27%) and two of the women (20%) reported heterosexual anal intercourse as a regular (at least once per month) sexual activity. Seven additional men (23%) reported heterosexual anal intercourse as an isolated occurrence in their past sexual history. Of the 30 men interviewed, only one (3.3%) reported bisexual activity, and this (insertive anal intercourse) occurred while the subject was incarcerated. None of the women reported bisexual practices.

No clear age trends are evident in the weekly frequency of injecting heroin and/or cocaine (Table II). On the average, males of the sample began using IV drugs at age 19 years (range, 11–36 yr) and females at age 23 years (range, 14–31 yr). The peak frequency of IV drug injections (nearly three injections per day) occurred in the 30–34-yr age group for both sexes. Older age groups showed a slight decline in frequency of use.

With respect to the correspondence between reported sexual activity within the couples, there was a marginally significant positive association between mutually occurring sexual activity reported by members of the nine pairs of subjects: ie, the intraclass correlation was 0.54 for the frequency of intercourse ($p < 0.10$). There was, however, a tendency for males to report higher activity than their mates—the average intrapair difference in frequency of intercourse per week was 1.1 encounters ($p < 0.10$). Interestingly, six of the men reported current activity with other women, whereas only one of the nine women reported activity with other men.

DISCUSSION

Several important findings emerge from this investigation. Notably, one major parameter of AIDS risk behavior among IV drug abusers has declined. The frequency of needle sharing has dropped from 46% to 14% between 1984 and 1988 as reported by newly admitted patients to a methadone treatment clinic in New York City. The same trend is reflected by differences in the frequencies of subjects' "ever sharing needles" stratified by the time at first use: ie, 89% of those who first used IV drugs before 1984 reported "ever sharing needles," compared to only 25% who started afterwards. This decline in needle sharing coincides with the citywide program advocating use of sterile hypodermic needles and syringes,[7] but it has also been attributed to possible denial.[8]

Despite the apparent awareness among IV drug users of the risk from needle sharing, our results indicate that high risk sexual behaviors are persisting among this population. The level of heterosexual activity among these individuals is high, and heterosexual activity limited to one partner is not the rule, particularly among male IV drug

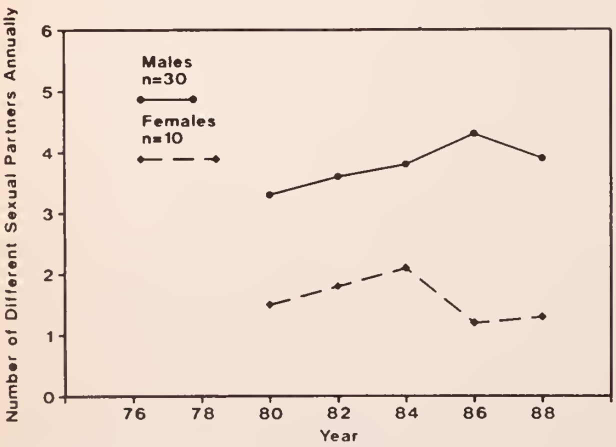

FIGURE 1. Age-adjusted number of different heterosexual partners per year among IV drug abusers in New York City.

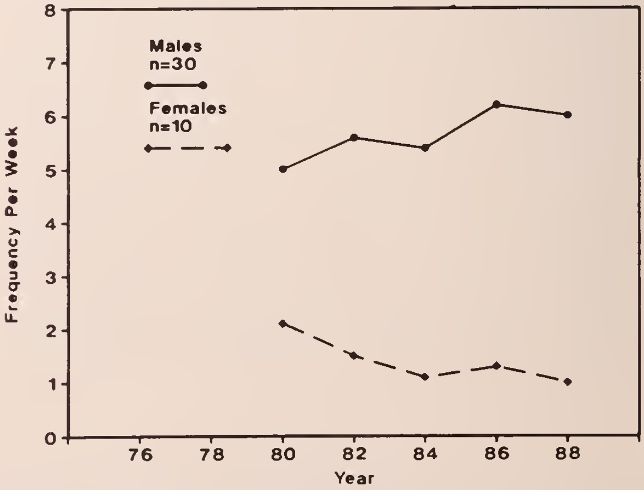

FIGURE 2. Frequency of coitus among IV drug abusers in New York City.

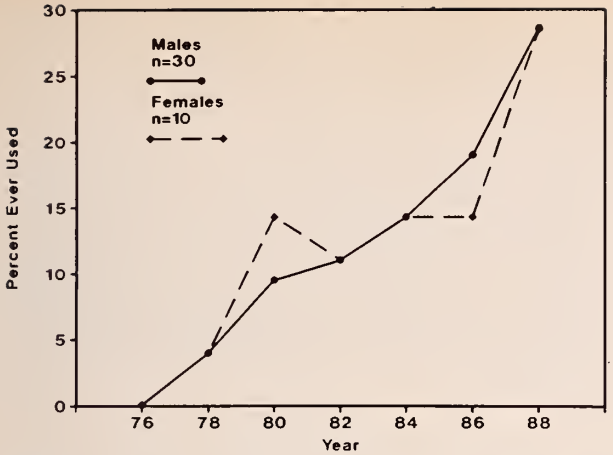

FIGURE 3. Frequency of condom use among IV drug abusers in New York City.

abusers. Men reported having intercourse with an average of four different partners per year with average frequency of about three times per week, and more than 80% of the men sampled reported engaging in current heterosexual activity with more than one female. A substantial number of subjects also reported regular anal intercourse (27% of males and 20% of females). Furthermore, condom utilization remains low among both males and the sexual partners of females of this high risk group (less than one in three reported ever using condoms in 1988).

It is also noteworthy that the reported utilization of IV drugs among these individuals has increased in the past four years. Specifically, the number of injections of heroin or cocaine per week increased from 10 in 1984 to 21 in 1988. This upward trend in utilization is consistent for both men and women.

Concurrent with the increased frequency of drug use, average weight and body mass was markedly reduced among all subjects, and a more detailed physical examination of eight individuals revealed one or more overt signs of nutritional deficiencies, such as pallor of the skin and mucous membranes, xerosis of the skin, muscle wasting, stomatitis, and cheilosis. These preliminary data therefore suggest that rates of malnutrition may be high among IV drug abusers, although effects of coexisting HIV infection cannot be ruled out since the study subjects did not undergo HIV testing.

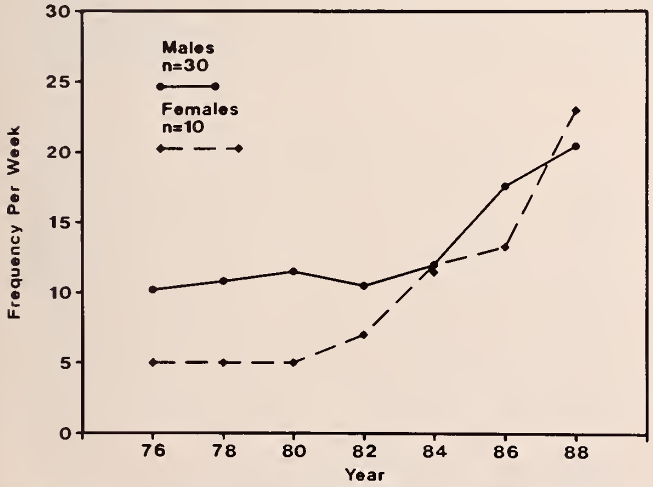

FIGURE 4. Frequency of injections of heroin or cocaine among IV drug abusers in New York City.

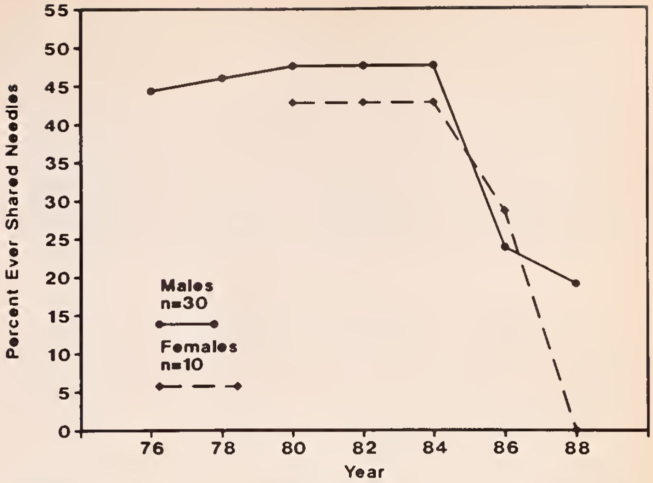

FIGURE 5. Frequency of needle sharing among IV drug abusers in New York City.

Stoneburner et al[9] recently noted an increase in mortality among IV drug abusers in New York City, which has occurred coincident with the AIDS epidemic but cannot be attributed to AIDS by its current definition. They go on to suggest that a wider spectrum of HIV-related diseases may be present among IV drug abusers and minorities than was previously suspected, and they stress the need to revise the diagnostic criteria for AIDS. Since, under the current diagnostic criteria for AIDS, IV drug abusers already comprise the majority of AIDS patients in the New York City area,[3] it is probable that different risk factors are present in IV drug abusers than in homosexual men.

The sample of intravenous drug abusers examined in this study is small, consisting only of patients self-selected into a methadone treatment clinic. Consequently, the

TABLE II. Age/Sex-Specific Levels of Sexual Activity and Drug Use Among IV Drug Abusers in New York City*

Age (yr)	Sexual Intercourse Per Week	Number Partners Per Year	IV Drug Injections Per Week
	Males (N=30)		
<20	6.1 (1.0)	4.8 (1.2)	9.0 (6.3)
20–24	7.1 (1.6)	5.3 (1.1)	12.3 (5.5)
25–29	5.4 (1.6)	5.3 (1.1)	10.7 (5.5)
30–34	7.9 (1.2)	3.9 (1.8)	18.0 (4.2)
35–39	4.1 (1.4)	2.7 (1.0)	17.7 (5.0)
40+	2.5 (1.2)	2.2 (0.8)	12.8 (4.0)
	Females (N=10)		
<20	3.0 (1.0)	1.3 (0.8)	16.3 (8.2)
20–24	1.6 (0.9)	1.6 (0.7)	16.8 (7.2)
25–29	2.0 (0.7)	1.3 (0.6)	9.0 (6.1)
30–34	1.0 (1.3)	0.5 (1.0)	26.0 (10.5)
35–39	0.6 (0.7)	1.8 (0.7)	15.8 (7.5)
40+	0.6 (0.7)	1.5 (0.8)	9.8 (8.6)

* Responses are age-specific, projected to the appropriate year between 1976 and 1988. Standard errors of means are in parentheses.

trends we observed should not be viewed as conclusive with respect to the population of intravenous drug users in New York City. Nevertheless, as a working hypothesis based on these results, we suggest that heightened dosing with heroin and cocaine coupled with nutritional deficiencies may play a role in enhancing the disease spectrum of HIV among IV drug abusers. We emphasize the need for increased research to identify the nature of these factors and to establish their roles in the manifestation of AIDS.

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Assessment of AIDS knowledge in selected New York State sexually transmitted disease clinics

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ABSTRACT. A 1988 survey regarding the level of knowledge about acquired immunodeficiency syndrome (AIDS) among 505 clients of sexually transmitted disease (STD) clinics demonstrated 80% correct responses. However, 13% thought AIDS could be transmitted via sharing utensils, and 51% were unaware that acquiring another STD could increase one's risk of human immunodeficiency virus (HIV) infection. In addition, only 10% always used condoms. An average 69% of patients obtained their AIDS information from newspapers, magazines, television, or radio; and 30% acquired information from medical or clinic staffs. In response to this survey, a videotape on AIDS was developed to show to patients of STD clinics while they are waiting to see a physician.

(NY State J Med 1990; 90:126–128)

In the United States, 81,083 adults with acquired immunodeficiency syndrome (AIDS) had been reported to the Centers for Disease Control by December 31, 1988, and an estimated 1.0 to 1.5 million persons had been infected with the human immunodeficiency virus (HIV).[1] The increasing spread of HIV among heterosexual persons who have multiple sexual partners, and the positive association between HIV positivity and genital ulcer diseases (syphilis, genital herpes) demonstrates that patients attending clinics for sexually transmitted diseases (STDs) are at high risk for HIV infection.[2–6] Until more effective medical therapies and preventive measures are developed, public education is likely to be the most important means for reducing risk behaviors associated with HIV infection.[7,8]

As of December 31, 1988, New York State had the largest number of AIDS cases of any state (20,476), with 1,003 (4.9%) cases considered to be heterosexually acquired AIDS cases.[9] Of these cases, 623 (62%) were in persons who reported sexual contact with a person with or at increased risk for HIV infection, and 380 (38%) were in persons born in countries with high rates of heterosexual HIV transmission. Since persons attending STD clinics are sexually active and at high risk for HIV infection and AIDS, the health department is committed to providing attendees with education and risk reduction messages to minimize disease transmission. A necessary first step, described in this report, was to evaluate the knowledge, attitudes, and behaviors of the populations utilizing STD clinics to assist in development of an educational program.

PATIENTS AND METHODS

Between January 18 and January 29, 1988, a pilot survey was conducted in three county health department STD clinics. All patients attending these clinics were asked to complete a questionnaire that was administered by a clinic staff member (nurse or public health advisor). Those patients who came repeatedly during the two-week period were not interviewed more than once. The questionnaire included basic demographic information, questions on AIDS knowledge, condom use, AIDS information sources, and AIDS education. The counties selected for this survey represented geographic areas across New York State: Monroe County (population, 722,000) in the west, Onondaga County (population, 463,000) in the middle of the state, and Nassau County (population, 1,320,000) in the southeast. STD clinic census and morbidity estimates for these counties for calendar 1987 were as follows: Monroe County—7,451 patient visits, 3,142 cases of gonorrhea, 35 cases of early (less than 1 yr)

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syphilis; Onondaga County—5,536 patient visits, 1,472 cases of gonorrhea, 33 cases of early syphilis; Nassau County—8,542 patient visits, 2,621 cases of gonorrhea, and 156 cases of early syphilis.

A total of 508 patients were interviewed; three questionnaires from patients who did not complete the interview were excluded from the analysis. A total score was calculated for each study subject, with one point for each correct answer on the five AIDS knowledge questions. No points were awarded for an incorrect or unsure answer. One-way analysis of variance was used to compare the mean score within each demographic variable, and was also used for each question to test the percent with correct responses. Chi-squared analysis was applied to compare condom use and AIDS information sources with the demographic variables.

RESULTS

Responses from 505 subjects who had completed the interviews were analyzed. Two hundred thirty-eight (47%) subjects were from Nassau County, 139 (27%) were from Onondaga County, and 128 (25%) were from Monroe County. Three hundred forty-six (69%) patients were male, 280 (55%) were less than 25 years of age, 268 (53%) were black, 18 (4%) were Hispanic, 225 (45%) had at least some college education, and 261 (52%) were first-time visitors.

Knowledge of AIDS was generally good: 485 (96%) knew that a pregnant woman can give her baby the AIDS virus if she is infected; 481 (95%) knew that the AIDS virus can be spread if blood or semen infected with AIDS gets into the body. However, 67 (13%) believed that AIDS could be spread by sharing plates, cups, and silverware; 257 (51%) did not realize that acquiring another STD might increase their risk for acquiring AIDS; and 42 (8%) thought that only intravenous drug users or gay men could get AIDS. The mean score of AIDS knowledge was high in patients from Nassau County, while the scores were low among persons over age 35 years and those with less than a high school education (Table I).

Condoms were used always by 53 (10%), sometimes by 298 (59%), and never by 142 (28%) of the patients. The "ever-use" of condoms, defined as persons answering always or sometimes for condom use, was significantly higher among persons who were from Nassau County (odds ratio (OR) = 1.84, confidence interval (CI) = 1.15–2.93), and in those with some college education (OR=1.67, CI = 1.01, 2.75). Females and persons of age 35 or more years used condoms significantly less frequently than males and younger age groups (Table II).

Most patients obtained some of their AIDS information from newspapers, magazines, radio, or television. Younger people and females were more likely to obtain their AIDS information from medical or clinic staff (Table III). Blacks were more likely to get some

TABLE I. Frequency Distribution and Mean Score of AIDS Knowledge by Demographic Variables Among 505 Respondents

Variable	Number	Percent	Mean Score	F test p value
County				
Onondaga	139	27.5	3.87	
Monroe	128	25.3	4.02	
Nassau	238	47.1	4.09	0.03
Age (yr)				
<25	280	55.4	4.00	
25–34	153	30.3	4.18	
35+	66	13.1	3.74	0.001
Race				
White	200	39.6	4.04	
Black	268	53.1	4.01	
Other	31	6.2	3.77	0.22
Sex				
Male	346	68.5	4.01	
Female	157	31.1	4.04	0.68
Education				
<12 yr	117	23.2	3.73	
12 yr	157	31.1	3.98	
>12 yr	225	44.6	4.18	<0.001
Total	505	100.0	4.01	

TABLE II. Comparison of "Ever Use" to "Never Use" of Condoms by Demographic Variables Among 505 Respondents

Variable	Ever Use N (%)	Never Use N (%)	Odds Ratio (95% CI)
County			
Onondaga	86 (65.2)	46 (34.8)	1.00
Monroe	83 (65.9)	43 (34.1)	1.03 (0.62–1.73)
Nassau	182 (77.4)	53 (22.6)	1.84 (1.15–2.93)*
Age (yr)			
<25	204 (74.4)	70 (25.5)	1.00
25–34	105 (69.1)	47 (30.9)	0.77 (0.49–1.19)
35+	38 (62.3)	23 (37.7)	0.57 (0.32–1.01)
Race			
White	139 (71.6)	55 (28.4)	1.00
Black	186 (71.0)	76 (29.0)	0.97 (0.64–1.46)
Other	20 (64.5)	11 (35.5)	0.72 (0.32–1.60)
Sex			
Male	250 (74.4)	86 (25.6)	1.00
Female	99 (63.9)	56 (36.1)	0.61 (0.40–0.91)*
Education			
<12 yr	75 (66.4)	38 (33.6)	1.00
12 yr	105 (67.7)	50 (32.3)	1.06 (0.63–1.78)
>12 yr	168 (76.7)	51 (23.3)	1.67 (1.01–2.75)*
Total	351 (69.5)	142 (28.1)	

* $p < 0.05$.

TABLE III. Percentage of AIDS Information Sources by Demographic Variables Among 505 Respondents

Variable	Friends	Medical or Clinic staff	Newspaper or Magazines	Radio or Television
County				
Onondaga	20.9	32.4	58.3	66.2
Monroe	19.5	25.8	70.3	70.3
Nassau	23.5	32.4	74.4	70.6
			($p<0.01$)	
Age (yr)				
<25	24.3	35.7	67.5	66.4
25–34	21.6	28.1	73.2	73.2
35+	13.6	16.7	65.2	72.7
		($p<0.01$)		
Race				
White	16.0	29.0	74.5	69.0
Black	26.9	34.3	65.3	70.5
Other	16.1	16.1	64.5	61.3
	($p<0.05$)			
Sex				
Male	20.2	27.2	70.2	69.9
Female	25.5	38.2	66.2	67.5
		($p<0.05$)		
Education				
<12 yr	19.7	24.8	47.9	65.0
12 yr	24.2	29.9	68.2	68.8
>12 yr	21.3	34.7	80.9	72.0
			($p<0.01$)	
Total	21.8	30.7	68.9	69.3

information from friends. Two hundred and fifteen patients (43%) asked for additional information regarding protection from AIDS, and 197 (39%) requested information on AIDS symptoms.

DISCUSSION

The transmission of HIV can occur through sexual contact with an infected person, sharing of infected needles, infected blood products, or perinatally from an infected mother to her child. Until there is a medication that decreases infectiousness, education and behavior change will be the main means to curtail the spread of HIV. Since unprotected sexual contact with an infected person is the common mechanism of transmission for all sexually transmitted diseases, the same populations are also at high risk for HIV. Thus, development of an educational program for use in STD clinics is essential.

This pilot survey was well received by both respondents and interviewers, with only 0.6% of clients not completing the interviews. Knowledge of AIDS was generally good, comparing favorably with the 1987 and 1988 National Health Interview Surveys (NHIS) of AIDS knowledge and attitudes, in which approximately 3,500 questionnaires per month were administered to randomly selected adults more than 17 years of age—one from each sampled household.[10] The August 1987 NHIS survey showed that 69% of adults answered "definitely true" that a pregnant woman can pass the AIDS virus to her baby, and 27% thought it "very unlikely" or "definitely not possible" to transmit the AIDS virus by sharing eating utensils; these responses improved to 80% and 45%, respectively, in 1988. The results from our survey showed even higher levels of knowledge, with 96% answering "yes" on whether the AIDS virus can be transmitted from an infected pregnant woman to her baby, and 87% answering "no" in response to whether the AIDS virus can be transmitted by sharing eating utensils. The national multimedia public awareness campaign has made an enormous impact on the general population's knowledge of AIDS. However, our results showed that only 39% of respondents realized that having an STD could increase the risk of acquiring HIV infection, and 8% were not aware that persons could become infected through heterosexual contact with an infected person. These responses confirm the importance of educating this high risk population.

Condom use has been recommended by many public health professionals, including the surgeon general of the United State Public Health Service,[11] as one of the most effective ways to prevent the transmission of HIV. Our survey findings, that only 10% of patients always used condoms, and the NHIS survey, which showed no change in perceived effectiveness of condoms in preventing HIV transmission between 1987 and 1988, indicate the need for increased promotion and education on condom use. Condoms should be made more widely available through health care settings such as STD and family planning clinics. The behavior changes among homosexual men which led to decreased rates of syphilis and gonorrhea have not yet been seen in the heterosexual population.[12-14] In New York City, syphilis has increased more than 100% during 1987 compared with 1986.[15] As sites frequented by the high risk heterosexual population, STD clinics have to increase counseling activities to reduce HIV infection, as well as syphilis and gonorrhea.

An average of 69% of STD clinic patients obtained their AIDS information from newspapers, magazines, television, or radio; and 30% of them acquired information from medical or clinical staff. In response to information obtained from these surveys, the New York State Department of Health developed a videotape on HIV and AIDS which is shown to STD clinic patients while they are in the clinic. This videotape, entitled "Eddie's Story," was produced in a "soap opera" style format and focuses on demonstrations of the transfer of information between friends. Several points were stressed throughout the video: limiting the number of sexual partners is a good idea; condoms should be used for all sexual encounters; and the same behavior that puts a person at risk for an STD also creates a risk for HIV infection. It is our intent to assess AIDS knowledge and the changes in behavior before and after the use of this video.

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AIDS education: Evaluation of school and worksite based presentations

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ABSTRACT. From 1986 through mid-1988, the Westchester County (NY) Health Department made AIDS education presentations upon request to junior and senior high school students and to adult educators and supervisors. In 1987, an evaluation component was added to measure the effectiveness of the presentations in increasing knowledge on AIDS-related topics. Questionnaires were field tested in a pilot study. During 1988, pre- and post-tests were administered 10–14 days before and after each scheduled presentation. Multivariate linear regression on the 847 pre- and 574 post-tests that were returned shows a statistically significant pre-test/post-test difference, controlling for test type, age, and gender, in both overall score ($t = 9.00$, $p < 0.0001$) and in total number of questions answered rather than left blank ($t = 6.27$, $p < 0.0001$). In logistic analyses controlling for test type, age, and gender, the greatest post-test improvements were in questions about differences between AIDS and HIV-related disorders (then called ARC) (adjusted odds ratio, 1.86; 95% confidence limits, 1.65, 2.10); AIDS and drug use (1.73; 1.40, 2.14); sexual practices and AIDS (1.64; 1.40, 1.91); and the definition of AIDS (1.52; 1.29, 1.81). There was also significant post-test improvement on questions dealing with risks in social proximity to persons with AIDS, virus transmission by persons with AIDS, and differences between AIDS and HIV. In comparing audiences to controls, multivariate linear regressions on post-test results (adjusted for test type, age, and taking a pre-test) show statistically significant differences in both overall score ($t = 1.98$, $p < 0.05$) and in total number of questions answered rather than left blank ($t = 3.53$, $p = 0.001$). Based on these results, it appears that the presentation increased audience knowledge of AIDS topics, particularly in certain technical areas. This finding is consistent with published evaluations of other AIDS education programs.

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In the period from 1986 through 1988, AIDS education presentations were given in schools and worksites in Westchester County (just north of the acquired immunodeficiency syndrome (AIDS) epicenter of New York City) by the Westchester County Department of Health. Sessions were given by invitation in more than 40 sites countywide, with a total audience of about 15,000. This paper presents the results of an evaluation of the presentations given in Westchester County during 1988.

A full 20% of all people with AIDS are in their 20s,[1] suggesting high rates of infection with the human immunodeficiency virus (HIV) during the teenage years. This is not surprising, given teenagers' experimentation with sex and drugs,[2] their high levels of misinformation about AIDS, and their general misperceptions about their own personal risks of infection.[3,4] This potentially deadly combination of risk factors has made AIDS education for teenagers (and even younger people) a primary AIDS prevention activity, as a means of improving awareness and knowledge of AIDS related issues, creating more accurate perceptions of personal risk, and, in turn, promoting behavioral change to reduce personal risk.

In addition to educating young people with the ultimate hope of behavior modification and AIDS prevention, AIDS education for teachers and on-the-job supervisors has become crucial to effective guidance of students and employees in dealing with AIDS related issues. For this population, knowledge enhancement is a primary goal of AIDS education.

In the United States, the content of AIDS education courses, like that of sex education courses, depends on state education agencies and local school districts. A recent national survey found that 87% of responding school districts had AIDS curricula, all dating from 1985 or later.[5] Nearly all curricula (96%) covered the risks of intercourse for teenagers. Other topics included abstinence as the best AIDS-preventive measure (91%), abstinence as the only AIDS-preventive measure (9%), and the use of condoms to prevent AIDS (86%).

Only a few previous studies have attempted to measure the effectiveness (in terms of changing knowledge and/or behavior) of local AIDS and sex education programs.[2,6–8] Two of these studies[2,7] concentrated on AIDS education and showed that programs impacted on certain areas of knowledge of AIDS. The Centers for Disease Control have set up a school-based monitoring program which will begin periodically surveying students in the areas of greatest HIV prevalence.[9] Results of the first survey (spring 1988) have not been published yet.

The purposes of the Westchester County AIDS education evaluation were to measure the effectiveness of an AIDS education presentation to students, teachers, and employees of varying ages in increasing knowledge of AIDS-related topics, and to pinpoint topics that appeared to be less well understood, so that more attention could be focused on them in future presentations.

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METHODS

Presentation. AIDS education presentations in the schools were sometimes previewed by the school's Board of Education and Parent Teacher Association before being seen by students. Parents were given the option of having their child not attend the presentation.

The introduction to the presentations began with an overview of the Westchester County Department of Health AIDS unit, its scope and function, and a discussion of the credentials of the AIDS educator, including his experience as a family therapist with AIDS patients and their families. The AIDS acronym was spelled out and a case definition was given. The lecture included topics such as AIDS statistics; modes of transmission of the virus (and how it can not be transmitted); an overview of the immune system and the roles of T-cells, B-cells, and white blood cells; definition and examples of opportunistic infection; and the recognition of the onset of the epidemic in the United States in the early 1980s. The differences between HIV, HIV-related disorders (then called AIDS-related complex, or ARC), and AIDS were categorized; the blood tests for AIDS were described; confidentiality issues were presented; and prognosis and treatment discussed.

A question and answer session for high school students and adults preceded a showing of "The AIDS Movie."[10] For junior high school students, blank cards were distributed after the lecture (so questions could be asked with total anonymity). The students next saw a synchronized slide show/audio tape, "AIDS Alert."[11] The cards were then collected and the question and answer session followed. The presentation generally lasted about one and a quarter hours.

Evaluation Design. In the spring of 1987, a first attempt at an evaluation component was added to the AIDS education program, with pre-tests administered immediately before and identical post-tests administered immediately after the presentation. When it was decided to undertake a more rigorous evaluation of program effectiveness, the evaluation time frame was changed to provide for a two-week lapse both before and after the presentation. The questionnaire was also revised, introducing a true/false and an equivalent (cognate) multiple choice question for each topic, with the intention of reducing the likelihood of improvement in post-test scores due to repeated exposure to identical questions. In addition, a pilot study was designed and implemented to assess the comparability of cognate questions, and to determine whether merely taking a pre-test would affect post-test performance. The latter determination was necessary because it was known in advance that it would not be possible to have an equivalent control group for each group that attended the presentation. Results of the pilot showed that individuals who did not attend the presentation did not do appreciably better on a second test (given several weeks later) than on a first, allowing us to expect that pre-test/post-test differences identified during the evaluation were not attributable to the testing procedure itself.

As noted, pre-tests were administered 10–14 days before the presentation and post-tests 10–14 days afterward. To ensure participant anonymity, no attempt was made to match an individual's pre- and post-tests. Participants received, on a random basis, either a true-false or a multiple choice test. Participants were told that if they did not know the answer to a particular question they were to leave answers blank rather than guess blindly. The post-test had two additional questions: "Did you take the pre-test?" and "Did you see the presentation?" The test groups included seventh grade students at a Westchester County junior high school, 10th through 12th grade students at a Westchester County high school, supervisory employees of the Westchester County Department of Social Services, and teachers at the Northern Westchester Board of Cooperative Education Services (BOCES).

Because of differences in the size of presentation groups and the accessibility of participants before and after the presentation, there were minor intergroup differences in test administration, as well as in the level of participation in the evaluation tests.

Statistical Analyses. Pearson's chi-square tests were used to compare answers on individual test items, both within and between groups. Mean scores and mean number of questions answered as opposed to those left blank were compared between groups with Student's t-tests.

To control for confounding factors (age, gender, and question type), pre-test/post-test differences for individual questions, for total score, and for total number of questions answered were analyzed with multivariate procedures: logistic analyses for individual questions and linear regressions for total score and total number of questions answered.

As a number of individuals indicated on the post-tests that they had not seen the presentation and/or had not taken the pre-test, it was possible to test for the effects of both the pre-test and the presentation on post-test scores.

RESULTS

The analysis included 847 pre-tests and 574 post-tests. Table I shows the distribution of participants by age, gender, and test group.

TABLE I. Distribution of Study Participants by Test Group, Gender, and Age

Test Group	Gender	Age to 29	30–39	40–49	50+	Unknown	Total
DSS	Male	4	15	20	12	1	52
	Female	21	50	45	35	22	173
	Unknown	1	5	0	2	29	37
	Total	26	70	65	49	52	262
BOCES	Male	1	13	26	19	1	60
	Female	27	60	69	44	16	216
	Unknown	2	4	0	2	13	21
	Total	30	77	95	65	30	297

Test Group	Gender	Age 10–14	15–19	Unknown	Total
High School	Male	3	142	3	148
	Female	1	170	4	175
	Unknown	0	18	17	35
	Total	4	330	24	358
Junior High	Male	247	0	4	251
	Female	223	0	6	229
	Unknown	12	0	12	24
	Total	482	0	22	504

TABLE II. Pre-test/Post-test Differences in Scores, by Age Group, Number and Percentage of Correct Answers

Post-test Question	Adults		High School		Junior High	
	Pre-test (N = 368)	Post-test (N = 191)	Pre-test (N = 229)	Post-test (N = 129)	Pre-test (N = 250)	Post-test (N = 254)
1 (AIDS definition)	320 87.0%	178 93.2%*	189 82.5%	119 92.2%*	175 70.0%	224 88.2%*
2 (AIDS/HIV differences)	257 69.8%	147 77.0%	146 63.8%	73 56.6%	98 39.2%	169 66.5%*
3 (AIDS/ARC differences)	174 47.3%	135 70.7%*	96 41.9%	92 71.3%*	91 36.4%	153 60.2%*
4 (Sexual transmission)	359 97.6%	186 97.4%	211 92.1%	124 96.1%	210 84.0%	204 80.3%
5 (Risks in proximity)	346 94.0%	187 97.9%*	216 94.3%	128 99.2%*	234 93.6%	241 94.9%
6 (HIV + persons)	319 86.7%	154 80.6%	183 79.9%	95 73.6%	166 66.4%	180 70.9%
7 (Persons with AIDS)	348 94.6%	185 96.9%	190 83.0%	116 89.9%	160 64.0%	199 78.3%*
8 (Drug use and AIDS)	348 94.6%	188 98.4%	207 90.4%	123 95.3%	184 73.6%	233 91.7%*
9 (Needle use and AIDS)	342 92.9%	176 92.1%	183 79.9%	107 82.9%	128 51.2%	130 51.2%
10 (Sex and AIDS)	314 85.3%	159 83.2%	184 80.3%	103 79.8%	149 59.6%	232 91.3%*
Mean total score	8.50 ±1.41	8.87 ±1.29	7.88 ±1.93	8.37 ±1.52*	6.38 ±2.09	7.74 ±1.68*
No. of questions answered	9.47 ±1.12	9.74 ±0.85*	9.47 ±1.19	9.91 ±0.49*	9.13 ±1.40	9.81 ±0.73*

* Significant pre/post difference, $p \leq 0.05$, using Pearson's χ^2 test for individual scores; Student's t test for mean total score and number of questions answered (vs left blank).

In univariate analyses, post-test overall scores were significantly better than pre-test scores (Table II). Comparison by individual questions showed significantly more correct answers on seven post-test questions and one pre-test question (Table III). In separate univariate analyses, age, gender, and question type also had significant effects on scores (results not shown).

Multiple logistic analyses on individual questions controlled for age, gender, and question type. The adjusted odds ratios (OR) for post-/pre-improvement are consistent with the univariate results (Table III). The greatest post-test over pre-test improvements were seen in questions about differences between AIDS and ARC (adjusted odds ratio 1.86; 95% confidence limits, 1.65, 2.10); AIDS and drug use (1.73; 1.40, 2.14); sexual practices and AIDS (1.64; 1.40, 1.91); and the definition of AIDS (1.52; 1.29, 1.81). There was also significant post-test improvement on questions dealing with risks in social proximity to persons with AIDS, virus transmission by persons with AIDS, and differences between AIDS and HIV.

Multivariate linear regression showed a statistically significant post-test/pre-test effect on overall score, controlling for test type, age, and gender ($t = 9.00$, $p < 0.0001$).

A separate analysis considered the odds of a question being answered correctly or incorrectly rather than left blank. For every question, the odds ratio of answering the question was greater for post-tests, in both unadjusted and adjusted comparisons (Table IV). A multivariate linear regression on the total number of questions answered showed a statistically significant post-test/pre-test effect, controlling for test type, age, and gender ($t = 6.27$, $p < 0.0001$).

Among the 574 individuals who returned a post-test, 456 (79.4%) answered "yes" to the question: "Did you see the AIDS presentation?" The others, who either answered "no" (N = 48) or did not answer the question (N = 70), were analyzed as a control group for evaluating the effects of the presentation. Table V presents the unadjusted odds ratios of correct post-test answers, for audiences as opposed to others. The likelihood of correct answers was also affected by test type, age, and having taken the pre-test (results not shown). In addition, for seven of ten questions, blanks were more likely on the pre-test. Controlling for test type, age, and having taken the pre-test, the adjusted odds ratios of correct answers (Table V)

TABLE III. Post-test versus Pre-test—Odds Ratio of Correct Answer

Question	Unadjusted Odds Ratio	Adjusted Odds Ratio*
1 (AIDS definition)	2.34[†] (1.68, 3.26)	1.52[†] (1.29, 1.81)
2 (AIDS/HIV differences)	1.45[†] (1.16, 1.81)	1.38[†] (1.23, 1.56)
3 (AIDS/ARC differences)	2.64[†] (2.12, 3.29)	1.86[†] (1.65, 2.10)
4 (Sexual transmission)	0.74 (0.51, 1.06)	0.93 (0.77, 1.12)
5 (Risks in proximity)	1.98[†] (1.14, 3.42)	1.40[†] (1.06, 1.85)
6 (HIV + persons)	0.79 (0.62, 1.02)	0.99 (0.86, 1.14)
7 (Persons with AIDS)	1.44[†] (1.07, 1.95)	1.39[†] (1.18, 1.64)
8 (Drug use and AIDS)	2.65[†] (1.74, 4.03)	1.73[†] (1.40, 2.14)
9 (Needle use and AIDS)	0.76[†] (0.60, 0.97)	0.97 (0.85, 1.10)
10 (Sex and AIDS)	1.91[†] (1.44, 2.54)	1.64[†] (1.40, 1.91)

* Based on coefficient of parameter in multivariate logistic regression, adjusting for test type (true/false or multiple choice), gender (male, female, or sex unknown), and age level (10–29, 30–49, or 50+ or unknown).

[†] 95% confidence limits (in parentheses) do not include 1.0.

TABLE IV. Post-test versus Pre-test—Odds Ratio of Answering (Versus Leaving Answer Blank)

Question	Unadjusted Odds Ratio	Adjusted Odds Ratio*
1 (AIDS definition)	5.08[†] (1.51, 17.04)	2.21[†] (1.20, 4.07)
2 (AIDS/HIV differences)	7.12[†] (4.06, 12.50)	2.88[†] (2.17, 3.83)
3 (AIDS/ARC differences)	5.34[†] (3.60, 7.94)	2.34[†] (1.91, 2.85)
4 (Sexual transmission)	1.91 (0.69, 5.34)	1.47 (0.88, 2.48)
5 (Risks in proximity)	2.97 (0.84, 10.46)	1.77 (0.94, 3.33)
6 (HIV + persons)	2.56[†] (1.46, 4.51)	1.80[†] (1.35, 2.40)
7 (Persons with AIDS)	1.62 (0.86, 3.06)	1.37 (0.99, 1.89)
8 (Drug use and AIDS)	2.17 (0.86, 5.47)	1.57 (0.98, 2.50)
9 (Needle use and AIDS)	2.51[†] (1.14, 5.53)	1.72[†] (1.15, 2.56)
10 (Sex and AIDS)	1.37 (0.66, 2.84)	1.24 (0.86, 1.81)

* Based on coefficient of parameter in multivariate logistic regression, adjusting for test type (true/false or multiple choice), gender (male, female, or sex unknown), and age level (10–29, 30–49, or 50+ or unknown).

[†] 95% confidence limits (in parentheses) do not include 1.0.

are comparable with the unadjusted, and show even stronger association with viewing of the presentation.

Multivariate linear regressions on post-test results, controlling for test type, age, and having taken the pre-test, showed statistically significant differences for audiences versus other participants, in both overall score ($t = 1.98, p < 0.05$) and in total number of questions answered rather than left blank ($t = 3.53, p < 0.001$).

TABLE V. Odds Ratio of Correct Post-test Answer for Those Seeing the Presentation versus Controls

Question	Unadjusted Odds Ratio	Adjusted Odds Ratio*
1 (AIDS definition)	0.77 (0.37, 1.63)	1.00 (0.61, 1.65)
2 (AIDS/HIV differences)	1.83[†] (1.21, 2.77)	1.88[†] (1.36, 2.59)
3 (AIDS/ARC differences)	1.33 (0.87, 2.02)	1.67[†] (1.21, 2.32)
4 (Sexual transmission)	0.48 (0.21, 1.08)	0.73 (0.39, 1.39)
5 (Risks in proximity)	2.55 (0.97, 6.73)	2.72[†] (1.42, 5.19)
6 (HIV + persons)	1.01 (0.29, 1.61)	1.15 (0.82, 1.62)
7 (Persons with AIDS)	1.40 (0.79, 2.46)	1.59 (0.99, 2.57)
8 (Drug use and AIDS)	1.43 (0.62, 3.31)	1.65 (0.93, 2.93)
9 (Needle use and AIDS)	0.71 (0.44, 1.15)	0.75 (0.48, 1.16)
10 (Sex and AIDS)	1.46 (0.84, 2.51)	0.98 (0.66, 1.46)

* Based on coefficient of parameter in multivariate logistic regression, adjusting for test type (true/false or multiple choice), having taken the pretest, and age level (10–29, 30–49, or 50+ or unknown).

[†] 95% confidence limits (in parentheses) do not include 1.0.

Parallel analyses, excluding individuals who did not answer the question about having seen the presentations, gave results in the same direction, although they were not statistically significant due to smaller numbers (results not shown).

DISCUSSION

The overall effectiveness of an AIDS curriculum depends not only on content, but on how it is taught at the classroom level,[12] how much of the information is remembered by the teenagers, and how much of that knowledge affects behavior.[4,13] Our evaluation focused on changes in knowledge only, and it was to some extent compromised by the high level of pre-presentation knowledge and/or some of our test questions being too easy. This situation obtained for adult responses to our questions 4, sexual transmission of AIDS; 7, persons with AIDS; 8, drug use and AIDS; and 9, needle use and AIDS (Table II).

The attrition of post-test participants ($N = 574$ compared with 847 pre-test participants) could have biased our results if either those who would have done poorly, or those who would have done better, were less likely to participate in the post-test. As participants were neither informed of their pre-test scores nor told what the correct answers were after the pre-test, and as the school children were informed explicitly that these tests did not "count," and that they themselves were not being tested or graded (ensured by anonymity), we think it likely that "dropouts" were randomly distributed in terms of their pre-test scores.

Ideally, our evaluation would have included controls, randomly chosen and excluded from the presentation. It was not possible to follow such a procedure. However, because some students and employees missed the presentation and/or the pre-test, we had a small quasi-control group with which to gauge the effectiveness of the presentation in increasing AIDS knowledge. Adjusting for test type, age, taking a pretest, and having seen the presentation had a significant effect on total post-test score and on total number of post-test questions answered instead of being left blank. Yet it is possible that those who missed the presentation were in general more sheltered than those who saw the presentation, creating a spuriously inflated result.

While differences in methodology and the rapid changes in general knowledge of AIDS over time make it difficult to compare our evaluation with others, inner-city (Chicago) junior high students tested in 1982–1983[6] (by their classroom teachers before, at five days, and 8–12 weeks after two hour-long presentations on sex education), showed an increase in knowledge comparable to that found in Westchester. The mean Chicago scores were 52% correct on the pretest, 69% on the post-test; mean Westchester junior high scores were 64% and 77%. Also comparable to our results were those from a 1985 study in San Francisco by DiClemente and co-workers,[7] in which a 30-question survey of student knowledge of AIDS was administered to more than 1,000 students ages 14–18. Results for all students showed substantial misinformation about modes of transmission of the AIDS virus and about disease course and treatment.[7] (The San Francisco survey was not repeated in the same student population to evaluate any changes in knowledge.) Likewise, a pre-test/post-test study among 11th grade Seattle area students[2] using different methods than ours showed similar results; ie, a significant gain from pre-test to post-test in knowledge of the definition of AIDS and how AIDS is (and is not) transmitted.

Particularly in light of this consistency across studies despite varying methods, our results suggest to us the following: first, that our presentation did increase audience knowledge of AIDS (especially for younger audiences); and second, that both our students and adults were, in general, knowledge-deficient in technical over behavioral areas. We therefore recommend emphasizing natural history, disease mechanism, and mode of transmission in AIDS education presentations to promote better technical understanding of AIDS. This enhanced understanding would be especially valuable to supervisors and teachers in their professional roles. In terms of students, however, where reduction of personal risk is the issue, it remains unclear as to whether enhanced understanding generally leads to behavior change. While such an assessment (ie, one of behavior change in relation to an intervention) would be costly, time consuming, and difficult to conduct rigorously, it is imperative that resources be channeled in this direction.

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AIDS behind bars

Epidemiology of New York State prison inmate cases, 1980–1988

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ABSTRACT. During the last five years, AIDS has become the preeminent health care problem in New York State correctional facilities. Through December 31, 1988, 915 cases of AIDS had been diagnosed among inmates. This represented approximately 1% of the cumulative AIDS cases in the United States, 4% of those in New York State, and 40% of those reported in state correctional systems nationwide. An analysis of epidemiologic data on these cases showed an annual increase in cases from 3 in 1981 to 227 in 1988, with an incidence greater than 400 per 100,000 inmates per year over the past four years. While most cases occurred in males (96%), females had the same high incidence rates (compared to the general population, in which female rates are one-eighth of males). Forty-seven percent of infected inmates were Hispanic, 38% black, and 13% white. *Pneumocystis carinii* pneumonia was the most common diagnosis (65%), while Kaposi's sarcoma was rare (3%). Previous intravenous drug use has been the major risk factor, seen in 95% of cases. A comparison of 54 inmate AIDS cases with 107 matched and 196 unmatched controls showed that inmates in whom AIDS developed had significantly lower white blood cell counts on entry into prison, lower hematocrits and serum albumin levels, and higher serum glutamic oxaloacetic transaminase and globulin counts. Through July 1989, 643 (70%) of these 915 inmates had died of AIDS, and HIV infection and AIDS account for 68% of recent inmate deaths.

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Since 1982, when the first seven cases of opportunistic infections in prisoners were described,[1] the acquired immunodeficiency syndrome (AIDS) has become a major health problem among inmates of New York State correc-

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tional facilities. Early reports concerning this population described the clinical features of the illness and linked most cases to intravenous drug use prior to incarceration.[2,3] A 1984 study showed inmates with AIDS to have low white blood cell counts on admission to the prison system, suggesting that HIV infection was acquired prior to incarceration, and that the incubation period from infection to symptoms was quite prolonged.[4] More recently, data have been presented on the association between tuberculosis and AIDS in the New York State prison inmate population.[5,6]

Outside of New York State, there has also been an increasing incidence of AIDS among inmates in local, state, federal, and international penal systems.[7-10] This report summarizes the epidemiology, mortality, and entrance laboratory data on the large number of AIDS cases among inmates in New York State correctional facilities, as a reference for other systems that have not been as severely affected. It summarizes the descriptive epidemiologic data on New York State AIDS cases among inmates, which had been diagnosed through December 31, 1988, and had been reported and confirmed through July 31, 1989. The characteristics of these cases were compared to those of AIDS cases in the US and in New York State, as reported through 1988. In addition, data are included from epidemiologic studies on mortality among inmates over time, and on the use of entry laboratory data as a predictor of AIDS development. Information on the association between AIDS and tuberculosis (TB) in inmates, which has been reported separately,[5,6] is also updated.

METHODS

The New York State correctional system is the third largest in the country, with 57 facilities housing 44,637 inmates as of December 31, 1988. Most facilities are in upstate New York, with censuses ranging from 50 to 2,600 per facility. Characteristics of inmates in the correctional system were determined by analyzing computer records of inmates incarcerated as of January 1, 1988.

Since 1982, the New York State Department of Health and Department of Correctional Services have conducted active AIDS surveillance, maintained case registries, and collected epidemiologic data on inmates in whom AIDS developed while in the state correctional system.

The inmate AIDS surveillance system includes state mandated reporting of case information to the Department of Health in the same format as required for other cases of AIDS in New York State. Independent reporting of cases by correctional facility to the Department of Correctional Services allowed for cross checking and collection of additional information. Data collected on the registries included demographic information, risk factors, and laboratory and mortality data. All cases were reviewed to verify that AIDS had been diagnosed while the inmate was in the prison system, and that the case met definition criteria established by the Centers for Disease Control (CDC), as well as to ensure that data collection was as comprehensive as possible.

The epidemiologic study of mortality among inmates involved a systematic review of legally mandated prison death and autopsy data collected by the Department of Correctional Services over the past 11 years (1978-1988). Health department researchers periodically reviewed medical records and autopsy reports on these deaths to determine those that were directly related to HIV or AIDS. All HIV-related deaths met CDC AIDS criteria except for 23 cases in 1987 which were reported as AIDS/ARC cases by physicians, but lacked specific chart documentation to meet the strict case definition.

Case-Control Study. In an attempt to identify features of inmates at highest risk for developing AIDS during incarceration, a case-control study was performed comparing entry demographic, clinical, and laboratory data on inmates in whom AIDS later developed with those of control inmates. To be eligible for inclusion as a case, an inmate must have met the following criteria. AIDS must have been diagnosed while the inmate was incarcerated in the New York State correctional system with diagnosis prior to January 1984 and with a confirmed report before April 1984. Again, CDC diagnostic criteria were used as the basis for the diagnosis of AIDS. In addition, the inmate must have been free of AIDS prodromal or AIDS-related complex (ARC) symptoms at the time of the prison entry screening health evaluation. Of the 66 inmates in whom AIDS developed while in state correctional facilities during the study inclusion dates, 12 were excluded because they had ARC symptoms at the time of their entry health evaluation. This left 54 inmates who met the case definition and were included in the study.

Two groups of control inmates were compared to case inmates. The first control group, based on sample size considerations, was composed of 200 inmates randomly selected from the Department of Corrections records using inmate identification numbers: 196 of them had complete records available for analysis. All these inmate controls had entered the correctional system between January 1978 and January 1984, the same time period as all of the case inmates. The second group of control inmates was selected from the corrections department admission records on the basis of five matching criteria. Each case inmate was matched to control inmates of the same sex, age (± three years), race, date of entry into the state prison system (± two months), and history of drug use (without differentiation of whether it was intravenous or not) prior to incarceration. For one of the 54 case inmates, data on only a single matched control was obtained, resulting in a total of 107 matched control inmates.

The screening health evaluation performed on all entering inmates included a history and physical examination. Routine laboratory studies included a complete blood cell count (CBC), serum chemistries (including total protein, albumin, and globulin), liver function tests, and a serologic test for syphilis. HIV antibody testing was not done at that time and has since been conducted only for medical reasons, because of concerns over safety and discrimination in this population. Case-control comparisons of these demographic and laboratory data were made using t-tests and logistic regression (BMDP package). White blood cell count (WBC), hematocrit, serum globulin, serum albumin, serum glutamic oxaloacetic transaminase level (SGOT), and place of residence (New York City or other) were compared for cases and matched controls. Logarithm transformation was applied to laboratory results, and race and county were dichotomized. For the comparison of case inmates and random controls, age, race, and history of drug use were also compared.

RESULTS

A review of inmate data showed that the inmate population had grown steadily from 16,074 on December 31, 1975, to 44,637 on December 31, 1988. On January 1, 1988, computerized inmate data showed that most inmates had originated from New York City: approximately 71% had been incarcerated from New York City and 12% from the four counties (Nassau, Suffolk, Westchester, and Rockland) close to New York City. Most inmates were males (96.4%); only 3.6% were females. The mean age was 30.5 years, the median age 29.4 years, and 49% were 20-29 years of age. The racial/ethnic distribution showed 50% to be black, 30% Hispanic and 20% white. Twenty-two percent of inmates had drug-related sentences: this occurred more commonly in Hispanics (39.2%) than in blacks (15.9%) and whites (14.3%) ($\chi^2 = 2732, p < 0.001$). Narcotic-related drug use was reported for 26% of inmates—significantly higher in Hispanics (36.9%) than in blacks (21.1%) or whites (24.1%) ($\chi^2 = 991, p < 0.001$). Heroin-related drug use was reported for 17%: Hispanics, 29.6%; blacks, 11.5%; and whites, 13.6% ($\chi^2 = 1825, p < 0.001$). Compared with males, females had significantly higher rates of drug-related sentences (34% vs 22%) ($\chi^2 = 120, p < 0.001$), of narcotic use (33% vs 26%) ($\chi^2 = 39.5, p < 0.001$), and of heroin use (29% vs 17%) ($\chi^2 = 155, p < 0.001$).

Through December 31, 1988, 915 cases of AIDS had been diagnosed among inmates in the New York State correctional system with confirmed reports received by July 31, 1989. During this period, inmates contributed 4% of the New York State and 25% of the upstate New York AIDS case burden. The epidemic curve of AIDS cases in inmates is shown in Figure 1 and includes cases, deaths, and incidence rates by year. The number of cases increased from three in 1981 to 227 in 1988. Incidence rates were 478, 574, and 531 (1988 data are incomplete) per 100,000 inmates per year for 1986, 1987, and 1988, respectively. The cumulative incidence rate for the average prison inmate population has been 2,738 per 100,000. Throughout the study period, 643 (70%) of inmates with AIDS were known to have died, with few long term survivors.

Cases have ranged in age from 18 to 64 years with a mean of 34.5 and median of 33 years. Eight hundred eighty (96.2%) were male, 35 (3.8%) were female (Table I). Compared to the US AIDS case population, inmates with AIDS are younger (79% less than 40 years of age vs 68%) ($\chi^2 = 43.4, p = < 0.001$) and are more often male (96% vs 91%) ($\chi^2 = 25.6, p < 0.001$). Larger differences occur in race and ethnic backgrounds: 13% vs 58% white, 38% vs 26% black, and 47% vs 15% Hispanic for the inmate vs US AIDS case population, respectively ($\chi^2 = 985, p < 0.001$). Inmates have also differed from the US AIDS population in terms of diagnosis and risk factors. Inmates more commonly have *Pneumocystis carinii* pneumonia (PCP) (66%), and Kaposi's sarcoma has been relatively rare (3%). This compares with 60% and 9% for the US AIDS cases, respectively. Intravenous drug use has been a risk factor among 95% of inmates compared to 27% of US AIDS cases ($\chi^2 = 2050, p < 0.001$). Only 10% of inmates have given a history of homosexuality/bisexuality, and 71% of these persons had used intravenous drugs. All 35 (100%) female inmates had IV drug use as their risk factor.

Although not as dramatic, similar differences were seen when comparing the 915 New York State inmate AIDS cases with the 20,815 New York State non-inmate adult cases diagnosed and reported within the same time period (Table II). For comparison, among the non-inmate group, 87% of cases were male, 67% were less than 40 years of age, 41% were white, 33% black, and 25% Hispanic. Leading risk factors for non-inmate cases were homosexuality/bisexuality for 49% of cases, intravenous drug use for 35%, heterosexual contact for 5%, and both IV drug use and homosexuality/bisexuality for 4%. As expected, a comparison of prison inmate AIDS cases (where IV drug use is a risk factor for 95% of cases) with non-inmate New York State IV drug use-related cases showed fewer dissimilarities. Among the latter, 80% of cases were male, and 75% were less than 40 years of age with a mean age of 36 years. Nineteen percent were white, 45% black, and 35% Hispanic.

Compared to the general inmate population, a higher percentage of inmates with AIDS came from New York City (87% vs 71%). Gender findings were similar: 3.8% of AIDS cases were female versus 3.6% of the inmate population (Table II). Inmates with AIDS were older than the general inmate population: AIDS cases had a mean age of 34.4 years with 76% more than or equal to 30 years of age, compared with 30.5 years and 46%, respectively, among the general inmate population ($\chi^2 = 199, p > 0.001$ for comparison of numbers ≥ 30 years of age). There were also statistically significant racial/ethnic differences seen between inmates with AIDS versus the general inmate population: 47% vs 30% Hispanic, 38% vs 50% black, and 13% vs 20% white, respectively ($\chi^2 = 155, p > 0.001$).

Within New York State, cumulative AIDS case rates per 100,000 population were much higher for inmates than for non-inmates for all demographic variables (Table II). Of note, however, male and female inmates had nearly equal AIDS case rates, with a male-to-female rate ratio of 0.9, while males in the general population had much higher rates and a male-to-female rate ratio of 7.8. By age, using the 20–29-year age group as reference, inmates had much

TABLE I. Gender, Age, Race, Diagnosis, and Risk Factors of New York State Inmate vs US AIDS Cases

	NY State Inmates* (N = 915)		US† (N = 81,418)	
Characteristic	**No.**	**%**	**No.**	**%**
Gender				
Male	880	96	74,435	91
Female	35	4	6,983	9
Age (yr)				
13–19	2	<1	335	<1
20–29	213	23	17,120	21
30–39	504	55	38,194	47
40–49	171	19	17,348	21
>50	25	3	8,421	10
Race/ethnicity				
White	122	13	47,307	58
Black	351	38	21,222	26
Hispanic	432	47	12,133	15
Other/unknown	10	1	567	1
Diagnosis†				
Pneumocystis carinii pneumonia	600	66	49,517	60
Other opportunistic diseases	290	31	25,916	31
Kaposi's sarcoma	25	3	7,331	9
Risk factor‡				
Homosexual/bisexual male	25	3	50,325	62
Intravenous drug abuser	802	88	16,151	20
Homosexual/bisexual & IV	63	7	5,874	7
Hemophilia/coagulation disorder	0	0	773	1
Heterosexual cases	3	<1	3,589	4
Transfusion, blood/components	2	<1	2,004	2
Other	20	2	2,662	3

* New York State cases diagnosed through December 31, 1988, with confirmed reports received through July 31, 1989.
† United States adult cases reported through January 2, 1989. Diagnostic figures included children. Race/ethnicity included 189 patients with unknown status.
‡ All 35 female inmates had IV drug use as their risk factor.

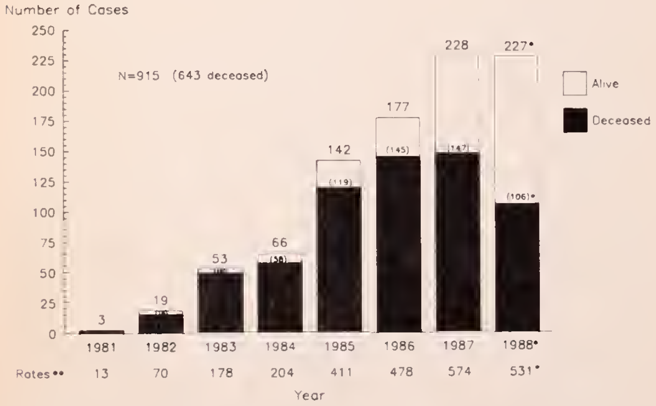
TABLE II. Comparison of New York State Inmate vs Non-Inmate AIDS Cases: Demographic Characteristics, Cumulative Population Rates and Rate Ratios

	Inmates*				Non-Inmates*			
Characteristic	% Among Inmates with AIDS	% Among Total Inmate Pop	Cumulative Rate/ 100,000	Rate Ratio	% Among Non-Inmates with AIDS	% Among Total NYS Pop	Cumulative Rate/ 100,000	Rate Ratio
Gender								
Male	96.2	96.4	2732	0.9	86.9	46.0	311	7.8
Female	3.8	3.6	2909	1.0	13.1	54.0	40	1.0
Age (yr)								
13–19	0.2	4.1	145	—	0.2	—	—	—
20–29	23.3	49.2	1295	1.0	17.7	24.1	121	1.0
30–39	55.1	32.6	4627	3.6	48.7	20.1	399	3.3
40–49	18.7	10.7	4783	3.7	23.1	15.1	252	2.1
>50	2.7	3.4	2201	1.7	10.3	40.7	42	0.3
Race/ethnicity								
White	13.3	19.7	1854	1.0	41.4	78.2	87	1.0
Black	38.4	49.7	2114	1.1	32.7	11.8	459	5.3
Hispanic	47.2	30.0	4310	2.3	24.9	8.1	505	5.8
Other/unknown	1.1	0.6	4975	2.7	1.0	2.0	87	1.0

* New York State populations used were: Inmates with AIDS = 915; Total inmate population = 33,413 (mean census 1981-1988, distribution of characteristics based on 1987 census); Adult non-inmates with AIDS = 20,815; Total New York State adult population = 12,617,340 (mean 1980 and estimated 1986 census, distribution of characteristics based on 1980 census).

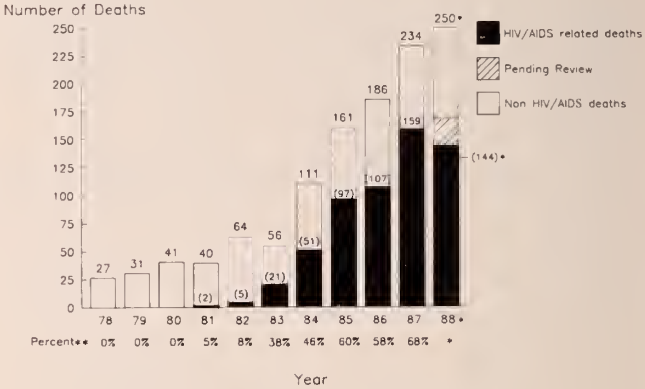
higher relative rates in the older age groups (highest rate in the 40–49-year age group) than the non-inmate cases (highest rate in the 30–39-year age group). By race/ethnicity, Hispanics had the highest rates per 100,000 among both inmates and non-inmates and the highest rate ratios compared to that of the whites (reference group). However, the rate and rate ratio differences for race were far less among inmates. While blacks and Hispanics contributed the greatest percentages of cases among inmates, when using rates to control for disproportionately large numbers of minorities in the inmate population, the rates were nearly equal among blacks and whites, with a rate ratio of 1.1.

Tuberculosis incidence rates among prison inmates have increased 773%, from 15 per 100,000 in 1976–1978 to 116 per 100,000 in 1986–1988 (incidence of 136 per 100,000 in 1988). Tuberculosis was common among inmates with AIDS, having occurred in 68 (7.4%) of the 915 cases. In addition, 65% of inmates with tuberculosis diagnosed during 1985–1988 had AIDS or were HIV seropositive. None of the tuberculosis cases tested for HIV had negative results. A high proportion of tuberculosis patients diagnosed between 1985 and 1988 had extrapulmonary manifestations (37%), and more than 80% had used street drugs.



• Case totals are those diagnosed through 1988 and reported through July 1989, 1988 data are incomplete
•• Incidence rates per 100,000 inmates per year.
••• Deceased cases are plotted by date of diagnosis rather than date of death

FIGURE 1. New York State inmate AIDS cases by year of diagnosis, 1981–1988.



•Provisional totals for 1988 (e.g. cause of death is pending for several inmates).
••Percent of deaths related to HIV/AIDS by year

FIGURE 2. Contribution of HIV/AIDS to mortality among prison inmates, 1978–1988.

Throughout the study period, 643 (70%) inmate AIDS cases died, with a cumulative mortality rate of 85% for those cases diagnosed prior to 1987 (Figure 1). Since the first two deaths related to AIDS occurred in 1981, AIDS has contributed to an ever-increasing percentage of total inmate mortality (Fig 2). (Note that the numbers differ somewhat between Figures 1 and 2 because of different reporting lag times and because some inmates included in Figure 2 who died of AIDS had been diagnosed prior to entry into the prison system and were thus not included in Figure 1.) While mortality rates for deaths unrelated to AIDS have remained relatively constant, mortality rates for HIV/AIDS per 100,000 inmates have increased steadily from 8.4 in 1981 to 400.0 in 1987. HIV/AIDS has become the leading cause of death among inmates, and was responsible for 68% of deaths in 1987.

The epidemiologic study comparing laboratory data demonstrated consistent differences between AIDS case inmates and both matched and random-control inmates. Inmates who later developed AIDS had significantly higher levels of serum globulin and SGOT and significantly lower white blood cell counts, hematocrit values, and serum albumin than both control groups (Table III). The most significant differences were seen in serum globulins (t = 4.45 and t = 7.18, for comparisons with matched and random controls, respec-

TABLE III. Selected Entry Laboratory Features of 54 New York State Inmates Who Later Developed AIDS vs Two Groups of Control Inmates[*]

Laboratory Value	Inmates with AIDS (N = 54) (Mean,[†] Mean ± 2SD)	Matched Control Inmates (N = 107) (Mean,[†] Mean ± 2SD)	t-Statistic	Random Control Inmates (N = 196) (Mean,[†] Mean ± 2SD)	t-Statistic
White blood cell count (cells/mm^3)	5129 (2692, 9772)	5998 (3436,10471)	−2.92[‡]	6012 (3556, 10162)	−3.42[‡]
Hematocrit (%)	44.26 (36.31, 53.95)	46.34 (40.55, 52.97)	−3.04[‡]	45.50 (39.08, 52.97)	−1.83
Serum glutamic oxaloacetic transaminase (KU)	40.55 (12.08, 136.14)	32.66 (10.67, 100.00)	2.04	26.24 (9.06, 76.03)	4.41[‡]
Albumin (g/dL)	4.19 (2.91, 6.03)	4.48 (3.95, 5.07)	−2.52[‡]	4.50 (3.97, 5.09)	−2.68[‡]
Globulin (g/dL)	3.74 (2.53, 5.53)	3.21 (2.13, 4.84)	4.45[‡]	2.99 (2.04, 4.39)	7.18[‡]

[*] All data log 10 transformed before analysis.

[†] Geometric mean.

[‡] $p < 0.05$ (Bonferroni test).

tively). While these laboratory measures indicated significant differences, the fact that the standard deviation limits widely overlapped indicated that these variables did not individually discriminate well between cases and controls. Using these variables, it would have been necessary to have followed 15% of inmates to have identified 85% of inmates who later developed AIDS.

In order to consider these variables and demographic variables simultaneously, an unmatched logistic regression comparing cases with unmatched controls was done. In this analysis, two cases with some missing laboratory results were eliminated. This simultaneous analysis showed that high serum globulin, low white blood counts, and advanced age were the best discriminators for determining in which inmates AIDS would develop. However, even the best simultaneous combination of these variables was not of general use, because of substantial overlap of the case population with the control groups.

DISCUSSION

The number of AIDS cases among inmates in New York State is substantial and is increasing. The 915 inmate cases diagnosed through December 31, 1988, was 1% of the US and 4% of the New York State total of AIDS cases, and was a larger number of cases than reported from all but 18 states in the US at that time.[11] During 1986–1988, the average annual incidence rate for AIDS among inmates was 529 per 100,000, and the 1981–1988 cumulative incidence rate was 2,738 per 100,000, giving this population group one of the highest incidence rates reported.[12,13] Through October 1987, approximately 40% of the AIDS inmate cases reported from US state correctional systems were from New York State.[8] (In comparison, New York State contributed 24% of the total US cases.[11]) The marked increase in AIDS inmate cases has been associated with a large increase in mortality due primarily to HIV/AIDS, which has now become the leading cause of death among inmates (responsible for 68% of deaths in 1987).

The reasons for the large number of cases of AIDS among inmates becomes clearer after reviewing the epidemiologic features of the cases. A history of intravenous drug use prior to incarceration, present in 95% of these cases, is clearly the major risk factor for HIV infection in this population. Previous drug use and/or drug-related crimes are common findings among inmates.[8,10] Among New York State inmates, 22% had drug-related sentences and 26% had reported narcotic use. Most inmates also originate from New York City, where IV drug users have been reported as having high rates of HIV infection.[14] Homosexuality or bisexuality was reported by 10% of AIDS inmate cases, but only 3% had this as their sole risk factor. While the potential role of prison homosexual activity in transmission of HIV has been debated, this type of activity has not been reported frequently by New York State inmates with AIDS. In addition, Kaposi's sarcoma, a diagnosis seen more commonly among homosexual cases, remains relatively rare in inmates.

The demographic characteristics of inmates with AIDS are slightly different from those of the general inmate population and are drastically different from AIDS cases seen nationally and in the New York State non-inmate population. These differences are likely related to the extremely large percentage of inmate AIDS cases (95%) with histories of IV drug use with over-representation of racial/ethnic minorities. Indeed a comparison with non-inmate IV drug use AIDS cases showed fewer dissimilarities. New York State inmates who develop AIDS originate primarily from New York City. They are older than the general inmate population but younger than the New York State and US AIDS case population. However, unlike the New York State and US cases, rates per 100,000 population are highest in the older, 40–49-year age group. Cases are primarily male, and 86% are from minority populations. While Hispanics comprise 30% of the inmate population, they contribute 47% of the AIDS cases. This discrepancy appears to be related to the higher proportion of drug use (narcotic use reported in 36.9% of Hispanics versus 21.1% in blacks and 24.0% in whites) and drug-associated crimes (39.2% among Hispanics versus 15.9% in blacks and 14.3% in whites) seen within the Hispanic inmate population. Tuberculosis has been reported in a high proportion of some IV drug using AIDS cases,[15] and was also commonly found in this population (present in 7.4% of AIDS cases). In addition, 65% of tuberculosis cases from 1985 to 1988 had AIDS or were HIV positive.

Surprisingly, females had rates of AIDS equal with males in this population. Again, this appears to be related to IV drug use and, indeed, females had significantly higher percentages of drug-related sentences (34% vs 22%), narcotic use (33% vs 26%), and heroin use (29% vs 17%) than males.

The case-control study of laboratory abnormalities at entry in inmates who later developed AIDS was conducted prior to the widespread availability of HIV serum antibody testing. The results were consistent with other reports that pre-symptomatic cases of AIDS had lower white blood cell counts and hematocrits, and higher SGOT and globulin levels than persons not incubating HIV infection.[16,17] These laboratory findings and demographic features were not sensitive and specific enough to identify inmates in whom clinical AIDS later developed, something which could be markedly enhanced by including results of HIV antibody testing. However, concerns over safety and discrimination have precluded HIV testing in this population except for medical reasons.

Of note, the matched control inmates had mean laboratory values (serum globulin, albumin, and SGOT) which fell between those found for case inmates and for random control inmates. We speculate that some control inmates were infected with HIV at the time of entry testing, especially the matched control inmates who were composed largely of drug users from New York City. These inmates probably included inmates in an even earlier pre-clinical phase of HIV infection. This potential misclassification may have served to diminish the discriminating ability of entrance laboratory tests. The case-control comparison does suggest that certain laboratory parameters may be useful in guiding selective medical HIV testing in a population in which universal screening is not practical or desirable. When combined with more specific T-cell abnormalities,[18-20] they may also help identify a subset of HIV-infected inmates at greatest risk for developing symptomatic AIDS and who might benefit from closer observation and follow-up and/or early initiation of antiviral therapy.

The increasing number of inmate AIDS cases has been a challenge to correctional system personnel. Although not described here, these confirmed cases represent only a fraction of the inmates who are infected with HIV or suffer from milder forms of HIV-related illness.[21] Difficult policy questions regarding HIV counseling, testing, and potential transmission have surfaced along with a number of health care issues.[7-10,22,23]

Pre-incarceration HIV-related risk factors were present among virtually all of the inmate AIDS cases. Furthermore, none of the inmates who were incarcerated prior to 1978 have developed AIDS. These two observations are consistent with the hypothesis that HIV infection among these prisoners occurs primarily prior to incarceration. However, in the absence of data from prospective serologic conversion studies, the extent to which HIV transmission may be taking place within the prison setting is unclear. In the meantime, correctional system inmates and staff need to be counseled and educated about HIV and its routes of transmission, with special emphasis on minimizing those types of risk activities that can facilitate transmission.

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A seroprevalence survey for human immunodeficiency virus antibody in mentally retarded adults

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ABSTRACT. The prevalence of human immunodeficiency virus (HIV) infection among adults who are mentally retarded is not known. Policies for those in residential settings are being established despite incomplete information. Knowledge regarding HIV seroprevalence would enable administrators to make more effective policy decisions concerning testing and HIV prevention.

Discarded sera from mentally retarded adults were anonymously tested for HIV antibody. Sera were collected from a health facility in Westchester County, NY, that provides care to developmentally disabled adults. After Identifications were removed, sera were coded and linked to demographic and clinical variables from hospital and laboratory records. Sera came from individuals living in both institutional and less restrictive community settings in metropolitan New York City and more distant locations in New York State, all of whom were seen by the above facility. No HIV antibody was detected in sera from 241 mentally retarded adults.

This study suggests that the prevalence of HIV antibody in mentally retarded adults is not high. Mandatory screening programs may not be appropriate for these individuals. Monies might be better spent on educational programs directed at AIDS prevention, and further development of ethical and safe policies for those who are mentally retarded.

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The prevalence of human immunodeficiency virus (HIV) infection in adults who are mentally retarded is not known. Impaired cognition and documented deficits in sex education[1] among those mentally retarded adults who are homosexually or heterosexually active may place them at risk for infection. The high incidence of hepatitis B among mentally retarded persons in residential settings[2–4] and the similarity in routes of transmission between hepatitis B and HIV[5–7] raise further concern about possible risk for this group. Increasing numbers of mentally retarded adults live in small community residences where most aspects of daily life are regulated by state agencies or other not-for-profit or private operators.[8] Knowledge regarding the seroprevalence of HIV infection among adults who are mentally retarded might allay unnecessary fears regarding HIV transmission within facilities, and may enhance policy making so that the rights of these individuals can be maximally protected.

The anonymous seroprevalence study method has been documented by others as an effective way to assess prevalence of HIV infection in defined populations.[9–12] We utilized this method, using discarded blood specimens, to look at the seroprevalence in mentally retarded adults who live in institutional as well as less restrictive community settings in metropolitan New York City and more distant locations in New York State.

METHODS

This study was performed at a clinic in Westchester County, NY, that is part of a facility providing care to mentally retarded people. The facility's primary functions include interdisciplinary training, outreach, and clinical services for the mentally retarded and developmentally delayed population. Clinical services include the provision of primary and secondary health care to those living in urban, rural, and suburban areas in Westchester County and seven mid-Hudson Valley counties, and to some residents of New York City. The patient population is approximately 57% white, 30% black, and 9% Hispanic.

Between January 1988 and April 1988, all blood samples that had been drawn for thyroid function studies from individuals 13 years of age or older were saved and assigned a code number. Corresponding patient charts were then used to obtain sex, age, address of residence, level of mental retardation, hepatitis B status, reason for thyroid function testing, and presence or absence of Down's syndrome. Labeling on charts indicating "hepatitis B" or "general" precautions was noted. At the time of this study, standard procedure at this institution was to code any sera suspected by the physician of being HIV infected with a "general" precautions label. These data were linked to blood specimens by the research code. Once all blood samples were collected and all demographic and other information was obtained from the charts, duplicates were discarded, and names and chart numbers of patients were destroyed. No chart review was performed for individuals living with their families. This study was approved by the institutional review board. Sera were tested for HIV antibody using a commercial ELISA (Du Pont kit, Wilmington, Del). Specimens repeatedly reactive by ELISA were tested by Western blot (Du Pont kit, Wilmington, Del). Western blots were considered positive when antibodies to p24 and/or gp41 were detected. Level of retardation was obtained from the medical evaluation done within the past year. Appropriate administrators at each facility provided descriptions of the residences. Settings were divided into the following categories:

- Families—those living with their families
- Foster care—one to six other mentally retarded adults living with a non-relative caretaker
- Apartment living—apartments with several mentally retarded adults with or without on-site staffing

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TABLE I. Profile of Study Population

	Number (N=172)	Percent
Sex		
Males	92	53.5
Females	80	46.5
Age (yr)		
13–24	21	12.2
25–34	65	37.8
35–49	57	33.1
50–77	29	16.9
Level of mental retardation		
Borderline	8	4.7
Mild	40	23.3
Moderate	54	31.4
Severe	39	22.7
Profound	31	18.0

- Group homes—community residences or small intermediate care facilities for fewer than 50 residents and on-site staff
- Institutions—residential schools, large intermediate care facilities, or community residences for 50 or more mentally retarded people with many day program activities on site.

RESULTS

No HIV antibody was detected in any sera from 241 mentally retarded adults. There were 172 (71.4%) specimens from those living in nonfamilial residential settings and 69 (28.6%) from those living with families.

Among the 172 living in residential settings (Table I), there was no significant difference between numbers of males and females (92/172 and 80/172 respectively). The mean age was similar (males, 37.2 yr; females, 38.1 yr). Ages ranged from 13 to 77 years (overall mean, 35 yr). IQ scores were in the borderline to moderate level of mental retardation for 102 (59.3%) subjects, and 70 (40.7%) subjects were severely or profoundly mentally retarded. A diagnosis of Down's syndrome was noted for 22 (12.8%) subjects.

The study population included 95 (39.4%) who were living in residential schools or institutions and 61 (25.3%) who were in group homes, including those that identified themselves as small intermediate care facilities (ICFs) (Table II). Group home capacity ranged from 6 to 24 clients. The majority of those living in group homes, 52/61 (85%), lived in free-standing homes in the community rather than on campuses. The level of mental retardation did not determine a particular living situation. While 64/99 (64.6%) of those who were borderline to moderately mentally retarded were living in institutions, only 28/70 (40%) of those who were severely or profoundly mentally retarded were in institutions.

The majority of specimens (90.1%) were sent for routine thyroid function testing in the presence or absence of documented thyroid disease; 9.9% of specimens were sent in the process of a work-up for symptoms suggestive of endocrine disease. Labeling with "general" precautions stickers indicated that the physician suspected infection with HIV in five (2.9%) specimens. Most subjects in residential settings had evidence of exposure to hepatitis B: 57 (35.2%) had not been vaccinated but had serologic markers for exposure—HBsAg or anti-HB—while 51 (31.5%) had immunity to hepatitis B secondary to hepatitis B vaccination. One third (54 subjects) were susceptible to hepatitis B infection.

DISCUSSION

In the United States, sexual transmission of HIV infection represents the most likely route, with 60.3% of cases arising from homosexual intercourse, and 4.0% attributable to heterosexual intercourse.[13] Sexual activity with HIV-infected partners might place those who are mentally retarded at risk. Sexual activity among many mentally retarded individuals is characterized by poor judgment and an inability to think about the consequences of one's behavior.[14] It has been noted that there is a comparable frequency of sexual intercourse among mildly retarded adolescents and those in the general population, whereas those who are moderately or severely retarded are less active.[15] A history of sexual assault has been documented in 25% of adolescents who are mentally retarded.[15]

Many mentally retarded adults do not have access to information regarding AIDS and "safer sex" practices. Most educational materials aimed at safer sexual behavior, including the use of condoms, have not been directed towards the cognitively or sensory impaired individual. Individuals served by agencies for the developmentally disabled, either in residential settings or day programs, are irregularly provided with information regarding sexual behavior, which may not necessarily apply to education regarding AIDS. In federally regulated, small intermediate care facilities (group homes), for example, AIDS education for residents is not a requirement.[16]

While there is no information regarding intravenous drug use in this group, Edgerton[17] has documented the low incidence of alcohol and non-intravenous drug use among mentally retarded adults living in community settings. It is likely that most mentally retarded individuals would not be capable of the demanding survival skills necessary to maintain oneself as an intravenous drug abuser.[18]

Some mentally retarded adults may be at risk for HIV infection through homosexual intercourse. Kastner et al[19] have reported on two mentally retarded men who became infected with HIV, presumably through homosexual intercourse with infected partners. Homosexual activity is known to occur in residential settings. This behavior has been seen by some to be primarily a consequence of sexual

TABLE II. Living Situation of Study Population

	Location of Residence				
Residence	Bronx	Westchester County	Putnam & Rockland	Orange/Sullivan/ Ulster/Dutchess Counties	Total
Family care	0	10	0	0	10
Apartment	0	5	1	0	6
Group home	10	44	2	6	61
Residential school	1	37	0	54	92
Other	0	3	0	0	3
Total	11	98	3	60	172

segregation in institutional settings.[1,20] Although the sexual orientation or risk-taking behaviors of those in this study sample are not known, the sera of 2.9% of those in residential settings were labeled with a sticker indicating that the physician suspected HIV infection. This suspicion was based on history that suggested high risk behavior. Despite this supposed history, this study did not demonstrate HIV antibody in any of these individuals.

The high prevalence of hepatitis B infection among mentally retarded, institutionalized adults has been well documented.[2,4] The similarity of routes of transmission for HIV and hepatitis B are well known.[5-7] One third (35.2%) of this study population demonstrated exposure to hepatitis B, and none had HIV antibody. This may possibly underscore what others have documented as the more highly infectious nature of hepatitis B relative to HIV infection,[21-24] as well as probable differences in the background level of seroprevalence of these viruses.

Willer and Intagliata[8] describe the process whereby many mentally retarded persons were taken out of institutions and placed in group homes, family (foster) care, supervised apartments, and with their own families of origin during the 1970s. This effort, they note, was largely the result of legislation that promised new rights to the mentally retarded individual. Freedom from unnecessary restraints and the ability to enjoy the benefits of human services available to nonretarded persons were among those rights. Concurrently, they discuss a decline in admissions to institutions; rather, people are placed in community-based residential care settings like those described above. In studying life among those who were deinstitutionalized, they found that in many aspects those living in various situations were not enjoying all of the benefits community life might offer. They found, for example, that most residents of group homes have few if any friends in the neighborhood. Of our sample, 6.4% were from New York City, all living in group homes felt to be integrated into the community because they were not on campuses with more than one other group home. Many of those who were living in Westchester County came from urban areas such as New York City where the seroprevalence of HIV is high. A lack of friends in the community may reduce the risk of acquiring HIV for those who live in areas of high prevalence.

Available seroprevalence statistics from the New York State Department of Health[25] on the cord blood of all newborns indicate that the counties in which the majority of these study participants reside had HIV seroprevalence rates of 0.21% to 0.32% (New York City-vicinity and mid-Hudson Valley) compared to 1.29% in New York City. Most of this study population live in areas of low or intermediate seroprevalence. This reduces the likelihood that transmission of HIV will occur in this group as compared with populations that live in the inner city.

Bayer et al[26] have suggested that mandatory screening for HIV antibody be considered in residential homes for retarded people in an effort to protect those who are uninfected. Tashjian[27] has argued that this would be a violation of human rights and could cause this group to be stigmatized or isolated by society. Nonetheless, Marchetti[28] reports that there is at least one state agency that has established mandatory screening for new admissions to institutions and/or contracted or state-operated community programs (group homes, sheltered workshops, etc). He also found that policy for some state-run programs includes segregation of individuals infected with HIV. Mandatory screening may not be of benefit to this group, as it is not for others. Although military laboratories have demonstrated a low false positive rate among low risk individuals, it is unlikely that most civilian laboratories maintain as rigid standards in the performance and interpretation of tests for HIV.[29] The consequence of mandatory screening of low risk populations in the civilian sector, especially in geographic areas with low seroprevalence, is likely to identify a significant number of individuals with false positive test results.[30,31]

A further issue of particular relevance to mentally retarded adults involves the feasibility of obtaining consent. New York State currently requires informed consent as well as pre- and post-test counseling.[32] For those who are mentally retarded this is problematic. Many of those who are mildly retarded are competent to make decisions regarding sexual matters.[33] The level of cognitive ability necessary to comprehend the long term implications of HIV testing still needs to be determined. If a mentally retarded adult is thought to be incompetent and has a court-appointed legal guardian who can give consent, recommendation for testing should be made with care and not dissociated from counseling. Learning how to counsel this group about the risks and benefits of HIV testing remains an important challenge.

Our study has several limitations. The sexual or other high risk behaviors of this group are unknown and difficult to ascertain. Conclusions are further limited by the small sample size. Most of those in the study came from geographic areas where HIV prevalence, as indicated by cord blood studies, is intermediate[25] and one may not be able to extrapolate directly the results to areas in which seroprevalence is significantly higher. The representativeness of this study group is difficult to define. Usual descriptive features of patient populations like race, level of education, and medical insurance do not apply in the same way that they do to the general population. Because this group primarily consists of those who are mentally retarded and living in state licensed facilities, their lifestyles are more reflective of services available through universal entitlements. All individuals living in these residential facilities receive Medicaid, but this is not a marker for the poverty circumstances typical to those who receive Medicaid. The lives of those in state-licensed facilities—controlled by regulations governing quality of life—tend to be rather similar, and in this way the sample is representative for mentally retarded adults.

Our preliminary results suggest that caution should attend recommendations to test mentally retarded persons for HIV or to institute mandatory screening policies in residential care facilities for the mentally retarded. Funding would be better directed towards effective AIDS education strategies and the further development of ethical and safe AIDS prevention policies for residential settings. Studies such as these should be carried out on larger and more geographically diverse groups prior to the institution of policies, so that the rights of those who are mentally retarded can be maximally protected.

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Lessons from New York City on HIV/AIDS in adolescents

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New York City (NYC) has led the nation in many ways in the past. Unfortunately, NYC may now be a crystal ball for the rest of the country, depicting how the human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS) epidemic will affect our nation's adolescents.

Six recent studies provide evidence that HIV has already made inroads into many segments of the adolescent population. New York City accounts for 20% of all reported cases of AIDS among 13–21-year-olds in the US.[1] The number of affected adolescents doubles every 14 months. The rate of reported cases in adolescents 13–21 years of age is roughly parallel to that of adults. The total number of reported cases (1,266 as of October 2, 1989) is only a few hundred less than the total number of cases in children from birth to 13 years (personal communication, D. Sondheimer, NICHHD, November 1989). This accounts for only 1% of total cases. Twenty-one percent of AIDS cases occur in young adults 20–29 years. Given the average latency of eight to ten years from infection to disease, undoubtedly many of these people became infected with HIV as adolescents, but did not exhibit symptoms until adulthood.[2]

The portrait of the epidemic among adolescents in New York City differs from that in children and adults. As seen in the Figure, the vast majority of cases in children less than 13 years of age is attributable to parents who were intravenous drug users (IVDUs) or their sexual partners. When comparing cases of AIDS in NYC among adolescents and adults, proportionately fewer adolescents reported homosexual or bisexual contact or intravenous drug use. Proportionately more adolescents reported heterosexual contact as the transmission route. This was particularly evident when teenage males were separated from females. Fifty-two percent of all cases among female teenagers in NYC and 47% of such cases in the rest of the United States excluding NYC were due to heterosexual intercourse.

Few studies of seroprevalence have included adolescents. Since October 1985, the US Department of Defense has routinely tested civilian applicants for military service for serologic evidence of infection with HIV type 1 (HIV-1).[3] Presence of HIV-1 antibody was confirmed by enzyme immunoassay and Western blot in 2,152 of 263,572 men and 42,489 women tested (1.4 per 1,000). The male to female ratio for teenagers under 20 years of age was 0.5. Recent data from seroprevalence studies among 69,233 Job Corps entrants 16 to 21 years of age revealed a seroprevalence rate of 3.9/1,000, roughly three times higher than the rate reported among young military recruits.[4] In a center serving runaway and homeless youths in New York City, 7% of the youths were HIV positive.[5] The rates increased with age, with seropositivity present in 16% of those who were 18 to 20 years of age. Most of these young people were well at the time of the survey. Anonymous serosurveys of 13,810 specimens from college campuses from students attending health services revealed rates of HIV seropositivity of 1/500.[6] Positive results were identified in five college campuses in different parts of the United States out of the total of 13 campuses sampled. HIV seroprevalence rates among all clinic patients attending a sexually transmitted disease (STD) clinic in Baltimore increased steadily with age, from 2.2% in those 15–19 years old to 9.9% in those 30 years of age or older.[7]

The prevalence of HIV infection was determined in babies at the time of birth throughout New York State between November 30, 1987, and November 30, 1988.[8] In-

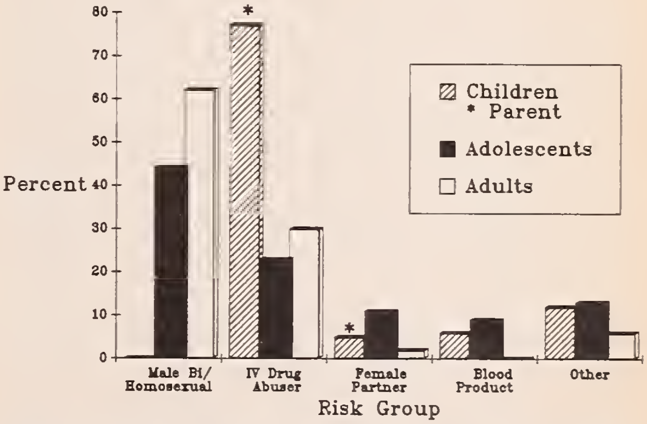

FIGURE 1. Reported cases of AIDS in New York City for three age groups: children <13 years; adolescents 13–21 years; adults ≥22 years (adapted from Vermund S, Hein K, Gayle HD, et al: Acquired immunodeficiency syndrome among adolescents: Case surveillance profiles in New York City and the rest of the United States. *Am J Dis Child* 1989; 143:1220–1225).

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fection rates for newborns whose mothers were aged 20 to 29 years (1.30%) and 30 to 39 years (1.35%) were significantly higher than rates for those with mothers younger than 20 years of age (0.72%). However, recent data point to a changing pattern, with only 10 HIV-positive babies born to mothers under age 20 years up until April 1988, but 88 such babies born in the 13 months between April 1988 and May 1989. Of all HIV-positive babies born in New York State, the rate was 1/1,000 for those babies born to 15-year-old mothers and 1/100 for HIV-positive babies born to 19-year-old mothers.

Only a small percentage of at-risk adolescents have been part of any type of seroprevalence study, whether anonymous or confidential. Therefore, the total extent of spread of HIV into the adolescent population is unknown.

COURSE AND TREATMENT OF HIV IN ADOLESCENTS

The natural history of HIV infection in adolescents is unknown. Studies are currently underway to determine the natural history of HIV infection in young children, adolescents, and adults with hemophilia. Adolescents appear to experience a slower progression than adults in the infected population with hemophilia.[9] Yet anecdotal reports from programs serving infected street youth point to a rapid progression, spanning several years from infection to death. There are reasons to believe that some aspects of the natural history may differ in adolescence. Since differences in mean survival time, types of HIV-related illnesses, and transmission categories exist between children and adults, it is not clear whether adolescents will follow the adult or child pattern, or will have a distinct course. Previous studies of other sexually transmitted diseases have documented increased morbidity in teenage females.[10]

The approriate use of medical interventions for adolescents is not clear; therapies being evaluated in national clinical trials program have not been tailored to teenagers. In fact, the first protocols to be developed restricted enrollment to adults older than 18 years of age or young children from birth through age 12. Although more protocols have recently become open to adolescents 13–17 years of age, only 44 adolescents had entered these trials as of November 1989. No adolescent age-specific outcome measures have been incorporated. This is a critical problem, because the dose (mg/kg), dosing schedule (frequency of drug administration), toxicity, and effectiveness of many classes of medications differ between children and adults.[11] There are physiologic changes in body size, body composition, and organ function during puberty that dictate close monitoring of therapeutic agents during periods of rapid growth.[12] The protocols must also be adapted to include adolescent-specific normal values in three areas: laboratory values (hemoglobin, hematocrit, alkaline phosphatase, etc); growth parameters (height, weight, and growth velocity); and neuropsychological assessments. The cognitive and psychological assessment instruments must not only be age specific, but must also take into consideration the special needs of disenfranchised youth. School dropouts and illiterate or underachieving youths, or those adolescents with different cultural or life style experiences, may make the use of tests standardized for middle class youngsters living at home inappropriate.

COMBATTING HIV/AIDS IN ADOLESCENTS

Prior to the HIV epidemic, health services specifically geared to the needs of adolescents were limited. The categorical approach to organizing services has produced a fragmented system of health care. Examples of programs for teenagers include family planning clinics, drug use or prevention programs, teenage pregnancy programs, and sexually transmitted disease clinics. HIV-related services are needed in these programs. A more holistic approach to care, in which health services are integrated with other youth services in one site, is another approach.[13] The development of a model comprehensive program for HIV seropositive adolescents has recently been funded by the Health Resources and Services Administration at Montefiore Medical Center in the Bronx. The special Adolescent AIDS Program at Montefiore Hospital provides clinical care, clinical research, and advocacy for high-risk and HIV-positive adolescents.

Additional resources are needed to foster incorporation of primary and secondary risk reduction strategies into programs, given the current demands placed on these programs. The HIV-related counseling, testing, and follow-up services for at-risk or HIV-positive teenagers, their partners, and families are different from those programs designed to counsel adults.[14] The trend toward making HIV testing more available at this point in the epidemic to all American citizens, particularly those living in high seroprevalence areas, will only make this gap wider.[15]

New York City has been a site for piloting innovative risk reduction and educational efforts geared specifically to adolescents. A few examples include the Star Teen Theatre group at Mount Sinai Hospital, the ARREST (AIDS Risk Reduction/Education Skills Training) Program at Montefiore's Adolescent AIDS Program, the street outreach programs of the New York City Department of Health, the Victim Services Agency ("Street Work Project") in Manhattan, Planned Parenthood's "Streetbeat" in the Bronx, the Hetrick-Martin Institute projects, and the special care unit for HIV-positive adolescents at Covenant House.

The first invitational conference on HIV/AIDS in adolescents was held in March 1988 in NYC. More than 100 invitees developed recommendations to guide people nationwide in controlling and managing the HIV epidemic in adolescents. These recommendations, as well as background papers, were published in a special edition of the *Journal of Adolescent Health Care* in May 1989.[16]

The Adolescent AIDS Network of New York City, a volunteer organization, has met monthly since July 1987 to alert health, education, and social service agencies to the HIV epidemic among adolescents. Committees have been formed to follow pressing medical issues, innovative education and risk reduction programs, and policy and legislative issues. The network hosted one of six federally sponsored regional conferences on AIDS in adolescents in May 1989. Sponsoring agencies included National Institute of Child Health and Human Development, National Institute on Drug Abuse, National Institute of Mental Health, Maternal and Child Health, Centers for Disease Control, and the Society for Adolescent Medicine. Other networks and conferences were held in Washington, DC, Chicago, Houston, Miami, and San Francisco to stimulate interagency planning, cooperation, and research.

Those working with high-risk or HIV-positive teenagers in New York City could now become role models for students entering the ranks of the health professions in the coming decade. Clearly we will need to expand the manpower capabilities and the opportunities for training providers who will care for HIV-positive teenagers, and promote risk reduction and prevention techniques appropriate for adolescents.

POTENTIAL SCOPE OF HIV SPREAD AMONG ADOLESCENTS

The notion of "risk groups" is not a useful concept in adolescence. Risk status may currently be more a reflection of geography (ie, prevalence of HIV in a given area) than being a member of a risk group.[17] By adulthood, the majority of adolescents will, in fact, be at risk for acquiring HIV. Risk-related behaviors cross over from traditional risk groups to all adolescents who are sexually experienced. For example, many teenage boys who identify themselves as being homosexual have not had intercourse.[18] Alternatively, unprotected receptive anal intercourse is reported in some surveys of heterosexual teenage girls.[19] Despite the current emphasis on abstinence and monogamy, the fact remains that more than half of all adolescents in the United States have had sexual intercourse by age 19.[20] Reported rates in black and white urban females are as high as 80% and 60%, respectively, by age 19.[21] Rates of most sexually transmitted diseases are highest among teenagers when rates are corrected for the percent of sexually experienced people at different ages.[22]

The next few years could make a tremendous difference in the lives of American youth. If effective prevention programs are designed and implemented, the course of the future epidemic could be radically altered. If not, we stand to repeat the course of the epidemic documented in adults and their infected offspring during the past nine years. AIDS has become the seventh leading cause of death among people ages 15–24 years.[23] Primary prevention programs should be age specific if they are to reach adolescents because of the cognitive and social differences between teenagers and the age groups bracketing adolescence. Secondary preventive efforts are also clearly needed, since HIV has already reached some segments of the adolescent population. A healthy, productive generation of adolescents in the 1990s will ensure that America has the healthy generation of adults needed to support the growing elderly population in the 21st century. The HIV epidemic threatens the viability, perhaps the very existence, of this next generation.

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THE CPC

Thoracic lymphadenopathy and disseminated *Pneumocystis carinii* in AIDS

CASE PRESENTATION

Steven Szebenyi, MD (Chief, Division of General Internal Medicine): This patient was a 30-year-old Hispanic man who was born in Puerto Rico and who lived in New York City most of his life. He was a registered nurse who had probably been exposed to tuberculosis in the course of his employment. He had never smoked cigarettes, consumed alcohol, used drugs intravenously, or been transfused with blood. He gave a history of homosexuality with only a few lifetime partners, and infrequent receptive anal intercourse with those partners. Supporting this assertion is the lack of a history of hepatitis or sexually transmitted disease. He was apparently well all of his life, except for a tonsillectomy as a child.

Approximately two years prior to his final admission, while he was in New York City, the patient was found to be seropositive for human immunodeficiency virus (HIV) infection. Several months later, he was admitted to a hospital for a cough and underwent bronchoscopy, at which time *Pneumocystis carinii* pneumonia was confirmed. At the same time, acid-fast bacilli were seen on a sputum smear, but no organisms were ever recovered on culture. He was treated with trimethoprim-sulfamethoxazole, to which he reacted with an anaphylactic episode. He completed treatment for his pneumonia with intravenous pentamidine. Other treatment at that time included the administration of zidovudine (AZT), 200 mg every four hours. He was treated for his presumed tuberculosis with isoniazid, ethambutol, and rifampin, receiving nine months of two-drug therapy.

Subsequently, the patient was well again for about ten months, at which time he was first admitted to Bassett Hospital with a cough and respirophasic chest pain. Findings of note were bibasilar rales, a serum level of lactate dehydrogenase (LDH) of 350 U/L, a mildly elevated aspartate aminotransferase (AST/SGOT) at 54 U/L, a lymphocyte count of 490/μL, and a chest film showing bilateral infiltrates consistent with *Pneumocystis carinii* pneumonia (Fig 1). His arterial blood gas determinations were satisfactory at that time, with a Po_2 of 93 mm Hg. An induced sputum specimen contained *Pneumocystis*. He received 16 days of intravenous pentamidine therapy in the hospital and five days at home subsequent to discharge, after which pentamidine was discontinued.

Although his condition improved following his discharge from the hospital, based on clinical and radiographic evaluations, he was readmitted about one month later. Several days before readmission, he presented again with fever, dyspnea, respirophasic chest pain, cough, and upper gastrointestinal complaints. A chest film showed improved interstitial infiltrates, interestingly with a new lingular consolidation. On examination, his temperature was 39°C, and his weight was 43.2 kg. His lungs were clear on physical examination, and abdominal findings were within normal limits. The white blood cell (WBC) count was 1,700/μL, with 78% mature polymorphonuclear (PMNs) leukocytes and 3% immature forms; the hematocrit was 30.3%, and the mean corpuscular volume (MCV) was increased, consistent with his zidovudine therapy. Induced sputum was again positive for *Pneumo-*

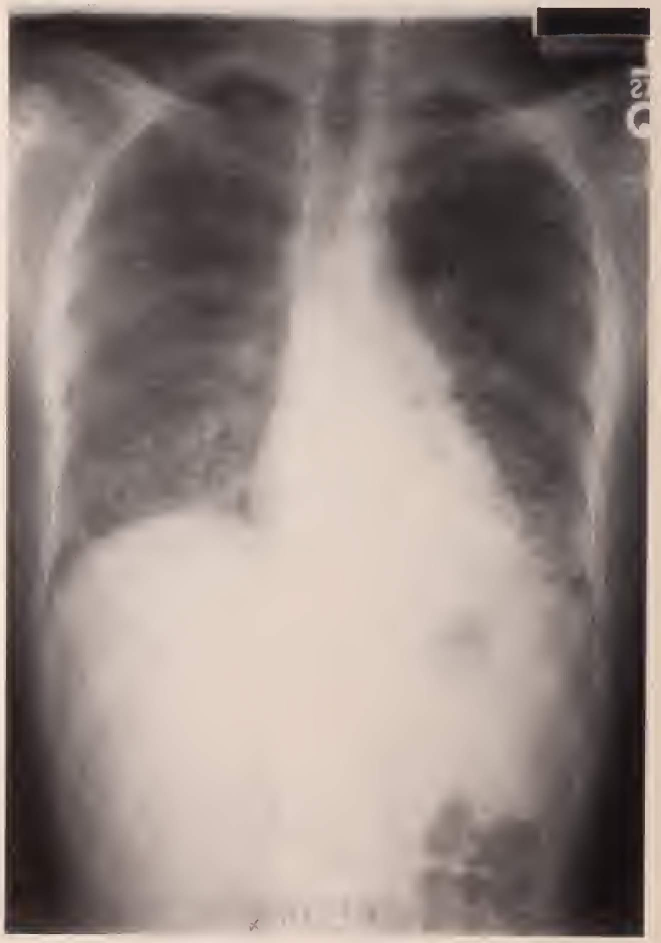

FIGURE 1. Chest film showing extensive parenchymal infiltrative changes in both lungs, compatible with *Pneumocystis carinii* pneumonia.

Based on a clinical conference held at Mary Imogene Bassett Hospital, Cooperstown, NY, on March 28, 1989.

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cystis carinii and not for the presence of other organisms. He was treated with intravenous pentamidine for five days, followed by two days of aerosol pentamidine, 600 mg/day, which he subsequently continued at home. He received two units of packed red cells during that admission; zidovudine was discontinued because of the low WBC, and he was started on ranitidine for presumed gastritis, although that was never firmly diagnosed. After he had completed his 21-day course of pentamidine, he was then maintained on 150 mg of aerosolized pentamidine every two weeks.

For a period of about seven months, the patient did quite well as an outpatient. His WBC count gradually increased to 3,100/μL, and zidovudine was restarted, initially as a half dose of 100 mg every four hours and subsequently to full doses again, which he tolerated for quite some time. Blood cultures for *Mycobacterium avium-intracellulare* were repeatedly negative.

His appetite improved slightly, as did his energy level during this time, and his weight peaked at 47 kg four months prior to his final admission. A month later, however, he missed his outpatient appointment, which was very unusual for him. It is still not clear what happened, but somewhere in that interval his condition deteriorated. When he came to see me one month later, he had some loss of appetite and weight, as well as a five-day history of fever, increased cough, respirophasic chest pain, chills, and cold sweats. His weight was 42.2 kg; temperature, 38.5°C; and respirations, 18/min. His physical examination was otherwise normal. The chest film, however, showed new bilateral and right paratracheal adenopathy, though there were no effusions or infiltrates (Fig 2). His WBC count was 1,400/μL with 72% mature PMNs; hematocrit, 32.3%; serum calcium, 8.1 mg/L; serum LDH, increased to 476 U/L; and serum SGOT, now considerably increased to 476 U/L. His albumin level was 3.5 g/dL. We again reduced the dose of zidovudine to 100 mg every four hours because of the low white blood cell count.

Two months before his final admission, a chest computed tomographic (CT) scan and a pelvic CT scan confirmed the extensive mediastinal lymphadenopathy, hilar adenopathy, and a slight enlargement of the spleen with areas of low attenuation. At that point he was referred to the surgical outpatient department for possible thoracotomy to diagnose the disease process affecting his lymph nodes and spleen. When he was seen there several weeks later, there was marked increase in the size of nodes as well as hepatosplenomegaly. One month before his final admission, he was admitted for an attempted mini-thoracotomy, at which time he was complaining of new onset of paresthesias in his extremities, increasing dyspnea, and chest pressure that was different from his previous discomfort. The hepatosplenomegaly was very notable at that point. The hematocrit was 35.1%, and the WBC count was 2,200/μL. The serum enzyme levels indicative of liver function were now deteriorating: LDH, 1,121 U/L; SGOT, 219 U/L; alkaline phosphatase, 376 U/L; and gamma glutamyl transpeptidase (GGTP), 162 U/L (normal range, 15–85 U/L). The serum bilirubin was 0.3 mg/dL; serum albumin, 1.1 g/dL; and serum calcium, 6.6 mg/dL. The arterial blood gas determinations on room air showed a pH of 7.49; P_{CO_2}, 34 mm Hg; and P_{O_2}, 65 mm Hg. The surgeons at that point were appropriately concerned about his nutritional status and his ability to withstand thoracotomy. A bone marrow biopsy was performed, but it did not indicate a diagnosis.

The patient's case was discussed at tumor board; the clinical conclusion at that point favored deferral of radiation or chemotherapy until a tissue diagnosis could be made. Mediastinoscopy and bronchoscopy were felt to be the best approaches, although the pulmonary consultants felt that bronchoscopy probably would be of low yield in this setting. The patient was rescheduled for admission for mediastinoscopy.

Eight to ten days later, his weight was up to 47 kg, with notable splenomegaly to 4 cm below the costal margin, a palpable liver edge, and abdominal distention compatible with ascites. His prothrombin time was elevated to 14.4 seconds (control, 11–12.5 sec), serum liver enzyme levels continued to increase, and the serum albumin level further decreased to 0.9 g/dL. He complained of some paresthesias, and a CT scan of his head was performed. It showed only mild cortical atrophy, a finding not at all unusual in this disease process.

Two weeks prior to his final admission, when seen in the surgical outpatient department for evaluation prior to the mediastinoscopy, the patient complained of increasing orthopnea and cough. His pulse rate was 150/min; he presented clinical evidence of increasing pleural effusions and ascites, and lower extremity edema. His blood count was unchanged; his prothrombin time was 15.8 sec (control, 11–12.5 sec), and the serum albumin was 0.8 g/dL. His chest film now showed further progression of adenopathy and pleural effusions; a repeat CT scan of the abdomen showed a liver of normal size, a 17-cm spleen, pleural effusions, ascites, and probable retroperitoneal adenopathy as well. Surgery was deferred, and the patient was admitted to the hospital.

Even at this point, the patient wanted all therapeutic avenues explored, though he was willing to accept a do-not-resuscitate (DNR) status. After much discussion, it was decided to treat the patient empirically for presumed lymphoma—the diagnosis that seemed to fit best with the clinical picture, although other possibilities were considered as well. The patient was discharged and treated on an outpatient basis with cyclophosphamide, vincristine, and prednisone.

About ten days later, increased dyspnea, fever, chest discomfort, and a new right upper lobe infiltrate on chest film necessitated his final readmission. On examination, his temperature was 39°C; pulse rate, 150/min; and respiratory rate, 44/min. Except for an ulcer at the corner of his mouth, no other changes were seen on physical examination. His hematocrit was 27.2%, and the WBC count was 4,800/μL, with a left shift. Liver enzyme levels had decreased, and the chest film showed increased adenopathy and infiltrates (Fig 3). The sputum was negative for *P. carinii*, but was positive on culture for *Staphylococcus aureus* and *Bordatella bronchiseptica*. Blood cultures were positive for *Streptococcus intermedius*, *B bronchiseptica*, and cytomegalovirus (CMV). Culture of the mouth ulcer grew *Herpes simplex* virus type I.

The patient received the aggressive medical management that he and his family requested, including antibiotic therapy together with acyclovir. However, renal failure, worsening coagulopathy, and respiratory distress progressed, and he died.

RADIOLOGIC PRESENTATION

Vittal Chanamolu, MD (Radiology Attending): The chest radiograph taken when the patient first came to this institution showed extensive infiltrates in the lung fields bilaterally (Fig 1). In a patient known to have HIV-positive status, this most likely represented *Pneumocystis carinii* infection. We must, however, consider other possibilities, including infections with mycobacteria, cytomegalovirus, toxoplasmosis, and other opportunistic fungi in the differential diagnosis.

The patient's chest films subsequently cleared and his condition improved. However, nine months later, even though the chest film showed clear lung fields, there was now extensive hilar adenopathy as well as right paratracheal and aorto-pulmonary window adenopathy (Fig 2). The CT scan of the chest a week later confirmed once again the extensive lymphadenopathy. The liver was of normal size, and there was mild enlargment of the spleen, which had low-density areas within it. I thought that the patient most likely had AIDS-related lymphoma at this time. However, in the differential diagnosis one would need to consider also Kaposi's sarcoma and other opportunistic infections.

Seven weeks thereafter, a repeat CT examination disclosed extensive bilateral pleural effusions and ascites. In the interval, the spleen had markedly increased in size, again with multiple low-density areas within it. The differential diagnosis remained the same. There may have been some new retroperitoneal adenopathy at this time.

The final chest radiograph taken several days prior to death showed extensive bilateral pulmonary infiltrates, which could possibly have been the cause of his death (Fig 3). There was also evidence of marked interim increase in the lymphadenopathy.

Patrick N. Dwyer, MD (Attending Physician, Hematology-Oncology): This patient teaches us much about AIDS and AIDS-related diseases. AIDS is a highly lethal disease, very common in young and middle-aged patients, and involves a combination of multisystemic disorders, as this patient demonstrates. The patient with AIDS benefits from the collaborative input of multiple subspecialty services. Empiric therapy is no stranger to this disease, as with toxoplasmic encephalitis, although empiric therapy for lymphoma is considered highly unorthodox. The value of a postmortem examination in this case was greatly increased by our inability to obtain a tissue diagnosis during this patient's lifetime.

This patient had serologic evidence of HIV infection, multiple episodes of *Pneumocystis carinii* pneumonia, and several hematologic abnormalities including anemia

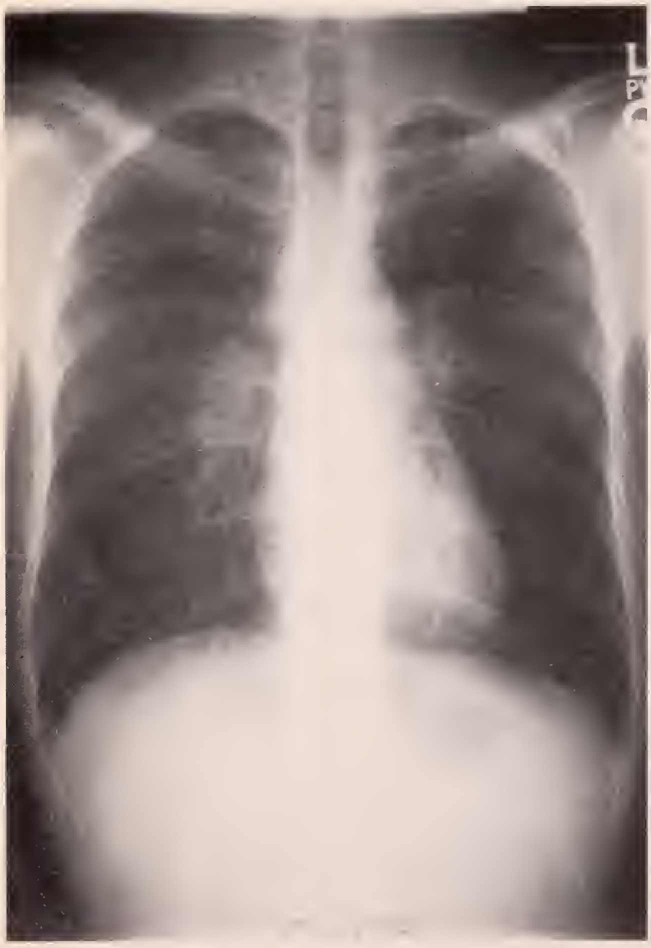

FIGURE 2. Chest film (taken eight and a half months after film in Fig 1), showing clearing of pulmonary infiltrates, and new right hilar, right paratracheal, and aorto-pulmonary window lymphadenopathy.

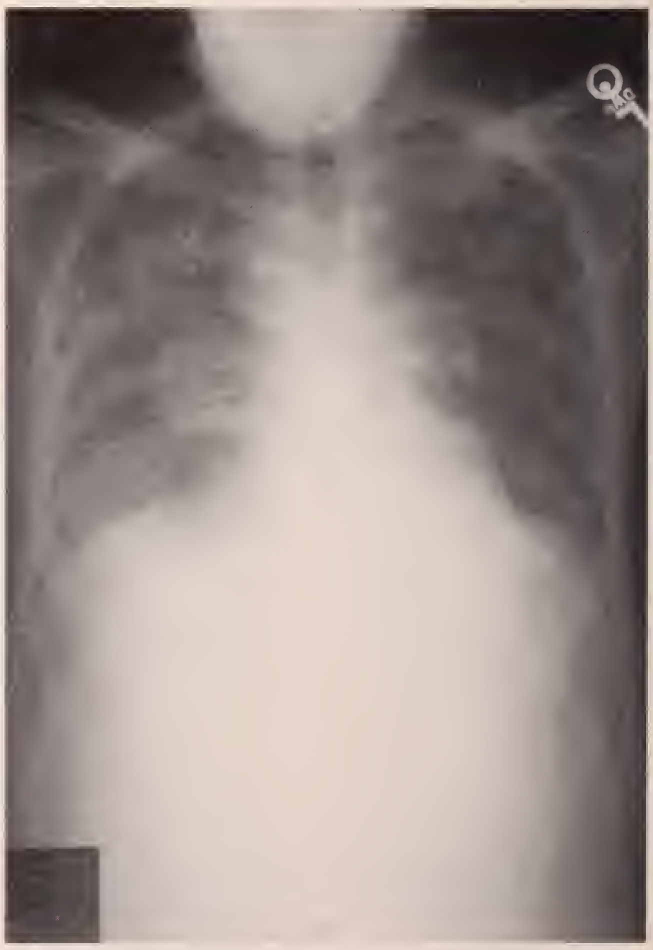

FIGURE 3. Chest film on final admission, demonstrating extensive bilateral pulmonary infiltrates and marked increase in hilar and mediastinal lymphadenopathy.

and profound leukopenia secondary to marked depression of the CD4 helper cells. Neurologic findings included cerebral atrophy on the CT scan and a terminal sensory-motor polyneuropathy, which is probably the most common neurologic manifestation of AIDS. He had three different herpes-group virus infections, including dermatomal zoster, herpes simplex, and cytomegalovirus, toward the end of the disease course.

A constitutional wasting illness was characterized by sweats, weight loss, fevers, very prominent hilar and mediastinal adenopathy, enlargement of the spleen with focal lesions, and failure of the liver. He had hypoprothrombinemia and hypoalbuminemia with resultant peripheral edema, ascites, and pleural effusions. Finally, he had terminal diffuse pneumonia and sepsis with *Bordatella bronchoseptica* and *Streptococcus intermedius*.

The wasting, weight loss, fevers, sweats, lymphadenopathy, and splenomegaly seen in patients with AIDS can result from invasion by a multitude of infectious organisms or from malignancy. The most common neoplasms are Kaposi's sarcoma and non-Hodgkin's lymphoma; the latter was considered by us to be highly probable in this patient. Let us review the list of diseases that can be related to hilar and mediastinal lymphadenopathy in this clinical setting.

Pneumocystis carinii pneumonia (PCP) is the most common life-threatening infection in AIDS, affecting 65% to 80% of patients with AIDS during their lifetimes. It has been traditionally classified as a protozoan disease, spread by respiratory transmission, with manifestations largely confined to the thorax. It causes a diffuse pneumonia that is more indolent and subtle in patients with AIDS than in patients with hematologic disorders. It causes fever, dyspnea, cough, and hypoxemia. The initial chest film often, as in this patient, is normal; but subsequent radiographs reveal at first unilateral and then bilateral interstitial infiltrates which may progress from reticular to reticulonodular to frank consolidation.

However, mediastinal and hilar lymphadenopathy are very rare with PCP. In a review of six different series of PCP comprising 487 patients, only 1.6% had mediastinal adenopathy, the cause of which was not documented by a pathologic tissue study in any of the cases.[1-6] Some of these patients improved with antiprotozoal therapy. Realistically, we must accept that PCP is unlikely to be related to the lymphadenopathy, despite our knowledge that this man did harbor such organisms.

The diagnosis of infection by *Pneumocystis carinii* is made by identifying the organisms in secretions. Effective therapeutic agents are co-trimoxazole and pentamidine, which, however, are associated with frequent adverse reactions, frequent relapses, occasional respiratory failure, and death.

Mycobacterium tuberculosis is another infection that occurs in about 10% of AIDS patients, most commonly in patients who are Haitian, black, Hispanic, or intravenous drug abusers. Although in AIDS the illness suggests a progressive, primary type of tuberculous infection, in reality it is believed to be a reactivation type of infection, generally presenting with fever and a pulmonary syndrome. The cavitation and apical infiltrates commonly seen in otherwise normal hosts with tuberculosis are not characteristic of tuberculous infection in AIDS patients; mid- and lower-lung zone infiltrates are observed in the majority of such patients. In fact, 35% of AIDS patients with tuberculosis will have no lung infiltrates at all on chest film. Hilar and mediastinal lymphadenopathy will be seen in from 20% to 59% of AIDS patients with this infection. Thus, in the patient under discussion today, the appearance of the chest radiograph gives us reason to consider seriously the possibility of tuberculosis.

In more than half of AIDS patients with *Mycobacterium tuberculosis*, the latter infection presents with extrathoracic disease involving multiple organs in the body. The diagnosis requires culture of the organism from the affected tissue. Few granulomata are seen in biopsied tissues from AIDS patients with tuberculosis, and acid-fast bacilli are very difficult to identify on tissue sections or smears, so that cultures are required in most cases. Also, tuberculosis skin testing with purified protein derivate (PPD) commonly gives negative results because AIDS is a disease of defective cell-mediated immunity. Generally, less than one fifth of AIDS patients will fail to respond to treatment for *Mycobacterium tuberculosis*. A three-drug antituberculosis regimen is recommended for several months, followed by two drugs for at least nine months and six months after culture conversion.[7] Most AIDS patients with tuberculosis will survive for a median of only seven months.

Immediately prior to the terminal phase of his illness, this patient received treatment for tuberculosis over a period of nine months. This fact is convincing evidence to dissuade us from considering that *Mycobacterium tuberculosis* played a role in the later events of his illness.

Mycobacterium avium-intracellulare (MAI) complex is also a very common mycobacterial infection in AIDS, occurring in approximately one third of AIDS cases. This organism is widely disseminated in the body, in contrast to *Mycobacterium tuberculosis*. It can involve the lymph nodes, especially the mesenteric lymph nodes, as well as multiple organs including the spleen, with occasional focal lesions such as this patient had.

Mycobacterium avium-complex infection can generate several clinical syndromes. These include a general systemic illness with fever, malaise, weight loss, and cytopenias; as well as three other discrete gastrointestinal presentations: diarrhea and abdominal pain due to colonic involvement; a Whipple-like malabsorption syndrome due to involvement of the small intestine; and obstructive jaundice due to lymphadenopathy in the porta hepatis.

However, only about 6% of patients with MAI have hilar and mediastinal lymphadenopathy. The diagnosis should be easily made by cultures. This patient's bone marrow was cultured for MAI slightly less than one month before he died. Such cultures can give false-negative results in less than 15% of cases; cultures of other tissues can also be used. There is no strong impetus to make this diagnosis in patients with AIDS, because therapy is ineffectual, even with investigational agents. Review of a comparative series of AIDS patients with and without MAI infection demonstrated no difference between the two groups in length of survival or days in the hospital.[8]

Several types of fungi can lead to the reported clinical presentation in patients with AIDS. Histoplasmosis is a disease that most commonly occurs among people living within the great river valleys in the United States. Its occurrence in patients with AIDS seems also to reflect this same geographic distribution. However, the Caribbean as well as South and Central America are endemic areas for histoplasmosis. While it is an uncommon infection, it should be considered here in light of the epidemiologic information. Patients with histoplasmosis may present with fever, weight loss, and gastrointestinal complaints, and with adenopathy in probably not more than 20% of patients. However, such patients may also develop hepatosplenomegaly, marked elevation of liver enzymes, and a terminal illness suggestive of septic shock, as did this patient. The chest radiograph may be normal in about one third of cases; generally, however, it shows diffuse or patchy infiltrates and intrathoracic lymphadenopathy. Histoplasmosis may present with several other syndromes as well.

The diagnosis of histoplasmosis requires bone marrow biopsy and culture, although the results are positive in only 69% of patients with the disease. Biopsy of other affected tissues can be used equally well to make the diagnosis. Therapy with amphotericin B with or without ketoconazole is effective initially, but antibiotic resistance and clinical relapse are common.

Cryptococcosis due to *Cryptococcus neoformans* is an uncommon but well recognized complication in AIDS, which can cause hilar and mediastinal lymphadenopathy. Generally this disease presents with pneumonia, either interstitial or lobar; occasionally pleural effusions can develop. Adenopathy is not a dominant feature of this illness, but occasionally truly massive adenopathy can develop with superior vena cava obstruction. Meningoencephalitis is the hallmark of this disease in up to 84% of patients; they usually demonstrate non-specific central nervous system findings and a striking lack of focal neurologic deficits. This disease can be widely disseminated at the time of biopsy or postmortem examination. The diagnosis is made by fungal cultures of either the blood or cerebrospinal fluid, or by detection of cryptococcal antigen in those fluids. Bronchoscopy is an effective means of diagnosis in patients with pulmonary infiltrates. The disease may respond initially to therapy with amphotericin B and a limited course of flucytosine, but there is a high failure and relapse rate thereafter.

Several malignant diseases commonly cause advanced hilar and mediastinal lymphadenopathy. Epidemic Kaposi's sarcoma (KS) occurs in up to one third of patients with AIDS, usually among homosexual patients. This neoplastic disease initially may generate few symptoms. Ninety-five percent of patients will have involvement of the skin or mucous membranes; in up to 50% of such patients with skin involvement, pulmonary KS will develop, and hilar and mediastinal lymphadenopathy will be a dominant manifestation in one third of this subset. Exudative pleural effusions are also common. Generally, such patients with KS will have nodular and diffuse infiltrates within the pulmonary parenchyma, and involvement of the gastrointestinal tract and of the spleen, sometimes with hypodense lesions, such as this patient displayed.

The diagnosis of KS is made by biopsy, or by the characteristic appearance of this vascular tumor at bronchoscopy. This neoplasm deserves important consideration, because it is responsive to either radiation for local disease, to interferon for disseminated disease in patients with intact CD4 levels, or occasionally to combination chemotherapy.

AIDS-related lymphoma was a leading consideration at the time that we gave this man his empiric trial of chemotherapy. Lymphoma occurs in only 3% of patients with AIDS, but can present with fever, sweats, weight loss, and commonly lymphadenopathy (abdominal, mediastinal and hilar, or peripheral). This disease has a marked predisposition to involve extranodal sites among AIDS patients. Frequently, the patients will present with central nervous system disease only; however, up to 40% of patients who do not present in such a manner have a high predisposition for meningeal involvement, as well as infiltration of the bone marrow and of the gastrointestinal tract. The disease can spread to multiple organs as well. The diagnosis is usually made by biopsy, although needle aspiration gives reliable results in experienced hands.

These particular lymphomas in AIDS patients are aggressive neoplasms, almost always of high or intermediate grade. Therapy with attenuated antilymphoma regimens, such as this patient received, are very effective, with complete remission rates of about 57%; however, the median survival is four to ten months after diagnosis. Primary central nervous system lymphoma has an especially poor prognosis.

AIDS-related Hodgkin's disease should be given brief consideration, because we frequently see hilar and mediastinal involvement as evidence of Hodgkin's disease in non-HIV-infected hosts. We should note, however, that even in AIDS patients with nodular sclerosing Hodgkin's disease, there is only a 15% incidence of hilar or mediastinal lymphadenopathy. Again, similar to the infections and other neoplasms that commonly occur in AIDS, Hodgkin's disease may assume an atypical form in the setting of AIDS, with extranodal involvement and advanced stage. It commonly presents with fevers, sweats, and weight loss, as did this patient. The diagnosis requires tissue biopsy. Treatment with anti-Hodgkin's regimens generates median survivals of about 15 months.

Hilar and mediastinal lymphadenopathy in the setting of AIDS always indicates serious disease; such node enlargement is not generally a component of the progressive generalized lymphadenopathy of AIDS or the AIDS-related complex (ARC), and it should not be considered as a secondary manifestation of PCP. Histologic and bacterial confirmations are always necessary to establish the correct diagnosis, since without them accurate diagnosis is impossible in view of the legion of organisms and tumors that can cause such a presentation in AIDS patients. There is no pathognomonic radiographic or clinical presentation that will establish the diagnosis in the absence of suitable tissue or bacteriologic studies.

Multiple opportunistic infections also can occur simultaneously in AIDS patients; the medical literature details such coexistence of different diseases, even within individual lymph nodes. It is important to emphasize that such complicating illnesses frequently display atypical clinical courses in AIDS patients.

At the time of this man's empiric chemotherapy for suspected lymphoma, the two leading diagnostic possibilities in my opinion were non-Hodgkin's lymphoma or *Mycobacterium avium*-complex. Subsequent to his trial of chemotherapy, we obtained more information, which, however, has created only more uncertainty and indecision.

Although this man could have had histoplasmosis, Kaposi's sarcoma, cryptococcosis, and other illnesses as well, the course of his illness does not lead toward any of those entities. It strongly suggests non-Hodgkin's lymphoma, with prominent lymphadenopathy, elevated LDH, and a large spleen; yet a very disturbing feature, to me, is his failure to respond to chemotherapy by day 13. That is clearly unusual and has caused me to move non-Hodgkin's lymphoma further down on my list.

Mycobacterium avium-complex also can present this way, commonly with splenomegaly with focal lesions. Prominent adenopathy is not so common, but does not dissuade me from this diagnosis. Also, as I said, it is a nontreatable disease with a poor outcome. But the clinical feature that diverts me from this alternative diagnosis is the report of bone marrow cultures negative for that organism. So I cannot accept either non-Hodgkin's lymphoma or MAI infection as the most likely diagnoses.

No other diagnoses can be strongly supported, though the clinical presentation could be suggestive of tuberculosis—a Hispanic nurse with prominent thoracic adenopathy. Again, there is a red flag here. *Mycobacterium tuberculosis* was not strongly considered during the patient's lifetime because of the recently completed regimen of triple anti-tuberculous drugs. However, patients who have been treated with multiple drugs for tuberculosis have been known to relapse with disseminated disease within a short period of time. We really must consider tuberculosis on our list of possible diagnoses as well.

One last comment: The organism *Bordatella bronchoseptica* was recovered from this man's blood and respiratory secretions. This is an extraordinarily uncommon pathogen in man, not believed to cause illness in persons with normal immune mechanisms, but frequently recovered from animals. It is a rare cause of pertussis, bronchitis, and pneumonia in debilitated hosts.

CLINICAL DIAGNOSES

Acquired immunodeficiency syndrome.

History of *Pneumocystis carinii* pneumonia.

Febrile wasting illness with hilar and mediastinal adenopathy, splenomegaly, hepatic synthetic failure (possibly due to non-Hodgkin's lymphoma, *Mycobacterium avium*-complex, or tuberculosis).

Terminal septicemia and pneumonia (due to *Bordatella bronchiseptica*, *Streptococcus intermedius*).

PATHOLOGY PRESENTATION

Harlan D. Alpern, MD (Pathologist-in-Chief): At autopsy, there was evidence of quintilobar pneumonia. The right lung had a mass of 860 g and the left lung 760 g, approximately twice normal. Cut sections revealed areas of the lung surfaces to be dark-red, rubbery, and indurated. There were focal cystic changes. Histologic sections revealed alveolar air spaces to be filled with amphophilic foamy material, and there was a mild interstitial inflammatory infiltrate.

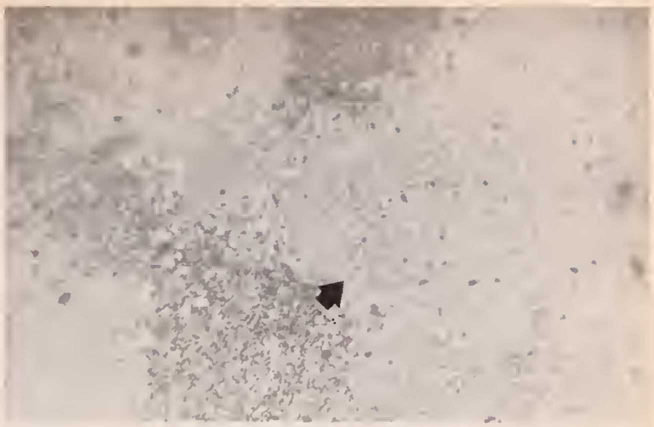

FIGURE 4. Confluent granulomata (arrow) in mediastinal lymph node (hematoxylin-eosin stain, original magnification × 40).

The sections stained by the Gomori methenamine silver (GMS) method demonstrated large numbers of *Pneumocystis carinii* cysts, many of which were bean-shaped and contained trophozoites. Other areas of the lungs lacked this intra-alveolar foamy material, but contained macrophages, occasional small lymphocytes, and some large mononuclear cells. At high magnification, these large mononuclear cells demonstrated huge intranuclear inclusions with perinuclear clear zones characteristic of cytomegalovirus infection. Viral cultures of the lungs taken at autopsy confirmed the presence of cytomegalovirus. The finding of this virus with *Pneumocystis carinii* is common. Sections stained for acid-fast organisms revealed none.

The lymph nodes in the pulmonary hilum and mediastinum were strikingly enlarged. On histologic examination there was marked lymphoid depletion, and there were confluent non-caseating granulomata (Fig 4). The GMS stain demonstrated *Pneumocystis carinii* organisms (Fig 5) in these lymph nodes.

The bone marrow was hypercellular, exceeding 90%, with marked myeloid hyperplasia, which is typical of AIDS. In the marrow were multiple granulomata. The GMS stain demonstrated *Pneumocystis carinii* in the granulomata (Fig 6). Stains for acid-fast organisms in the bone marrow were negative.

The spleen was large, measuring 900 g, approximately six times the normal mass. Lymphoid elements were strikingly depleted, which is typical of AIDS. There were also

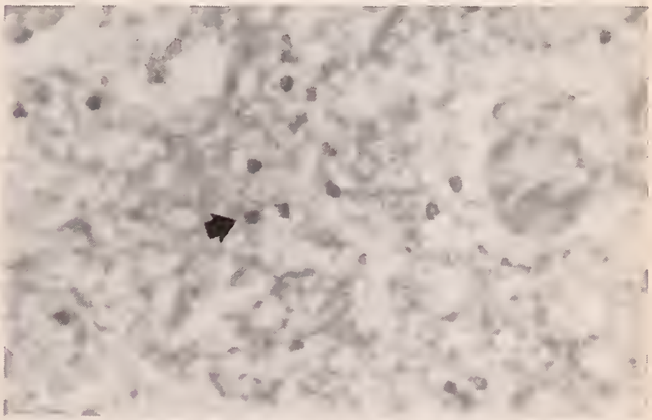

FIGURE 5. *Pneumocystis carinii* (arrow) in mediastinal lymph node (GMS stain, original magnification × 400).

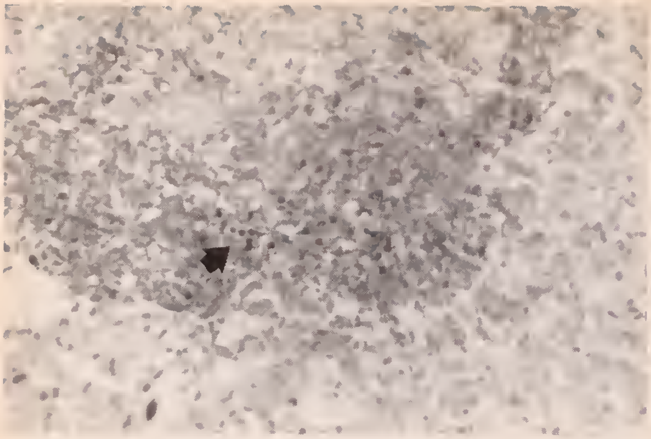

FIGURE 6. *Pneumocystis carinii* (cluster at arrow) in bone marrow granuloma (GMS stain, original magnification × 200).

large numbers of granulomata within the spleen, and the GMS-stained sections demonstrated *Pneumocystis carinii* in these granulomata.

The liver was not enlarged, with a mass of 1,200 g. There were large numbers of yellow-gray nodular lesions in the liver, measuring from 1 to 2 mm. On histologic examination, these were granulomata, and the GMS stain demonstrated *Pneumocystis carinii* within them.

The kidneys similarly demonstrated multiple yellow-gray nodules, especially in the cortex, measuring 1–2 mm in diameter. The histologic sections also showed *Pneumocystis carinii*, with areas of associated calcification.

Pneumocystis carinii pneumonia is common in patients with the acquired immunodeficiency syndrome. In fact, it represents the most common cause of death in these patients. Although *Pneumocystis carinii* infection in the lungs is frequently observed, documented *Pneumocystis carinii* infection outside the lungs is very rare. Dr Margaret Grimes and her colleagues of the Columbia University College of Physicians and Surgeons reviewed the English literature in 1987 and found 16 such cases, to which they added a case of their own.[9] Widely disseminated *Pneumocystis carinii* beyond pulmonary hilar and mediastinal lymph nodes, as in this case, is even rarer.[9]

The route of dissemination of *Pneumocystis carinii* from the lungs is presumed to be both lymphatic and hematogenous. The granulomatous inflammatory process as a response to the PCP was seen, however, even before the recognition of AIDS, when it was found in other immunosuppressed or immunodeficient individuals. Although *Pneumocystis carinii* has historically been classified as a protozoan, recent electron micrographic[10] and gene sequencing[11] studies provide evidence that *Pneumocystis carinii* is more properly classified as a fungus than as a protozoan.

In summary, this 30-year-old man with AIDS died of respiratory insufficiency due to *Pneumocystis carinii* and cytomegalovirus pneumonia. An incidental and most extraordinary finding was widely disseminated *Pneumocystis carinii*.

Dr Szebenyi: Early in 1989, the FDA approved treatment with aerosolized pentamidine as an investigational new drug; the approved protocol calls for aerosolized pentamidine, 300 mg once a month, delivered by a Respigard II nebulizer.

David Blumenstock, MD (Attending Surgeon): One of the lessons to be learned from this patient is that the open biopsy procedure should have been undertaken in spite of the patient's rapid deterioration. Whether the information so obtained would have altered the outcome is difficult to say.

Dr Alpern: I am certain that we would have made the diagnosis at surgery, just as we did at autopsy. The amphophilic material would have strongly raised the suspicion, and the GMS stain would have demonstrated the organisms. It is unfortunate that the bone marrow biopsy that was performed during life, and which I reviewed at the time of autopsy, did not demonstrate the granulomata.

In the patient that Grimes et al reported,[9] the condition was diagnosed during the patient's life from endoscopic biopsies of the esophagus and duodenum. Other authors have made the diagnosis antemortem by bone marrow examination.[12]

David S. Pratt, MD (Attending Physician, Pulmonary Medicine): In view of the findings at autopsy, I do not think that a transbronchial biopsy would have helped very much. Even patients with AIDS who are treated aggressively for *Pneumocystis carinii* may harbor residual organisms, so that if a bronchoscopic biopsy in this man had demonstrated *Pneumocystis carinii* in the lung parenchyma, it would not have suggested the diagnosis of disseminated disease, which you made at autopsy. Consequently, to some extent, I feel that my reluctance concerning bronchoscopy was justified in this particular patient at that time.

Dr Alpern: I agree. It was known that the patient had *Pneumocystis* in the lungs. It was the widespread extrapulmonary disease that was not known, especially the mediastinal enlargement that was suspicious for lymphoma. The a priori likelihood that the mediastinal enlargement was due to *Pneumocystis* was very low, and the finding of these organisms in the lung, so common in AIDS patients, would not have thrown any additional light on the cause of the mediastinal lymphadenopathy.

Nancy B. Merrell, MD (Attending Physician, Gastroenterology): Patients with diffuse granulomatous disease often have multisystem involvement, specifically in the bone marrow and liver. Percutaneous liver biopsy for both histologic and bacteriologic evaluations would have been most useful in this situation. It is well known that single percutaneous liver biopsies do not have a good yield in the search for metastatic infectious processes. This yield can be increased by performing two or three liver biopsies; but with the aid of laparoscopy, the yield of multiple percutaneous liver biopsies can be increased dramatically, especially if capsular or subcapsular involvement of the liver can be visualized.[13-15]

Dr Alpern: Yes. There were many granulomata in the liver, and the liver biopsy might very likely have established the diagnosis.

Dr Dwyer: Could the attenuated antilymphoma therapy given to this patient have influenced the course of his *Pneumocystis* infection? He received attenuated CVP (cyclophosphamide, vincristine, prednisone) chemotherapy more than two months *after* the development of his hilar and mediastinal lymphadenopathy. When impending respiratory failure from *Pneumocystis carinii* pneumonia is treated with prednisone, a significant anecdotal experience suggests that significant improvement in respiratory function may occur. The seven-day course of prednisone that this patient received almost a month prior to his death clearly could have worsened his impaired immune defenses and participated in the deterioration of his infections with *Pneumocystis* and cytomegalovirus. However, I do not think that his non-myelosuppressive doses of cyclophosphamide had any significant impact. The empiric chemotherapy in this patient, who could not withstand definitive biopsy, involved careful consideration of the potential benefits versus the risks of worsening the opportunistic infections in this young man, who was so close to death.

The medical literature concerning AIDS patients with *Pneumocystis carinii* infection indicates that lymphadenopathy rarely develops in such patients; such nodes were almost never biopsied, but there are occasional patients in whom the hilar and mediastinal lymph nodes reduced in size with treatment for the *Pneumocystis* infection. In any event, this case offers an extraordinarily rare presentation of *Pneumocystis carinii* infection.

FINAL ANATOMIC DIAGNOSES

Pneumocystis carinii and cytomegalovirus pneumonia—bilateral, lungs.

Acquired immunodeficiency syndrome (AIDS).

Disseminated granulomatous *Pneumocystis carinii* infection involving pulmonary hilar and mediastinal lymph nodes, spleen, liver, kidneys, bone marrow, pancreas, thyroid, and adrenal glands.

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A medical student's dilemma

Barry A. Solomon

Who would have thought. A friend of mine was recently diagnosed as having AIDS. I had learned about the epidemiology, genetic constituency, medical complications, diagnostic methods, and prognosis of AIDS. For the most part, medical school has prepared me with the scientific data base necessary to practice medicine. Should I visit him? After all, he is hospitalized in another state. I have classes daily and frequent examinations.

I stood at the door to his room. It was 6:30 PM. I had never personally known anyone who had been admitted to a hospital. I pulled the curtain aside to see him but all I saw were nurses, machines, tubes, bags, and fluorescent lights. The sounds were deafening; the smell unforgettable. My mouth became dry.

"Hello, Evan." He did not respond. I took his hand but was unsure of my act's purpose. I cared about him, but how much empathy should I express? Since he is my friend, can I, should I, show my concern and identify with his pain and inner suffering, if that is at all possible, or should I remain the detached professional in order to be his pillar of strength? What is best for him? For me?

Evan had always been a free spirit, autonomous. Now he was intubated. The respirator rhythmically forced oxygen into his lungs, billowing his chest. I focused on his rising and falling chest, mesmerized. How could I, a medical student, a friend, look him in the eyes? He was not the person I used to know. Just a few weeks ago we were together at his mother's house, speaking about unimportant topics and laughing. We are both young. He exercised regularly. I did not. Now he was dying. I did not know who was more nervous or embarrassed. I knew that on learning of his diagnosis he would become ambivalent, turn inward, rebel, and fail to reason.

Confusion. Sympathy or empathy?

I squeezed his hand. His eyes opened and met mine. He stared. What should I do now? Should I speak to him? What should I say? What if I say the wrong thing and worry him more? Evan knew I was going to be a physician and thought I knew almost everything. So he thought. What should I do now? I stared back. His eyelids were partially open, slightly swollen, with sticky white particles in his eyelashes. After what seemed to be an eternity I moved my eyes to look at his body (or to avoid his stare). He was pale and had lost weight. "How are you feeling?" "Looking good." "Are they treating you okay?" Evan communicated with me by moving his eyes. Evan is lucky. I knew his family cared about him and that I cared. We would be understanding, tolerant, and accepting of him. But what of the other patients who lack support and are being treated with impersonal health care?

I continued communicating with Evan although I do not remember the content of that communication. Although I did not tell Evan in so many words, I hoped he would understand that I cared for him and would be there for him when he needed me. I reluctantly restrained most of my emotions.

His nurse came to the bedside and administered a drug through his IV tube to paralyze him so that he could breathe more easily. He was excited and becoming short of breath. My wife tapped me on the shoulder and told me it was 9:00 PM and time to leave. I looked at Evan. How can I leave him like this? He will be all right. Sure he will. What should I say to him? Goodbye? See you later? Will I? Evan stared at me and reached out his hand. I placed my hand within his. He squeezed. I squeezed back. I left my hand there until his hand fell to the bed.

As I was leaving I realized how grateful I should be to my teacher in the doctor-patient relationship course I had taken in medical school. He exposed my mind to certain nuances of patient relations and taught me to recognize certain emotional limits. I used to believe a medical student would, and should, react with innate responses to such situations. I learned that few if any of us are truly prepared for the emotional confrontation awaiting us in our management of patients.

I will be your pillar, Evan. See you later.

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Disseminated *Pneumocystis carinii* infection in the acquired immunodeficiency syndrome

Stefano Ravalli, MD; Roger L. Garcia, MD; Raf A. Vincent, MD; Ruvan Shein, MD

Pneumocystis carinii is the most common opportunistic agent affecting the population with the acquired immunodeficiency syndrome (AIDS); it is diagnosed in about 60% of these patients.[1] However, despite increasingly frequent reports of focal extrapulmonary involvement by *P carinii*,[2-8] extensive multisystemic, extrapulmonary dissemination has rarely been reported. A review of the English literature yielded only six such cases: three in patients infected with the human immunodeficiency virus (HIV),[9-11] and three in patients with other causes of immunosuppression.[12-14] We report on a patient with AIDS with *P carinii* infection characterized by extensive dissemination.

Case Report

A 32-year-old man with AIDS was admitted to the hospital for syncope, diarrhea, and dehydration. He was bisexual and did not use intravenous drugs. The patient had a history of cytomegalovirus (CMV) colitis diagnosed by colonoscopy and multiple biopsies on three different occasions, beginning in July 1987. *Pneumocystis carinii* pneumonia (PCP) had been diagnosed by bronchial biopsy in April 1987 and recurred in July 1987 and March 1988. During the first episode of PCP, a bronchial washing also revealed CMV inclusions. His previous history also included giardiasis and hepatitis B infection.

He was initially treated with intravenous trimethoprim-sulfamethoxazole; his regimen was later changed to intravenous pentamidine. He later received aerosolized pentamidine for PCP prophylaxis every two weeks for more than one year prior to the last admission. The patient also took, from May 1987, oral azidothymidine (AZT), 200 mg every four hours, which was poorly tolerated, requiring occasional interruption of the treatment. During the year and a half prior to the November 1988 admission, the patient's main problem was chronic watery diarrhea and weight loss, requiring multiple admissions for fluid and electrolyte management. In addition, he suffered from headaches, seizures, hallucinations, and a progressive sensory-motor neuropathy. Computed tomographic (CT) scans of the head showed only signs of cerebral atrophy.

At the time of his last admission to the hospital he was cachectic (weight, 49 kg) and afebrile, and had a supine blood pressure of 80/60 mm Hg with orthostasis. He had no cough or sputum production, shortness of breath, or pleuritic chest pain; however, examination of his chest revealed bronchial breath sounds posteriorly over the left upper lung field, and chest radiographs showed bilateral diffuse infiltrates. Significant laboratory data included the following: white blood cell count, 4.0×10^9/L (normal, 5–11 $\times 10^9$/L); hemoglobin, 12.5 g/dL (normal, 14.0–18.0 g/dL); platelet count, 68.0×10^9/L (normal, 150–450 $\times 10^9$/L); total protein, 3.1 g/dL (normal, 6.0–8.5 g/dL); albumin, 1.0 g/dL (normal, 3.0–5.5 g/dL); glucose, 155 mg/dL (normal, 70–125 mg/dL); blood urea nitrogen (BUN), 32 mg/dL (normal, 10–26 mg/dL); creatinine, 1.7 mg/dL (normal, 0.7–1.5 mg/dL); lactic dehydrogenase (LDH), 653 U/L (normal, 60–200 U/L); serum aspartate aminotransferase (AST), 167 U/L (normal, 0–41 U/L); serum alanine aminotransferase (ALT), 68 U/L (normal, 0–45 U/L); alkaline phosphatase, 663 U/L (normal, 30–115 U/L). Despite aggressive hydration and empirical antibiotic treatment, the patient had a rapid downhill course. He remained hypotensive and oliguric, and he died on the fourth hospital day.

Pathologic Findings. The body was emaciated without other externally visible pathologic changes. The right and left lungs were consolidated, weighing 730 g and 700 g, respectively. The visceral pleurae were dotted with multiple gray nodules, measuring 5 mm in average diameter. The lung parenchyma was congested, firm, and subcrepitant. The spleen was enlarged (600 g) and its parenchyma studded with yellow-gray, confluent, nodular infiltrates. The same findings were observed in the kidneys, throughout the cortex and medulla (Fig 1). Scattered, less numerous, gray nodules were present in the myocardium and were visible through the endocardium (Fig 2). Similar minute nodules were present in the liver.

Microscopic examination of the lungs revealed honeycombed, eosinophilic material in the alveoli, within alveolar septa, and filling numerous blood vessels (Fig 3, left). Multiple areas of calcification were also present within this material. The foamy masses had the characteristic appearance of clusters of *P carinii* cysts, and silver methenamine stain (GMS) demonstrated the typical round or helmet-shaped cyst wall with a focal elongated thickening. These distinct conglomerates were also present as infiltrates in mediastinal and retroperitoneal lymph nodes, liver (Fig 3, center), spleen, aorta, heart (Fig 3, right), endocrine and exocrine pancreas, gallbladder, esophagus,

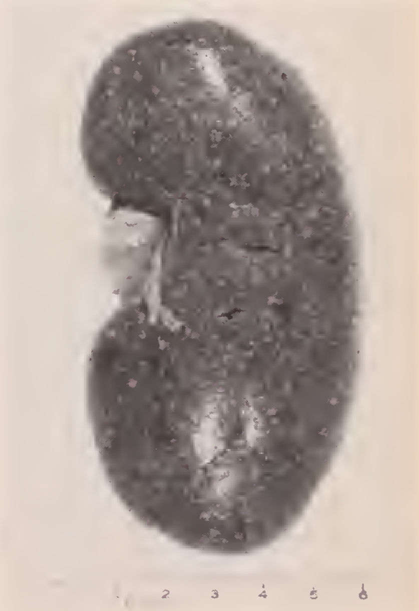

FIGURE 1. The kidneys and other parenchymatous organs were heavily colonized by *P carinii*, visible grossly as small, irregular nodularity throughout the cortex and medulla.

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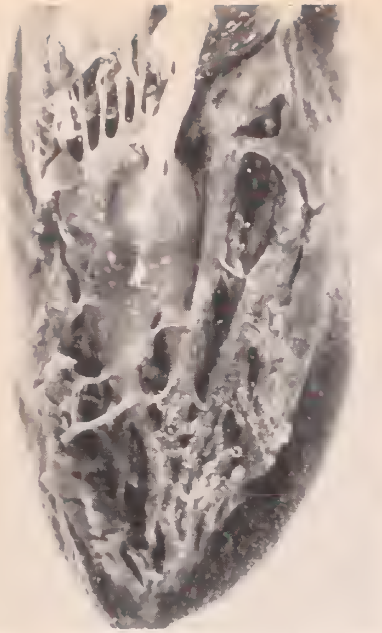

FIGURE 2. Numerous *P carinii* infiltrates could be seen under the endocardium in all chambers of the heart.

stomach, small and large intestines, kidneys, urinary bladder, testes, thyroid, adrenals, sympathetic ganglia, and bone marrow. In many of these sites, the cysts were present within blood vessels, including the vasa vasorum of the aorta and the glomerular capillaries of the kidneys (Fig 4).

In the liver, *P carinii* massively embolized the branches of the portal vein and extended to the sinusoids and central veins, but was not present in the hepatic arteries or bile ducts. No inflammatory reaction was elicited by its presence in any of the organs involved. The alveolar pneumocytes were prominent, but there were virtually no plasma cells. The gastric mucosa exhibited a superficial chronic gastritis, but there was no spatial relation to the scattered *P carinii* infiltrates, and there were no visible inflammatory cells of any kind within the latter. In addition to *Pneumocystis*, cytomegaloviral inclusions were observed in the lungs, adrenals, gallbladder, and gastrointestinal tract, but without erosions or ulcerations in the latter,

Consent for the examination of the brain was not granted in this case; therefore, correlation between the neurologic findings and the central nervous system pathology could not be established.

DISCUSSION

Pneumocystis carinii pneumonia is the most common opportunistic infection in AIDS[1] and accounts for significant mortality in this population.[15] *P carinii* is a eukaryotic microorganism of uncertain taxonomy. Whereas several authors consider it to be a protozoan, others think that it more closely resembles a fungus. Recently, phylogenetic analysis of *Pneumocystis* 16S-like ribosomal RNA (rRNA) has apparently demonstrated it to be a member of the fungi.[16]

The first report of extrapulmonary *P carinii* infection in a patient with AIDS is that of Kwok and coworkers,[6] who demonstrated its presence in the retina of a patient who died of PCP and renal failure. Since then, several other reports have appeared in the literature, describing the involvement of bone marrow,[3] skin,[7] thyroid,[5] temporal bone,[4,17] small intestine,[2] spleen[8] (also M. C. Raviglione, personal communication, January 1989), and liver.[18] In three additional cases,[9-11] widely disseminated infection was reported. Extrapulmonary pneumocystosis has also been reported in patients with immunodeficiency due to pathologic processes other than AIDS,[19-24] with extensive multi-organ involvement in three instances.[12-14]

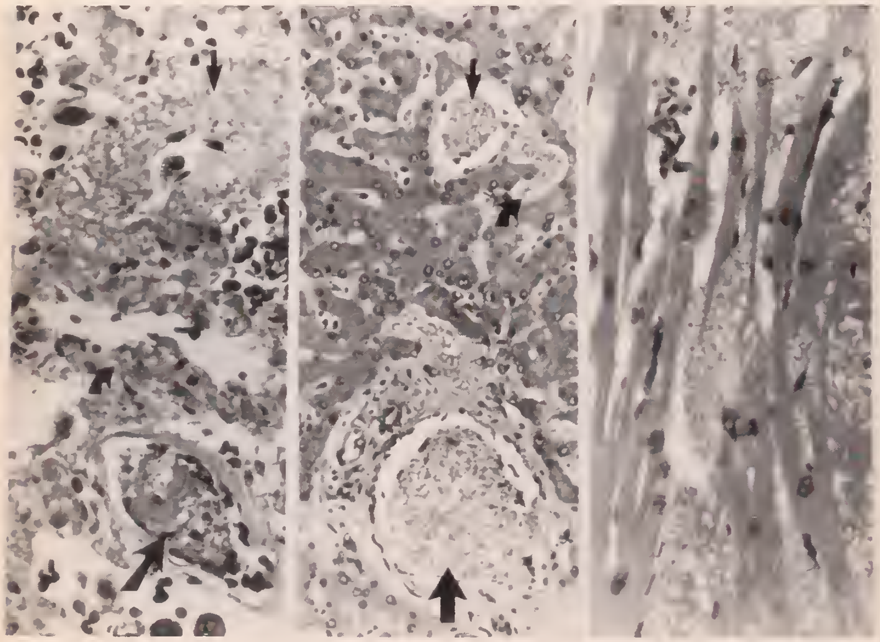

FIGURE 3. **Left,** The lung showed *P carinii* in the alveoli (top arrow), within the septa (center arrow), and in the pulmonary veins (bottom arrow). **Center,** In the liver, *P carinii* embolized portal veins (bottom arrow), sinusoids (center arrow), and central veins (top arrow). **Right,** The myocardium, like other tissues, exhibited numerous organisms, without inflammatory reaction (hematoxylin-eosin stain; original magnification × 235 (left), × 117 (center), × 210 (right)).

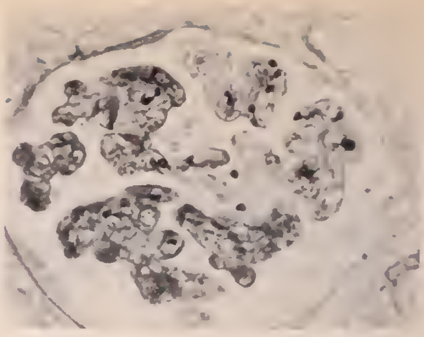

FIGURE 4. *P carinii* cysts in the glomerular capillaries (methenamine silver stain, original magnification × 400).

The present case, with overwhelming dissemination, shows that there is no organ or tissue that is immune to colonization. It also demonstrates that the dissemination takes place from its putative port of entry in the lungs through lymphogenous and hematogenous routes.

It is interesting to note that, from its massive invasion of the spleen and perhaps the intestines, *Pneumocystis* had secondarily colonized the liver through the portal vein, sinusoids, and central veins, thus closing the circuit to the lungs.

It is also of note that this case, and other similar cases, showed no associated inflammatory response. Whether the dissemination follows the total loss of immune response by the host or it precedes and overwhelms it, may be a moot point in this stage of the disease. But it will be of great practical interest to correlate the generalization of the pneumocystosis, in this and similar cases[18] (also M. C. Raviglione, personal communication, January 1989), with the use of aerosolized pentamidine as prophylaxis, to uncover any possible cause-effect relationship. The exact timing of the dissemination may prove to be a more elusive puzzle, since there are no specific clinical tests for *P carinii* and we still depend on its direct visualization in the tissues. This may be reliably obtained in the lungs by means of bronchial washings and transbronchial biopsies,[25] with the aid of special stains (GMS) or by induced fluorescence.[26] However, the diagnosis of pneumocystosis in other organs is more difficult and unreliable. In this patient, colonic biopsies on three different occasions, as well as esophageal and duodenal biopsies, did not disclose the presence of *P carinii* in the gastrointestinal tract. However, the likely possibility of sampling error, of site as well as of organ, precludes any valid conclusion. Only the accumulation of repeated observations may indicate a significant trend, or lack thereof, in the future.

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Erosive arthritis, atypical psoriasis, and *Pneumocystis carinii* pneumonia in an HIV-positive man

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The cutaneous manifestations of the acquired immunodeficiency syndrome (AIDS) range from seborrheic dermatitis to Kaposi's sarcoma.[1] Psoriasis and AIDS have previously been reported to occur in association, although this may only represent the chance co-occurrence of two common illnesses. The seronegative arthritis of Reiter's syndrome has likewise been reported in association with AIDS. This report presents a case of erosive seronegative arthritis and psoriasis as presenting manifestations in a patient with AIDS and *Pneumocystis carinii* pneumonia.

CASE REPORT

A 41-year-old heterosexual male was admitted to the hospital because of fever, cough, and a skin rash. He denied all risk factors for infection with human immunodeficiency virus (HIV) except for an accidental needle stick (while working in a dentist's office) one year earlier. Eight months before admission, a pruritic, erythematous, papular rash developed first on his right leg but gradually spread to involve all four extremities, his back, and scalp. Early in the illness, he was diagnosed as having shingles and was given an oral antibiotic without obtaining relief. Five months before admission, pain developed in both ankles and in the third and fourth proximal interphalangeal (PIP) joints of the right hand. One week before admission he began to have a cough productive of white sputum, dyspnea, and fever with rigors. There was no history of tuberculosis or hepatitis, he had no drug allergies, and he used no medications.

The patient's temperature was 39.1°C; blood pressure, 130/80 mm Hg; and pulse, 105 beats/min. Examination of the skin revealed scaling and excoriations of the scalp; numerous plaques with erythematous borders and hyperkeratotic, scaling centers distributed over his arms, legs, and back; and smaller erythematous papules in the same distribution (Fig 1). His nails were dystrophic, but not pitted. The conjunctivae were noninjected. There was a thick, potassium hydroxide-positive exudate on the oropharynx but no oral ulcers. There was no significant adenopathy. The chest was clear, and cardiac auscultation was normal. There were no genital skin lesions and no urethral

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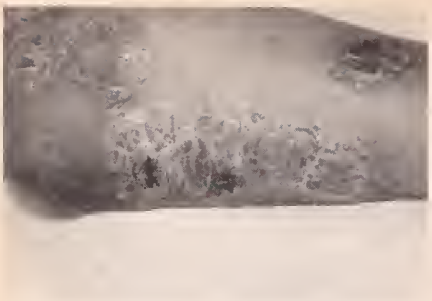

FIGURE 1. Hyperkeratotic plaques with erythematous borders on the extensor surface of the elbow.

exudate. There was significant tenderness, warmth, and erythema of the right ankle, with a small effusion and limitation of passive motion. Effusions were also present at the third and fourth PIP joints of the right hand, with limited extension of the fourth digit. The neurologic examination was within normal limits.

The hermacrit was 36%, and the white blood cell count was 8,700 cells/mm^3 with 83% polymorphonuclear leukocytes and 3% eosinophils on the peripheral blood smear. Analysis of arterial blood gas on room air showed a pH of 7.45; P_{CO_2}, 30 mm Hg; and P_{O_2}, 74 mm Hg. A serum VDRL test was nonreactive. A test for rheumatoid factor was negative. The chest roentgenogram showed a diffuse, bilateral, reticulonodular infiltrate. Roentgenograms of the hands and ankles demonstrated narrowing of the right fourth PIP joint space (Fig 2). Urinalysis was normal. The electrocardiogram showed sinus tachycardia.

Empiric drug therapy with intravenous trimethoprim-sulfamethoxazole was initiated. On the fourth hospital day, after three sputum specimens were negative for *Pneumocystis carinii* by the Gram-Weigart method, the patient underwent bronchoscopy. Bronchoalveolar lavage fluid and brush specimens were positive for *Pneumocystis carinii*. Anti-HIV antibody was present (by ELISA and Western blot). Skin biopsies, obtained on the first hospital day, were consistent with psoriasis, although they were atypical due to the presence of eosinophils in the cellular infiltrate and the unusual depth of the infiltrate, which extended to the eccrine sweat glands (Fig 3). HLA typing showed the patient to be positive for the B27 antigen (as well as A2, A3, B14, Cw2, and Cw8).

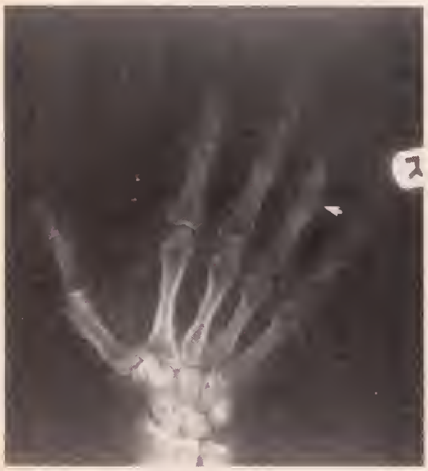

FIGURE 2. Roentgenogram of the right hand demonstrating narrowing of the fourth PIP joint space.

DISCUSSION

An association between infection with the human immunodeficiency virus and psoriasiform skin lesions has previously been noted by several authors. Duvic and collaborators[2] found 13 cases of psoriasis in a population of more than 1,000 patients with AIDS or AIDS-related complex (ARC). Guttate lesions were seen in 11 of the 13 patients, an unusually high proportion. In nine of these patients, skin lesions developed concurrently with the development of symptomatic HIV disease. Lazar and Roenigk[3] reported on seven patients with psoriasis and either AIDS or ARC. The onset of AIDS or ARC occurred within two years of the development of the skin lesions in two of these patients.

Reiter's syndrome (RS), a disease with several features similar to psoriatic arthritis, has also been demonstrated to occur in association with HIV infection. Winchester et al[4] saw 13 HIV-positive patients with RS. In four of these patients, the arthritis developed within one month of the onset of AIDS or ARC. Duvic's series of patients with psoriasis and AIDS included two patients with arthritis, urethritis, and conjunctivitis, and another with conjunctivitis and urethritis but without arthritis—all possible cases of RS.[2,5]

A search of the English language literature revealed three cases of psoriatic arthritis and AIDS. Winchester et al[4] described a patient, with a ten-year history of a seronegative symmetric polyarthritis, in whom extensive psoriasiform skin lesions developed concurrently with the onset of lymphadenopathy, oral thrush, and pancytopenia. Lambert and Kaye[6] described a patient with psoriasis and distal interphalangeal joint arthritis in whom AIDS developed five months after beginning methotrexate therapy. Lazar and Roenigk[3] included one patient with psoriatic arthritis in their series.

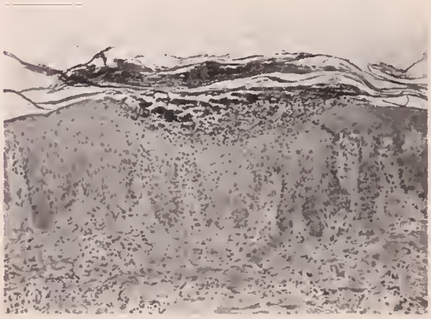

FIGURE 3. Spongiotic pustule (hematoxylin-eosin stain; original magnification ×10).

These cases, as well as the case presented here, demonstrate an association between psoriasis, seronegative arthritis, and symptomatic HIV infection. In this patient, widespread psoriasiform skin lesions and an erosive seronegative oligoarticular arthritis developed eight months and five months, respectively, prior to the diagnosis of *Pneumocystis carinii* pneumonia. In analyzing this association, it is suggestive that Duvic and colleagues[7] found the prevalence of psoriasis to be 1–2% in their patients, the same as the prevalence of disease in the population as a whole. The cases, then, may simply reflect the simultaneous occurrence of two common diseases.

Although psoriasis may not occur with greater frequency in the AIDS population, when it does occur, its severity appears to be greatly increased. Virtually all reports have emphasized the explosive and extensive nature of the skin lesions in HIV-associated psoriasis. There is often a close temporal association between the onset of skin lesions and the development of frank immunodeficiency.

We hypothesize that although HIV infection does not increase the incidence of psoriasis, it leads to increased severity of disease in patients with a pre-existing psoriatic diathesis. This increased severity is reflected in an extensive, and often atypical, distribution of the skin lesions, a refractoriness to therapy, and, perhaps, an increase in the extracutaneous manifestations of disease, such as arthritis.

Alternatively, it could be the case that the apparent increase in severity reflects selection bias, and that HIV-infected patients suffer from an increased incidence of less severe psoriasis that has not come to our attention. This is unlikely, however, because most patients with AIDS do come under medical attention.

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Deep venous thrombosis in patients with AIDS

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***Pneumocystis carinii* pneumonia** and Kaposi's sarcoma were first reported in male homosexuals with the acquired immunodeficiency syndrome (AIDS) in 1981.[1,2] Since that time, the range of illnesses and laboratory abnormalities associated with AIDS has continued to grow. In patients presenting with painful leg swelling, edema, and a positive Homans' sign, the diagnosis of deep venous thrombosis (DVT) must be considered. However, hyperalgesic pseudothrombophlebitis, recently reported in male homosexuals with AIDS, mimics deep venous thrombosis.[3] We recently treated five patients with AIDS with leg swelling and tenderness who did have deep venous thrombosis. The data from these five cases form the basis of this report.

PATIENTS AND METHODS

The charts of all patients with deep venous thrombosis and AIDS admitted to the Long Island Jewish Medical Center and Queens Hospital Center over the previous four years (1984–1988) were reviewed. During that period of time, we treated 75 patients with positive DVT on venogram and approximately 400 patients with AIDS. Five patients were found to have documented deep venous thrombosis and AIDS. The charts of these five patients were reviewed for age, time from diagnosis of AIDS to presentation of the deep venous thrombosis, location of the deep venous thrombosis, hematocrit, white blood cell count, platelet count, and albumin level. Associated medical problems including all associated infections and all current medications were reviewed.

CASE REPORTS

Case 1. A 34-year-old man was diagnosed as having AIDS two months prior to admission. He denied intravenous drug use and homosexual activity and claimed to have contracted the disease through heterosexual contacts with prostitutes. The diagnosis was made by a bronchoalveolar lavage that was positive for *Pneumocystis carinii*. The diagnoses on admission were oral candidiasis and tuberculosis. He was placed on clotrimazole, pentamidine, azidothymidine, isoniazid rifampin, pyrazinamide, and sulfadoxine. He did well and was discharged from the hospital.

Two months later he presented to the emergency department complaining of a diffuse erythematous rash. A diagnosis of erythema multiforme was made; sulfadoxine and pyrimethamine were deemed the causative agents and were discontinued. While in the hospital, he complained of pain in the right calf. Physical examination of the calf revealed a swollen, tender extremity with no palpable cord. A presumptive diagnosis of deep venous thrombosis was made, and a venogram demonstrated multiple filling defects in the calf veins, with no thrombus seen in the popliteal, femoral, or iliac veins. Heparin therapy was started, with subsequent improvement of symptoms. The patient was discharged with continued warfarin sodium therapy and has done well.

Case 2. A 48-year-old man was admitted to the hospital with complaints of extreme weakness, fever, chills, cough, and episodes of falling. The patient had been tested for AIDS five months prior to admission, but no diagnosis was made at that time. The patient's risk factor for AIDS was homosexual activity. On admission, the patient was febrile (102°F) and had a white blood cell count of 13,000/mm^3. He was noted to have oral candidiasis, rectal herpes, and a left lower lobe infiltrate. Subsequent cultures were positive for *Pseudomonas*, *Aeruginosa*, and *Candida*. The patient was started on rifampin, acyclovir, ticarcillin, amikacin, ceftazidine, and amphotericin B. While in the hospital, the patient's right thigh became swollen and cyanotic. A diagnosis of phlegmasia cerulea dolens was made, and a venogram revealed complete thrombosis from the ankle to the common femoral vein; the inferior vena cava was not involved. The patient was started on heparin therapy, and his symptoms improved. On the tenth day the patient was noted to be tachypneic; an arterial blood gas was significant for hypoxemia. A diagnosis of a pulmonary embolus was made, and since the patient had adequate anticoagulation factors, a Greenfield filter was inserted. The patient's overall condition continued to deteriorate, and the family requested no further intervention. He died two months later. Autopsy findings demonstrated thrombosis of the right femoral and iliac veins with occlusion of the lower vena cava. In addition, the patient had bilateral pulmonary infarcts and a pulmonary embolus, as well as severe *Mycobacterium* pneumonia, cytomegalovirus pneumonitis, and acute bronchopneumonia.

Case 3. A 48-year-old homosexual male was admitted to the hospital for evaluation of a mass in the left lower abdominal quadrant. The patient was known to have AIDS-related complex on the basis of human immunodeficiency virus (HIV) testing performed four years previously. On admission, the patient was taking azidothymidine and trimethoprim-sulfamethoxazole. Eight days prior to admission, the patient presented to the emergency department complaining of pain and swelling of his left thigh. A venogram performed at that time was negative for deep venous thrombosis. On admission, the patient was still complaining of pain and swelling of the thigh. Physical examination revealed a firm, non-tender mass in the left lower quadrant and a swollen, warm, tender thigh. A computed tomographic (CT) scan obtained earlier was read as a left lower quadrant mass and left iliofemoral thrombosis. Noninvasive studies were consistent with bilateral external iliac and femoral deep venous thrombosis. An inferior vena cavogram was obtained before surgery and showed a patent inferior vena cava. The patient was taken to the operating room for

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an exploratory laparotomy and biopsy of the mass. Histological examination revealed a B cell immunoblastic lymphoma. After surgery, heparin therapy was instituted, as was a chemotherapy regimen of vincristine, doxorubicin hydrochloride, methotrexate, and bleomycin. The thigh pain and swelling resolved with shrinking of the tumor mass. The patient did well and was discharged to home.

Case 4. A 27-year-old homosexual man was found to have AIDS six months prior to admission. He had had an open lung biopsy positive for *Pneumocystis carinii* and had been treated with pentamidine and doxorubicin hydrochloride. In the intervening six months the patient had been in the hospital twice for non-*P carinii* pneumonia. At this time, the patient presented to the emergency department with a two-week history of pain and swelling of his right calf. On physical examination, the calf was swollen and tender, with a positive Homan's sign. A venogram demonstrated right calf deep venous thrombosis with no iliofemoral extension. The patient was treated with heparin and warfarin sodium and was discharged to home.

Case 5. A 39-year-old man was admitted to the hospital for dehydration. A diagnosis of AIDS had been made two years prior to admission when he presented with *Pneumocystis carinii* pneumonia. The patient's risk factors were a history of intravenous drug use, and blood transfusions secondary to a stab wound. At the time of admission, he was noted to have oral candidiasis and rectal herpes. While hospitalized, a *Mycobacterium avium-intracellulare* bacteremia developed. He was treated with amphotericin B, ansamycin, acyclovir, trimethoprim-sulfamethoxazole, clofazimine, isoniazid, and clotrimazole. During this hospital stay, he was noted to have a swollen and tender right leg. A venogram revealed extensive deep venous thrombosis involving the calf and thigh veins; the iliofemoral segment was clear. The patient underwent heparin therapy and was subsequently started on warfarin sodium with improvement of symptoms. The patient's respiratory symptoms continued to worsen. He required intubation and mechanical ventilation, and eventually a tracheostomy. The patient remained dependent on a ventilator until he died four months later.

Results. The clinical parameters for the five patients are listed in Table I. Follow-up was obtained in all patients. All patients were male, with a mean age of 39.2 years. Four of the five were homosexuals, and one patient was an intravenous drug abuser. The mean time from diagnosis of AIDS to the time of deep venous thrombosis was 18 months. Three of the patients were found to have DVT while in the hospital for treatment of AIDS-associated diseases. DVT was localized to the calf in two patients and extended to the thigh in three patients. The mean hematocrit at the time of diagnosis was 36%; mean white blood cell count, 8.1; mean platelet count, 354,000; and mean albumin level, 2.8. All patients who had infectious complications were on a multitude of medications at the time of the DVT; three patients were on isoniazid and three on trimethoprim-sulfamethoxazole at the time of DVT. One patient required insertion of a Greenfield filter for pulmonary embolism while on heparin.

TABLE I. Clinical Findings in Five Patients with AIDS and Deep Venous Thrombosis

Parameters	Mean Values and Standard Deviation
Age (yrs)	32.9 ± 9.0
Time in months from diagnosis of AIDS to deep venous thrombosis	18.2 ± 20
Location of deep venous thrombosis: Calf	4/5
Extension to thigh	3/5
Hematocrit	36% ± 4.8%
White blood cell count	8.1 ± 4.1
Platelet count	354,000 ± 105,800
Albumin level	2.98 ± 0.89

DISCUSSION

More than 24,626 cases of AIDS had been diagnosed in New York City as of January 1990, which represented 21% of the nationwide total (personal communication, New York City Department of Health AIDS Surveillance Team, February 1990). Each month more than 300 new cases are diagnosed, and AIDS has now surpassed trauma as the leading cause of death in men 25-44 years of age and women 25-34 years of age.[4,5] As our knowledge and experience with AIDS increases, the variety of both opportunistic infections and non-opportunistic disorders associated with this disease broadens. Despite the overall younger age distribution, these patients are subject to all the complications known to seriously ill patients. This report describes five male AIDS patients who developed deep venous thrombosis with no known risk factors. As pulmonary embolus remains a major source of morbidity and mortality, it is essential to diagnose and treat deep venous thrombosis as rapidly as possible. In addition to cellulitis, popliteal cysts, and tendon rupture, Abramson et al[3] have recently described a new syndrome, hyperalgesic pseudothrombophlebitis, which closely resembles DVT in male patients with AIDS.[3] This disorder presents in an identical fashion but without filling defects on venography.

The data from these five patients indicate that the development of DVT in patients with AIDS occurs most commonly in males who have an albumin level of less than 3. The diagnosis in these series was made by venogram in four patients and by CT scan in the one patient who had a negative venogram eight days prior to admission. Because of the multitude of associated medical problems and pharmacologic agents usually used to treat these patients, we believe that the decision to use anticoagulation therapy in these patients should be based on a definite diagnosis. Venography remains the diagnostic test of choice to identify those patients who require anticoagulation and should be used aggressively to rule out the diagnosis.

The location of the DVT, patient age, and hospital course all seem unrelated to the development of DVT. The presenting symptoms ranged from calf pain and swelling to the characteristic swollen cyanotic thigh of phlegmasia cerulea dolens. It would seem that bed rest is an unlikely causative factor, since two patients were ambulatory at the time of diagnosis. We could not directly implicate a hypercoagulable state in any patient, although low albumin levels have been associated with deep venous thrombosis in patients with cancer. It has been postulated that malnutrition results in decreased liver production of noncritical proteins such as antithrombin III, resulting in a relative hypercoagulable state.[6]

The array of clinical pathologic factors associated with these immunocompromised hosts continues to increase and surprise the treating clinician. Deep venous thrombosis is particularly worrisome, as its sequelae can lead to sudden death in these young patients. This report indicates that DVT is a real problem in the patient with symptomatic HIV infection. Malnourished patients with AIDS who have limb swelling and calf tenderness should be aggressively evaluated by venography for DVT. Anticoagulation with heparin followed by warfarin sodium remains the mainstay of therapy in the patients with positive venograms.

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The Milton Rosenberg, MD, Memorial Award for Residents in Obstetrics-Gynecology

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Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Resolution of cavitary *Pneumocystis carinii* pneumonia

TO THE EDITOR: We recently reported in the *Journal*[1] a case of a patient with *Pneumocystis carinii* pneumonia (PCP) who presented with multiple pulmonary parenchymal cavitations (Fig 1). Although cavitations remain an unusual roentgenographic finding in PCP, several reports similar to ours have appeared in the recent literature.[2-4] The natural history of these lesions in those who survive has not been documented.

The patient we described in our report began to show roentgenographic improvement of her pulmonary process during her prolonged hospital stay. Following discharge she was reluctant to have further radiologic examinations. Fifteen months after her initial presentation with PCP she returned to our emergency department with complaints of weakness and dyspnea on exertion. She was found to be markedly anemic. Her chest roentgenogram was free of infiltrates or cavities (Fig 2). She underwent transfusion and was discharged for outpatient follow-up.

The isolated reports of cavitary PCP that have appeared in the literature thus far have not defined the roentgenographic course of these lesions. Complete radiologic resolution was an unexpected finding in our case.

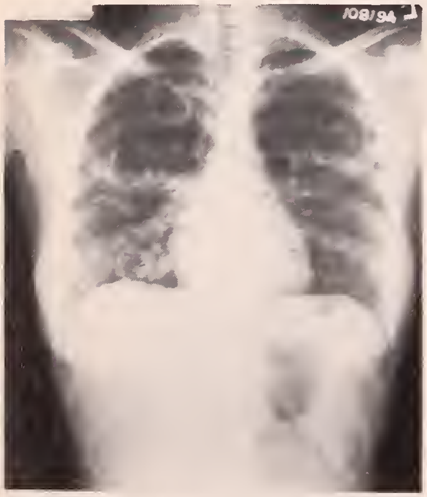

FIGURE 1. Chest roentgenogram from patient's hospital admission demonstrating multiple pulmonary parenchymal cavitations and interstitial infiltrates.

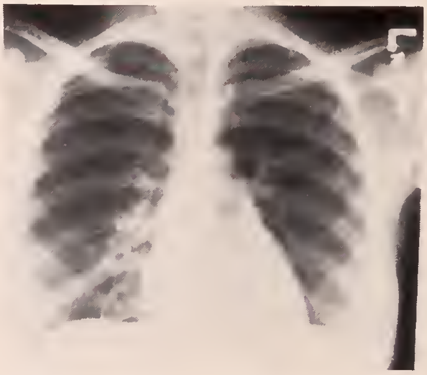

FIGURE 2. Fifteen months later, complete resolution of the previous findings.

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Multifocal human papillomavirus infections of the genital tract in HIV seropositive women

TO THE EDITOR: Recent findings have suggested a high prevalence of cervical human papillomavirus (HPV) infection and/or cervical intraepithelial neoplasia (CIN) in human immunodeficiency virus (HIV) seropositive women.[1] Multicentric HPV infection of the genital tract is frequent in the general population with CIN. We investigated 53 consecutive confirmed HIV infected women with a mean age of 28.2 years (range, 19-67 years) for evidence of cervical and vulvar intraepithelial neoplasia. All were asymptomatic (CDC group II). We performed cervical smears in all 53 patients (100%), as well as colposcopy of the cervix (70%) and vulva (57%) and directed biopsies (60%) whenever the previous investigations were abnormal.

Thirty-seven cervical smears (69.8%) showed evidence of HPV infection, either isolated (41.5%) or associated with CIN (28.3%). Colposcopic patterns suggested HPV infection in 78.4% of the patients examined.

Macroscopic examination of the vulva showed genital warts in 17 patients (32%), three of whom had associated herpesvirus infections. Small vulvar lesions which were barely visible macroscopically were best assessed using the colposcope after addition of 5% acetic acid. Four papulae, four leucoplakiae, and 11 acetowhite reactions were thus diagnosed. The histological findings are reported in Table I.

Our findings indicate that the virus may be present in any part of the genital tract. Sixty percent of our patients had multicentric HPV infection. Thor-

TABLE I. Cervical and Vulvar Biopsy Findings

Findings	Number (N = 32)	%
Cervix (32 biopsies)		
Human papillomavirus (flat condyloma)	17	53.0
Human papillomavirus + cervical intraepithelial neoplasia I	3	9.4
Human papillomavirus + cervical intraepithelial neoplasia II	9	28.3
Human papillomavirus + cervical intraepithelial neoplasia III	2	6.2
Human papillomavirus + invasive cancer	1	3.1
Vulva (31 biopsies)		
Normal	7	26.6
Condyloma acuminata	10	32.3
Flat condyloma	5	16.0
Vulvar intraepithelial neoplasia I	5	16.0
Vulvar intraepithelial neoplasia II	3	5.9
Herpesvirus	1	3.2

ough examination of the vulva appears to be contributive to diagnosis. The use of the colposcope to perform vulvoscopy allows for better diagnosis of small lesions. In our experience, VIN appeared to be associated only with papules and hyperkeratosis surface.

Sexual practices are likely to account at least partly for the association between HIV and HPV infection. As in most previous studies, this population is at high risk for sexually transmitted diseases. Thirty-three (62.5%) are or had been parenteral drug users, 20.7% are from endemic regions of Africa, 9.5% were contaminated by HIV positive partners and only 7.5% were infected by blood transfusions. Epidemiologic studies are underway to determine whether latent immune deficiency in HIV positive patients favors infection and/or its evolution toward neoplasia. Furthermore, analysis of biopsy specimens for papillomavirus sequences by Southern blot hybridation should allow for comparison with HIV negative populations[2] and with non-HIV-immunodeficient populations (eg, renal transplant patients).

The frequency of HPV infections emphasizes the importance of their diagnosis and treatment in HIV seropositive women. Transformation of cervical carcinoma in situ into invasive cancer may be rapid.[3] Conversely, the potential for transformation of vulvar HPV lesions is small. However, microtrauma and irritation created by the vulvar lesions may be a risk factor for transmission of HIV at the time of sexual intercourse.[4] This may be an argument for treating vulvar as well as cervical lesions.

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Physicians needed to work with AIDS patients

TO THE EDITOR: Recently, a news program on the National Public Radio emphasized the despair of the head of the AIDS ward of Harlem Hospital in New York City who desperately needs physician coverage of a ward with ever increasing numbers of patients. Some of the Veterans Administration hospitals are in a similar though less desperate plight.

Because of social security and individual retirement accounts and because of the onus of malpractice and difficulties with Medicaid and Medicare, increasing numbers of healthy older physicians are retiring. They represent a source of part-time help for institutions in need of them. Could not these physicians be recruited to work one week a month or so, with the hospitals providing malpractice coverage?

Work after social security is not very profitable. However, the pleasure of being useful and maintaining old and acquiring new skills make monetary rewards insignificant.

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AIDS legislation needed

TO THE EDITOR: As the complex interplay of medical, psychological, sociological, and economic features of the acquired immunodeficiency syndrome (AIDS) epidemic has evolved, attempts to control this deadly disease have demanded a multifaceted approach employing both therapeutic and preventive measures. Education of individuals at highest risk for human immunodeficiency virus (HIV) infection has been given the highest priority, and emphasizes avoidance of dangerous drug-associated needle sharing and sexual behavior most likely to transmit disease. For the clinician, the duty to warn has supplanted the traditional code of doctor-patient confidentiality in the greater name of public health and the protection of society.

In recent years at our institution, we have been confronted with several tragic instances of endangerment or assault wherein HIV-seropositive persons have knowingly exposed or infected peers or partners by virtue of their own unethical or malicious actions. Like others in the legal and law enforcement communities, we reiterate that statutory legislation must be enacted that would make incarceration mandatory for HIV-seropositive individuals who knowingly engage in high risk sexual and drug using activity without disclosing their infected status to others. For example, such legislation would make it a serious criminal offense for HIV-infected prostitutes to engage in sex with clients, and for any infected person whose continued homosexual or heterosexual activities knowingly endanger the lives of uninformed partners. The irresponsibility and immorality of such actions have far-reaching implications, and threaten the safety not only of individuals but of society at large through continued, unsuspected transmission of this lethal agent. Strict criminal laws are needed to protect the rights and lives of innocent citizens.

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Baseline survey for the Healthy Heart Program

In 1985, New York State had the nation's highest age-adjusted rate of mortality among men and women from coronary heart disease.[1] Data from the past 20 years show that, relative to other states, New York has exhibited a long-standing tendency toward increased mortality from heart disease.[2] Race-specific analyses conducted by the Mary Lasker Heart and Hypertension Institute of the New York State Department of Health indicated that coronary heart disease mortality in New York was elevated for nonwhites as well as for whites.

In response to this problem, the New York State Department of Health initiated the New York State Healthy Heart Program, an effort to apply what has been learned from broad-based interventions for coronary heart disease prevention in the United States[3-5] and Finland.[6] Organizations in eight sites across the state are working with local media, schools, restaurants, grocery stores, employers, health care providers and other community institutions to develop strategies for improving the cardiovascular health of local residents.

The locations of the intervention sites are shown in Figure 1. Projects outside New York City include Niagara County, Tompkins and Cayuga counties, Otsego and Schoharie counties and the City of Yonkers. Within New York City, interventions are being planned for Washington Heights, Central Harlem, East Harlem and Bedford-Stuyvesant.

One measure of the impact of the Healthy Heart Program will be change over time in the level of preventive practices and risk factors for coronary heart disease, as measured by random telephone surveys of residents in the intervention communities. The respondents were questioned about cardiovascular risk factors, such as sex, age, race, blood pressure, smoking, dietary fat and cholesterol, obesity and sedentary lifestyle. The baseline survey for the Healthy Heart Program was conducted in the beginning of 1989, and selected preliminary findings are described in this report. The telephone survey questionnaire was modeled after those used by the Centers for Disease Control Behavioral Risk Factor Survey and the South Carolina Cardiovascular Disease Prevention Project. Telephone exchanges were identified for the intervention counties and urban areas, and a dual frame sampling design was employed to randomly identify one respondent (between 18 and 64 years of age) per household for an interview. Substitutions were not allowed within the contacted household, and a maximum of four attempts were made to interview the selected individual. The interview lasted approximately 45 minutes, and 66% of eligible adults in the sampling frame (for all sites combined) agreed to participate.

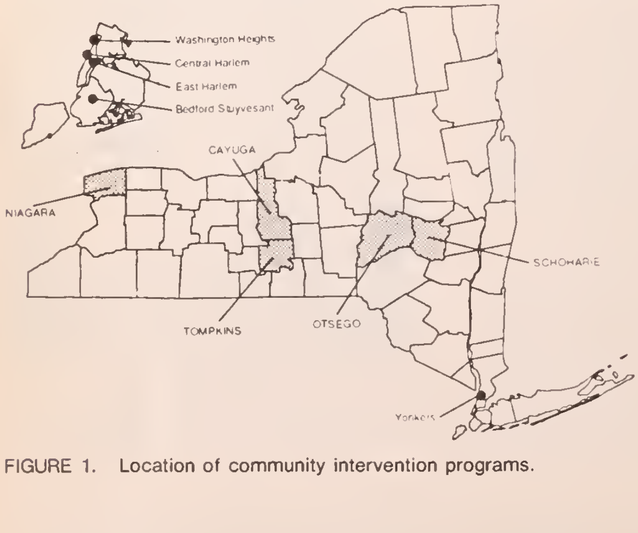

FIGURE 1. Location of community intervention programs.

Characteristics of respondents from the eight intervention sites are summarized in Table I. There was little variation by age or sex; the average age of respondents was near or at age 40 and the majority of respondents (55% to 61%) were women. There were substantial and predictable differences in race, with largely white samples outside of New York City, but combinations of black, Hispanic and white in the communities of New York City. Response rates also varied somewhat. Participation was highest in the rural sites and East Harlem, and lowest in Yonkers and the remaining sites of New York City. The continuing evaluation of the Healthy Heart Program will attempt to examine the impact of selective response to this and other efforts at data collection.

The survey included questions on the recency of measurement of blood pressure and cholesterol. Responses are shown for both risk factors in Figure 2. The data indicate that annual blood pressure checks are uniformly common across the intervention sites. This reported level of blood pressure monitoring is consistent with data from the National Center for Health Statistics[7] and suggests that the Healthy Heart Program need not create new opportunities for adults to have their blood pressure measured. However, based on additional data from the survey, the Healthy Heart Program may work with existing resources for hypertension screening and management to improve the follow-up of diagnosed patients and to reduce barriers to compliance. The data on cholesterol screening reflect the growth of interest and activities related to this subject. The 1987 Behavioral Risk Factor Survey found that only 33% of adults in New York had *ever* had their cholesterol tested. This represented the second lowest value of 33 participating states.[8] Two years later, a higher proportion of adults (43% overall) reported having cholesterol screened in the past year alone. However, there is still need for improvement in the level of this preventive practice, particu-

TABLE I. Characteristics of Respondents to the Healthy Heart Program Baseline Telephone Survey, By Intervention Site

	Niagara	Yonkers	Otsego/ Schoharie	Cayuga/ Tompkins	Washington Heights	Central Harlem	Bedford-Stuyvesant	East Harlem
Number of Respondents	506	506	250	450	851	515	501	600
Mean age	40	40	39	38	38	39	38	39
% Male	46	44	44	46	41	45	39	40
% Black	4	8	2	2	16	68	74	42
% Hispanic	<1	3	<1	1	46	11	14	28
% White	93	81	95	92	32	13	6	23
% Participation	66	61	74	79	60	60	60	73

larly in the rural counties.

Risk factors that have been identified as targets for intervention in the Healthy Heart Program are high blood pressure, smoking, excessive dietary fat and cholesterol, obesity and sedentary lifestyle. Estimated levels of sedentary lifestyle are shown by intervention site in Figure 3. In contrast to cholesterol screening (where rural counties may be lagging behind), sedentary behavior is least common in the rural areas and most prevalent in three of the four sites in New York City. The level of inactivity described in this survey is similar to values estimated for the US adult population.[9] However, in a 1987 Centers for Disease Control report that combined the completely sedentary with those who exercise at a level below that necessary for cardiorespiratory benefit,[10] New York ranked last in physical activity out of the 33 states in the survey. Therefore, environmental changes that would encourage increased physical activity, such as the organization of exercise clubs or better marking and lighting on jogging trails, may be especially appropriate for the local Healthy Heart Program to recommend and implement.

Further analyses of these data are now being performed to suggest appropriate strategies to reduce high risk factor levels in various subgroups. This information will be useful to the individual Healthy Heart programs as they plan their interventions. It will also be compared with responses from similar survey data being collected in other parts of the state to characterize the cardiovascular risk status of the residents of these communities.

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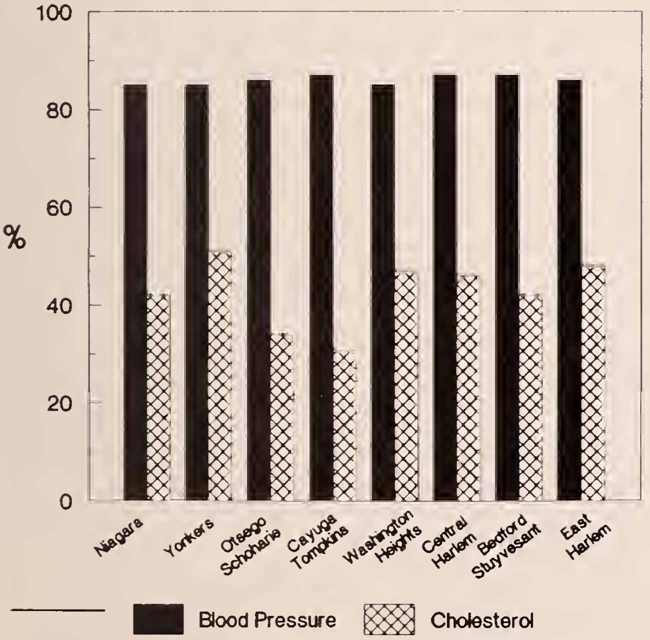

FIGURE 2. Percent of respondents who have had blood pressure and cholesterol measured in the past year, by intervention site.

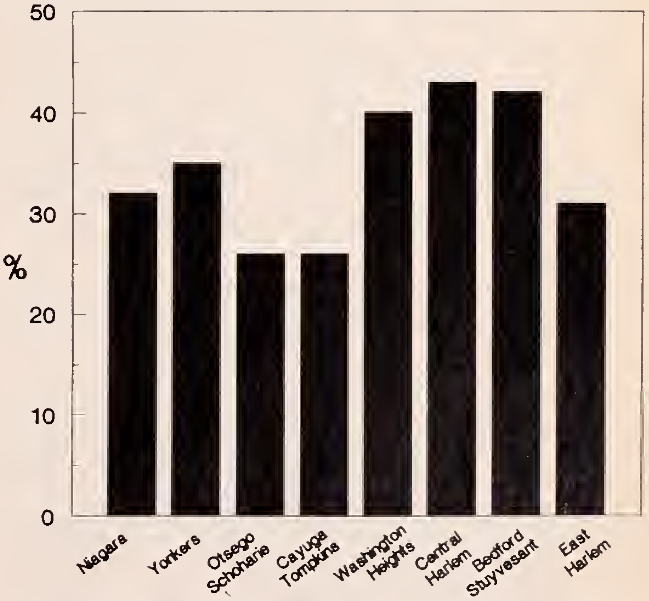

FIGURE 3. Percent of respondents reporting no physical activity for leisure, by intervention site.

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Screening for congenital syphilis begins

Effective December 6, 1989, all babies born in New York State are required to receive serological screening for exposure to syphilis. Such screening will allow for early diagnosis, treatment and follow-up of the newborn with congenital syphilis.

Reported cases of congenital syphilis have increased more than six-fold in New York State in the last three years, from 57 cases in 1986 to 377 cases in 1988; between 800 and 1,000 cases are expected in 1989. These congenital syphilis cases are a direct result of an increase in early infectious syphilis in women. Early syphilis has more than doubled, from 4,607 in 1986 to 10,749 in 1988. Almost half the cases were in women; many of these women became pregnant while infected.

Although prenatal screening for syphilis is required, many women who deliver an infant with syphilis are members of groups that traditionally receive inadequate prenatal care (ie, age 18 or under, unmarried and of low socioeconomic and educational status). Other women may be negative for syphilis when tested early in pregnancy, but become infected between screening and delivery.

To diagnose those infants born with syphilis, the new regulation (Title 10, Subpart 69-2, of the Official Compilation of Codes, Rules and Regulations of the State of New York) includes the following:

- All infants born after 22 weeks, whether alive or dead, must have a sample of blood removed from the umbilical cord and submitted to an approved laboratory for standard serological tests for syphilis.
- If, at time of birth, the body blood from the mother is tested for syphilis, the cord blood test requirement is waived. The infant's body blood must be tested after any positive test result of the mother's blood.
- Bloods submitted for testing must be clearly marked as cord, newborn or mother/delivery to facilitate reporting; all samples must be tested with a quantitative nontreponemal test. Any positive test result must be confirmed by a treponemal test on the *same* sample.
- All positive serologic results must be reported to the local health department within 24 hours of receipt of the test results.
- The responsible physician or birth attendant must ascertain that both the mother and the infant are treated with appropriate therapy, and that follow-up is arranged. The responsible physician or birth attendant must cooperate with the designated public health investigators to assure that diagnosis, therapy and any necessary follow-up testing meet current standards of care.

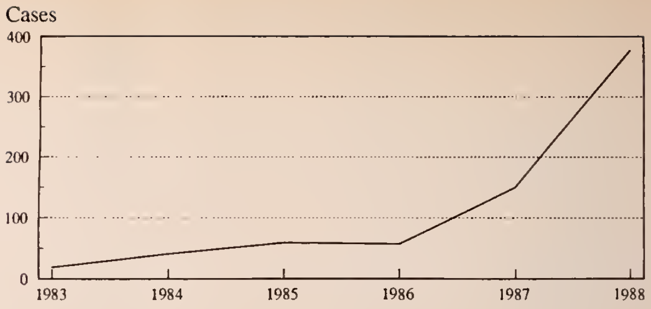

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FIGURE 1. Congenital syphilis in New York State: confirmed and compatible cases, 1983–1988.

National and state statistics document that at least half of all women giving birth to congenitally infected infants have received *no* prenatal care. Therefore, it is imperative to test all infants at the time of birth since many carry no detectable signs or symptoms of the disease. Serious consequences of syphilis in the infant may not become obvious until months or years later when bone deformities, organ destruction or central nervous system damage may be revealed.

Questions regarding diagnosis, treatment or case follow-up of cases of early or congenital syphilis may be directed to the New York Sexually Transmitted Disease Control Program, (518) 474-3598.

Toy safety

The Consumer Product Safety Commission (CPSC) reports that more than 100,000 people are injured by toys each year. Nearly 85% of these injuries involve children under 15 years old and over half involve children under five. Most injuries result from falls or collision with toys and are relatively minor. However, severe injuries do occur. Fifty-two toy-related deaths were reported to CPSC from January 1986 through November 1987. Half of these deaths were due to aspiration or ingestion of balloons, small parts or small toys such as marbles or balls. Over 40% of these deaths involved children riding a toy into the path of a motor vehicle or into a swimming pool. Riding toys, excluding bicycles, are associated with more injuries and deaths than any other single type of toy.[1]

The Toy Manufacturers of America, Inc (TMA), in cooperation with CPSC, have developed voluntary safety standards for toys. These voluntary measures are used by manufacturers to reduce the possibility of defects that could present risks of injury to children. CPSC also develops, promulgates and enforces mandatory safety standards for electric toys, pacifiers, bicycles, infant rattles, toys with sharp objects and lead-based paint used on toys. CPSC tests toys for compliance and investigates consumer complaints. Investigations led to the recall of 116 toys and children's articles during fiscal year 1987.

Public health educators and others who deal with children and parents can help to avoid toy-related injuries by providing parents with the following guidelines for toy selection and use:

- Select toys to suit the age, maturity, physical size, skill level, abilities and interests of the individual child.
- Supervise children, keeping toys with small parts away from younger children, as well as from children who still put toys in their mouths.
- Examine toys regularly for jagged edges and loose, small parts. Discard or repair toys immediately if problems are found.
- Store toys not in use where they will not be stepped on or tripped over.[1]

Local health agencies and other public health organizations can implement toy safety education programs and other programs that provide surveillance of toy injury risks. One such program, which began in 1971 in Monroe County, involves the Monroe County Health Department, consumer activists, news media and retail businesses. In this program, students from the State University of New York at Brockport canvass retail stores, conduct surveys and evaluate available toys. Recommendations are made to store managers to voluntarily remove toys that are considered to be unsafe and to identify problem toys before ordering for the following year. Consumer groups also request that retailers participate in this injury prevention program by publicizing toy safety information in advertisements. Media coverage of project activities also helps to alert the public to the need for toy safety awareness.

The two largest trauma centers in Monroe County, Rochester General and Strong Memorial, participated in CPSC's National Electronic Injury Surveillance System, established in 1974. At that time, approximately 60% of all accidental injuries in the county requiring emergency treatment and/or hospitalization were treated at the two facilities. Data from this surveillance project indicated a decline in toy-related injuries from 256 in 1974 to 77 in 1981 in the two hospitals. In addition, the Monroe County Health Department estimated that the county toy safety program prevented over 1,000 toy-related injuries by 1984, saving at least $100,000 in direct health care costs.[2] The county continued the program through the 1989 holiday season.

For a copy of *Ride the Toy Safety Train* brochure, contact Publications, New York State Department of Health, at (518) 474-5370.

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Extent of underreporting of AIDS documented in South Carolina

In comparing AIDS cases identified through a review of hospital discharge records with those cases reported to the state AIDS case registry, authors Conway and colleagues found an underreporting or underrecognition rate in South Carolina considerably greater than expected (*JAMA* 1989; 262:2859–2863). The authors reported selective trends in nonreporting and possible reasons for the discrepancy between the numbers of actual cases of AIDS and reported cases. With respect to AIDS, "given the current high social and economic costs of the disease, it is important both for epidemiologic studies and for health resource planning to have as accurate and complete a case count as possible."

Taking advantage of the statewide uniform hospital billing system established in South Carolina in January 1986, the authors were readily able to review the 596,585 hospital discharge record files introduced into the system from all nonfederally funded hospitals between January 1, 1986, and June 30, 1987. A computer search of these files identified 1,513 records with "ICD-9-CM codes consistent with AIDS-related and AIDS-defining conditions included in the 1987 revised [Centers for Disease Control] CDC AIDS case definition." These records were then reviewed by three of the authors.

AIDS has been a reportable disease in South Carolina since 1982, with a passive reporting system in which physicians and hospitals voluntarily submit reports to the state Department of Health and Environmental Control and to the Centers for Disease Control. Of the 1,513 hospital visits identified through hospital discharge records, 153 patients met the CDC AIDS case definition and should have been reported. These cases were then compared with the state AIDS case registry through March 31, 1988 (with the extra time allotted for reporting delays and CDC verification). The authors found that 91 of 153 (59.5%) of patients had been reported.

Conway et al reported several interesting findings:

- White patients with AIDS were significantly more likely to be reported to the state registry than were blacks or all nonwhites.
- Male patients were more likely to be reported than females with AIDS, although this finding was not statistically significant.
- Patients with a specific risk factor for AIDS stated in their hospital records were significantly more likely to be reported.

The authors suggest that differences in AIDS reporting practices from state to state may depend on the prevalence of a disease and the related importance given to disease surveillance. In areas of high incidence of AIDS, especially urban areas, there has been good reporting of AIDS, possibly due to a more active reporting system with good surveillance supported by federal funds. Conway and colleagues cite a recent study in Oregon in which implementation of active surveillance was followed by a 37% improvement in case detection. In late 1987, South Carolina began receiving federal support for the development of more active AIDS case surveillance measures.

The authors present other possible reasons for underreporting, including a lack of understanding of the disease reporting process, confidentiality concerns, and a lack of knowledge by physicians of the clinical conditions associated with AIDS.

The 59.5% reporting rate found in this study may even be a conservative estimate, as some patients did not meet the definition of AIDS due to insufficient information in the hospital records. "If underreporting to the degree observed in this study is widespread in the United States, current estimates regarding the extent of the epidemic should be viewed with caution." In addition to this warning, Conway et al emphasized the danger of the selective underreporting of females and minority patients, as the risk of AIDS continues to increase in these populations.

Food and Drug Administration halves dosage for AZT

On January 16, 1990, the Food and Drug Administration (FDA) recommended halving the dosage of AZT (zidovudine) for treating patients with the acquired immunodeficiency syndrome (AIDS). The FDA recommended a reduction of the dosage from 1,200 mg to 600 mg, after concluding that the lower dose was as therapeutically effective as the higher one and produced far fewer untoward reactions. The FDA based its recommendations on two studies conducted by the National Institute of Allergies and Infectious Diseases. At the present time, some 40,000 persons in the United States are taking AZT. The annual cost for therapy per person at the high dosage has been from $5,600 to $6,400. This cost will now be halved with the lower dosage for many patients. *NY Times*, January 17, 1990.

Growing role of bacterial infections in AIDS-related morbidity and mortality

The Centers for Disease Control's (CDC) 1987 revision of the surveillance case definition for AIDS included the addition of *Salmonella* septicemia as an opportunistic infection indicative of a diagnosis of AIDS. Prior to the 1987 revision, nonmycobacterial bacterial infections were not considered as being indicative of human immunodeficiency virus (HIV) infection or AIDS. Nichols and colleagues contend that "bacterial infections in general, and *S aureus* infections in particular, are important causes of morbidity and mortality in patients with AIDS (*Am J Clin Pathol* 1989; 92:787–790).

The authors reviewed the findings of autopsies performed on 46 patients with AIDS between January 1983 and January 1988. Postmortem cultures yielding bacteria were only acknowledged as infections if the autopsy revealed histologic findings of infection and gram-positive or gram-negative organisms on Brown and Brenn staining.

NEWS BRIEFS are compiled and written by Vicki Glaser.

Thirty-eight of the 46 cases (83%) had evidence of non-mycobacterial bacterial infections based on antemortem or postmortem cultures. In 37% (17/46) of these patients, bacterial infections were a significant factor in the cause of death. The bacterial infections uncovered on autopsy were not clinically diagnosed prior to death in 12 of 46 (26%) patients.

The most common cause of bacterial infection in this autopsy series was *Staphylococcus aureus*. In fact, "*S. aureus* infection was the fourth most common specific infection identified at autopsy among our patients with AIDS, behind cytomegalovirus infection, *Pneumocystis carinii* pneumonia (PCP), and invasive candidiasis." Other bacterial pathogens identified at autospy included *Escherichia coli*, coagulase-negative *Staphylococci*, *Pseudomonas aeruginosa*, *Yersinia pseudotuberculosis*, *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Capnocytophaga*.

S aureus is gaining recognition as a cause of pneumonia in patients with AIDS, often occurring concurrently with or after *P carinii* or cytomegalovirus pneumonia. The authors suggest that *S aureus* pneumonia be part of the differential diagnosis of pulmonary disorders in patients with AIDS.

Controversy grows over parallel track for experimental AIDS drug

In response to increasing pressure from advocates for people with AIDS for greater availability of experimental AIDS drugs, the National Institute of Allergy and Infectious Diseases recently instituted a parallel track testing program. The first application of the parallel track approach has involved the new drug dideoxyinosine (DDI), which was to be tested in a conventional clinical trial format, as well as being available to patients with AIDS who could not take part in the trials.

Criticisms of the parallel track program all too quickly became reality, as nearly 20 times more people initially sought the free distribution of DDI than signed up to participate in clinical trials (*NY Times*, November 21, 1989, pp A1, C15). This led researchers and federal officials to question whether they would attract enough volunteers to complete the trials.

The broader issue here is whether drug manufacturers and physicians will be able to determine if the benefits of experimental drugs such as DDI outweigh the adverse effects of these toxic drugs. Only through supervised clinical trials that involve large numbers of people and compare the effects of the new drug to those of existing treatments or to no treatment at all can this question be adequately addressed, according to critics of the parallel track program.

In support of this position, Dr Samuel Broder, director of the National Cancer Institute, has stated that "AZT would have been completely dismissed as a drug that's too toxic," had the statistical analysis of data collected in clinical trials not shown that the beneficial effects of AZT outweighed the risks of its deleterious effects.

Advocates of the parallel track program defend the greater availability of potentially beneficial drugs and criticize the drug testing process. With respect to the problems in recruiting participants for clinical trials with DDI, they contend that, "because of bureaucratic problems the DDI trials started supplying the drug several weeks after it was available through expanded access programs." Patients do not want to wait several weeks to try a new drug that might benefit them. Some patients do not want to participate in clinical trials because they have only a 50% chance of actually receiving the new drug, versus a placebo or other treatment.

Patients with AIDS and researchers alike hope that the potential short term benefits derived from expanded access to experimental drugs do not jeopardize the valuable information gained in the long term from controlled clinical studies.

Perinatally acquired HIV infection: AIDS' youngest victims

Most young children with AIDS acquired the human immunodeficiency virus (HIV) perinatally from their infected mothers. Compared to HIV infection in adults, HIV in children has a relatively short incubation. In a study of 172 children with perinatally acquired HIV who were born between 1979 and 1987, more than half the children exhibited symptoms of HIV infection within the first year of life, and clinical disease was evident in 78% of the study group by two years of age (*N Engl J Med* 1989; 321:1791-1796).

The median age at diagnosis was eight months. Mortality from HIV-related disorders was highest during the first 12 months of life; 17% of the 172 children died before one year of age. After the first year, the mortality rate declined and continued at a fairly constant level, with no plateau evident in the survival curve. Boys and girls had similar survival times. "Survival decreased markedly immediately after diagnosis, indicating the substantial effect of the first clinical illness on the prognosis. The median survival from the time of diagnosis was 38 months (95 percent confidence interval, 24 to 60 months)."

The authors noted a significant effect of age at diagnosis on survival. "Children who were under the age of one at diagnosis had a significantly shorter survival after diagnosis (24.8 months) than children who were one year old or older ($P < 0.001$)."

Scott et al analyzed the frequency, age at diagnosis, and survival patterns of seven common clinical manifestations of HIV-associated disease: *Pneumocystis carinii* pneumonia, candida esophagitis, lymphoid interstitial pneumonia, recurrent bacterial infection, cardiomyopathy, encephalopathy, and renal disease. The most common disorder (affecting 30% of the children) and the most likely disorder to appear first was lymphoid interstitial pneumonia. Encephalopathy and *Pneumocystis carinii* pneumonia were also frequently the initial manifestations of HIV infection. Renal disease was rare, and there were no cases of cardiomyopathy.

"Age at diagnosis and pattern of disease were independent and important prognostic factors for survival," concluded the authors. These factors can play an important role in understanding the course of the disease and in planning treatment protocols.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1988; 88:456–462). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

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(continued from p 10A)

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CANADA

Apr 5–7. **Office Gynecology.** 16 Cat I Credits. Montreal. Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024. Tel: (202) 638-5577.

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ADVERSE REACTIONS:

The most frequently observed adverse reactions include light-headedness, dizziness, sedation, nausea and vomiting These effects seem to be more prominent in ambulatory than in nonambulatory patients and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include:

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Gastrointestinal System: The antiemetic phenothiazines are useful in suppressing the nausea and vomiting which may occur (see above); however, some phenothiazine derivatives seem to be antianalgesic and to increase the amount of narcotic required to produce pain relief, while other phenothiazines reduce the amount of narcotic required to produce a given level of analgesia. Prolonged administration of VICODIN/VICODIN ES Tablets may produce constipation.

Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported

Respiratory Depression: Hydrocodone bitartrate may produce dose-related respiratory depression by acting directly on the brain stem respiratory center. Hydrocodone also affects the center that controls respiratory rhythm, and may produce irregular and periodic breathing If significant respiratory depression occurs, it may be antagonized by the use of naloxone hydrochloride. Apply other supportive measures when indicated.

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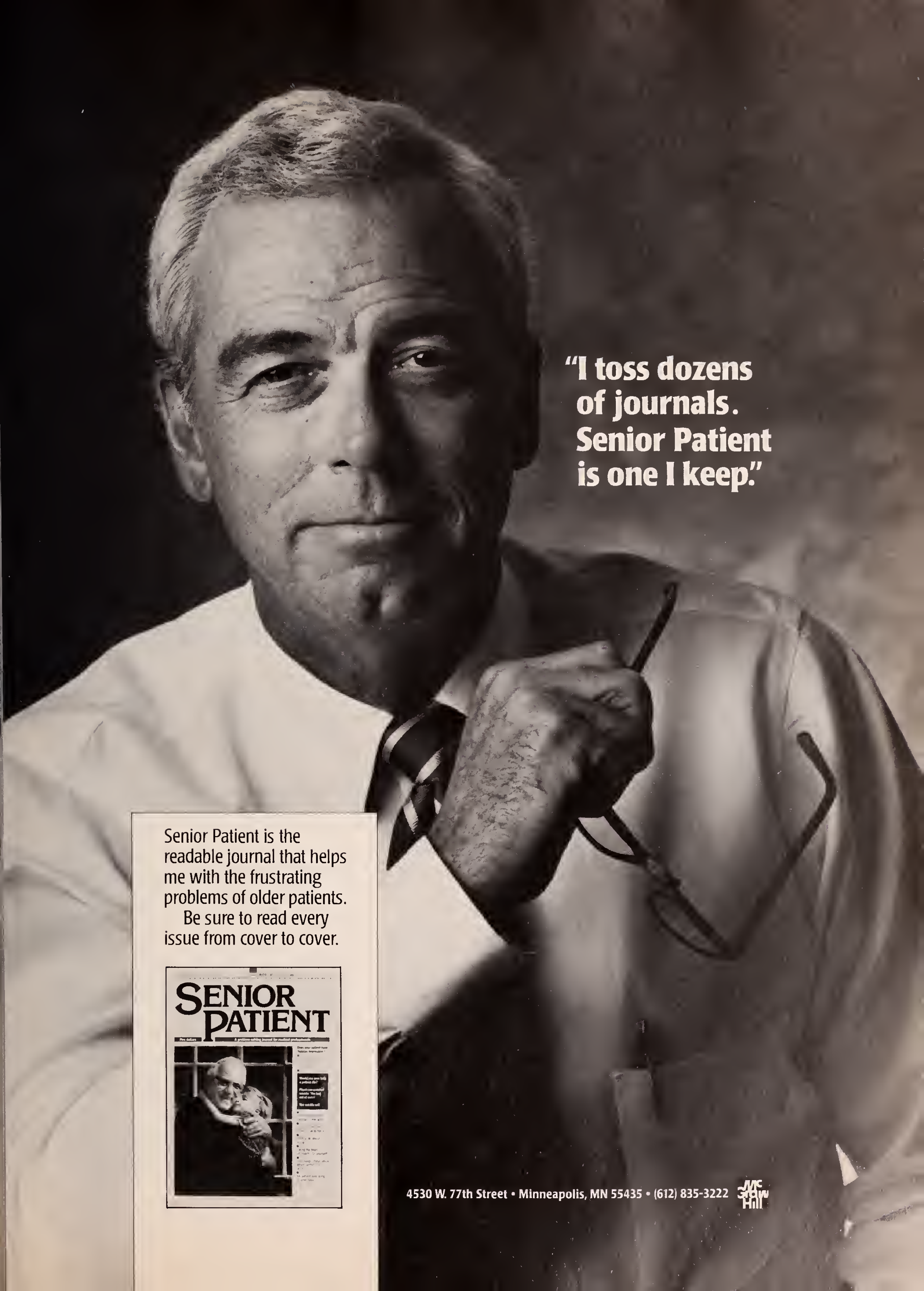
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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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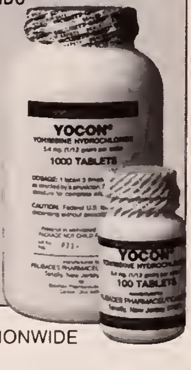

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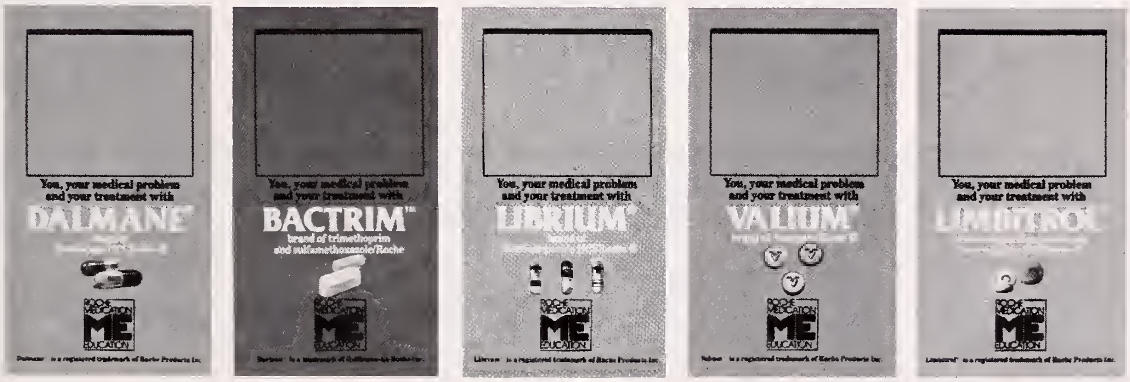
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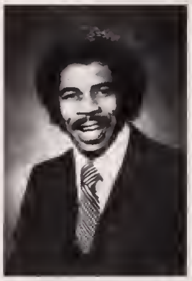
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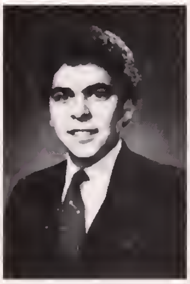
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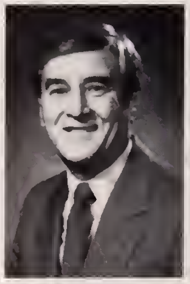
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Contents

COMMENTARIES

Ovarian small cell carcinoma 171
THOMAS M. ULBRIGHT, MD

Small cell carcinoma of the ovary: A therapeutic challenge 172
ROBERT E. SCULLY, MD, ROBERT H. YOUNG, MD

RESEARCH PAPERS

The public health benefit of increasing tobacco taxes in New York State 174
K. MICHAEL CUMMINGS, PHD, MPH; RUSSELL SCIANDRA, MA

Abdominal aortic aneurysmectomy without preoperative angiography: A prospective study 176
STEVEN G. FRIEDMAN, MD; BRUCE A. KERNER, MD; K. V. KRISHNASASTRY, MD; WILLIAM DOSCHER, MD; STEPHEN L. DECKOFF, MD; MATTHEW S. FRIEDMAN

REVIEW ARTICLE

Children of substance abusers in New York State 179
SHERRY DEREN, PHD; BLANCHE FRANK, PHD; JAMES SCHMEIDLER, PHD

ESSAYS IN PEDIATRICS

Changing concepts of the pathogenesis of diabetic nephropathy 185
FELICIA WILION, MD, MICHAEL D. BAILIE, MD, PHD

THE CPC

Churg-Strauss syndrome 190

SPECIAL ARTICLE

Passover matzoh and chocolate Easter eggs 196
PASCAL JAMES IMPERATO, MD

HISTORY

Henry Keller, MD, of New York and the Alyn Hospital in Jerusalem 201
MYER MAKIN, MD, SIMON T. WINTER, MD

CASE REPORTS

Para-endocrine hypercalcemia and ovarian small cell carcinoma 206
JOHN H. MALFETANO, MD; EDWARD DEGNAN, MD; RUDOLFO FLORENTIN, MD

Endometrial carcinoma in five patients with breast cancer on tamoxifen therapy 207
ANNA MATHEW, MD; ADA B. CHABON, MD; BERNARD KABAKOW, MD; MITCHELL DRUCKER, MD; RICHARD J. HIRSCHMAN, MD

Superior vena cava syndrome associated with a Hickman catheter 208
HARRY DAVIDSON KERR, MD

Isolated adrenocorticotrophic hormone deficiency presenting as chronic hyponatremia 210
PATRICIA B. CARROLL, MD; LEE MCHENRY, MD; JOSEPH G. VERBALIS, MD

LETTERS TO THE EDITOR

Paraesophageal hernia 214
ARTHUR SICULAR, MD

Animal experimentation for medical research 214
STEPHEN KAUFMAN, MD

Lead poisoning during home renovation 215
MELISSA M. SEDLIS, MD

An alma mater by any other name is an alma mater 215
N. A. HARVEY, MD

Tumor metastasis to an infusaport site 216
RONALD ZELNICK, MD; MARC CITRON, MD; ERIC MUNOZ, MD

Visual hallucinations as grief reaction without the Charles Bonnet syndrome 216
JEROME M. SCHNECK, MD

LEADS FROM EPIDEMIOLOGY NOTES 218
BOOK REVIEWS 221
NEWS BRIEFS 225
OBITUARIES 227
MEDICAL MEETINGS AND LECTURES 6A

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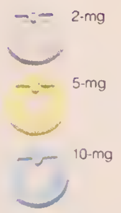

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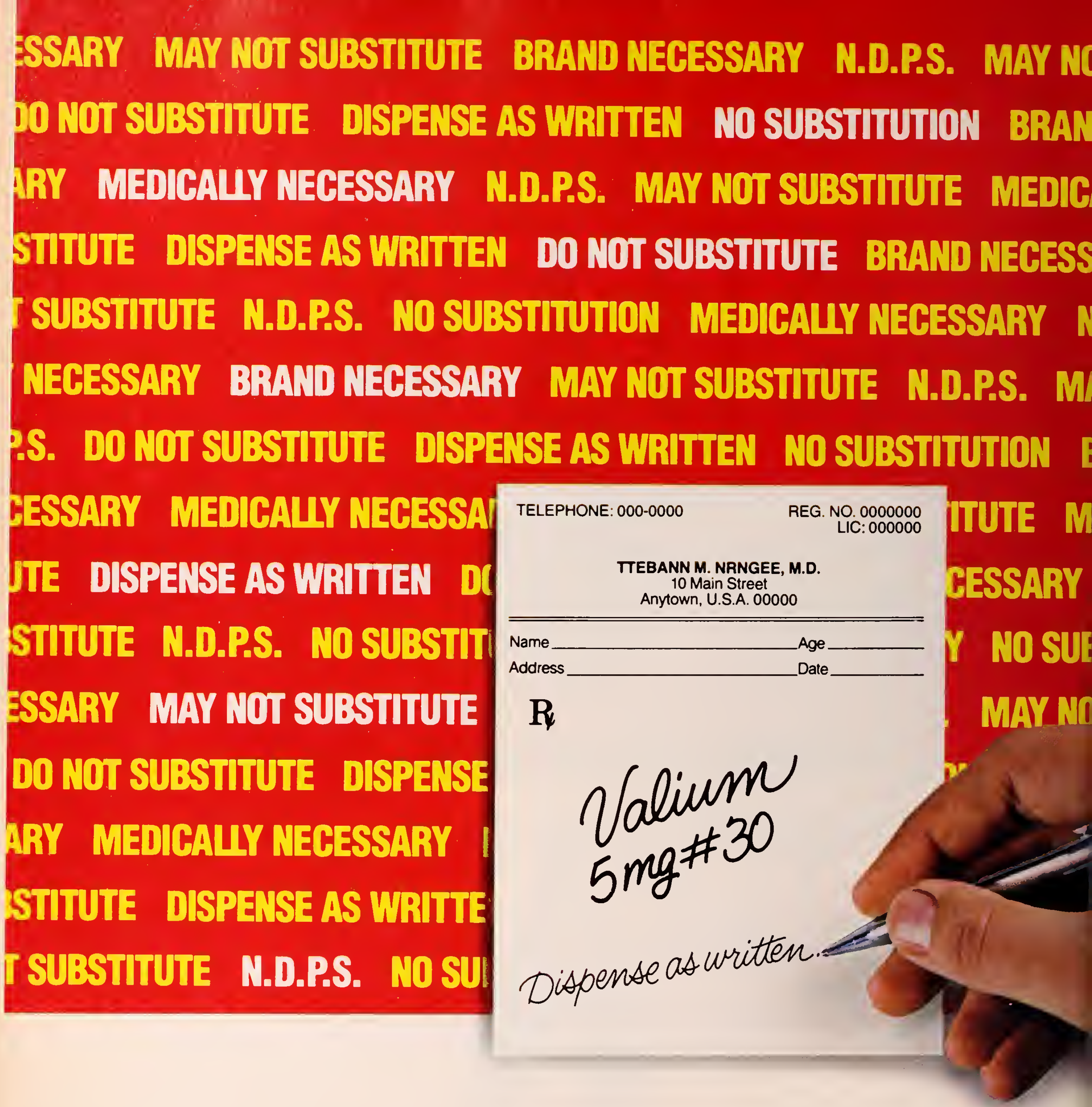
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2. Wewers MD, Casolaro MA, Sellers SE, et al: Replacement therapy for alpha$_1$-antitrypsin deficiency associated with emphysema. *N Engl J Med* 1987;316:1055-1062.

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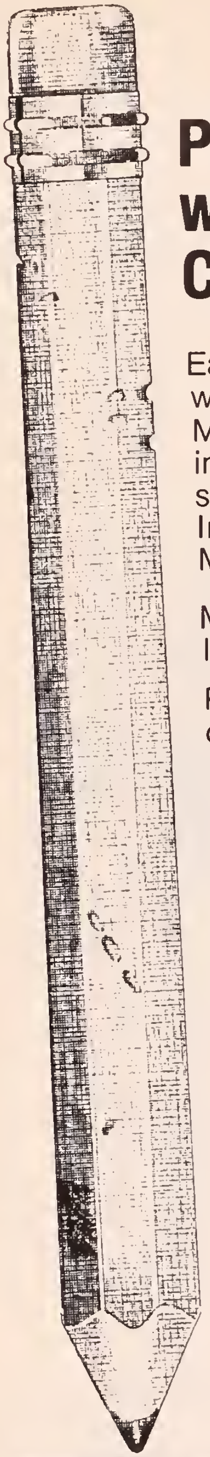

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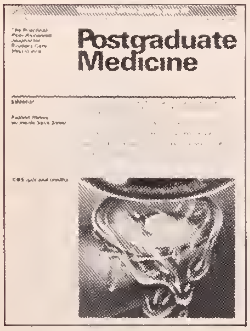

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(*continued from p 6A*)

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COMMENTARIES

Ovarian small cell carcinoma

In this issue of the *New York State Journal of Medicine*, Malfetano et al[1] describe the clinical and pathologic features of a nine-year-old girl with so-called "small cell" carcinoma of the ovary. Their case report describes the youngest patient thus far reported with this enigmatic neoplasm, which was first authoritatively reported in 1982 in a series of 11 patients by Dickersin and coworkers.[2] The basis for that original series was the consultative pathology practice of Dr Robert E. Scully of Harvard Medical School and the Massachusetts General Hospital, which serves to illustrate that this neoplasm is a relatively unusual form of ovarian cancer, but one which has enhanced clinical importance because of the young age of the affected patients. The average age of the patients reported by Dickersin et al was 22 years, with a range of 13 to 35 years.[2] In a recent update of 75 cases, presented in abstract form, the average age was 23 years, with a range of 10 to 42 years.[3] Perhaps the most striking clinical feature of small cell carcinoma of the ovary is its strong, but not invariable, association with paraneoplastic hypercalcemia. In the experience of Young et al[3] with this neoplasm, approximately two-thirds of the cases have associated hypercalcemia; reported serum calcium levels have ranged as high as 18 mg/dL.[2] The patient reported by Malfetano et al[1] had one serum calcium determination of 19.6 mg/dL in her terminal phase. In several cases, including the one reported in this issue, hypercalcemia remitted following bulk excision of the tumor and recurred with neoplastic recurrence. Despite this strong link with hypercalcemia, the pathogenesis of the altered calcium metabolism remains unknown. What does appear clear is that the tumor does not produce antigenically-unaltered parathormone. Reported parathormone levels have not been elevated in patients,[2] including the patient reported by Malfetano et al, and attempts to demonstrate parathormone through immunohistochemistry[4] or extraction of tumor tissues[5] have also been unsuccessful. It is simultaneously clear, however, that the hypercalcemia associated with this neoplasm is an example of "humoral hypercalcemia," since direct bone involvement is absent in most patients. It has been suggested that small cell carcinoma of the ovary may produce other bone resorbing substances such as prostaglandins of the E series,[6,7] or a parathyroid hormone-like peptide which is not generally recognized by antibodies directed against parathormone.[8] Future cases should therefore be investigated for such possibilities.

Despite the characteristic clinical picture of small cell carcinoma of the ovary—ie, a young woman or girl with an ovarian mass and hypercalcemia—the diagnosis nonetheless remains a pathologic one which has furthermore permitted the recognition of nonhypercalcemic cases. Generally these ovarian tumors are solid, with small cystic foci and large necrotic areas. Sheet-like arrangements of cells are usually interrupted by cystic, follicle-like structures filled with lightly staining eosinophilic fluid.[2,9] A cord-like arrangement of cells is also a prominent pattern. Mitotic figures are numerous. Many of the neoplastic cells, as the nomenclature of this tumor implies, are small with densely hyperchromatic nuclei and scant cytoplasm. In many cases, however, a larger cell component, with vesicular nuclei, prominent nucleoli, and abundant cytoplasm, is intermingled with the small cell component, and in some cases this component may be predominant.[3,9] The diagnosis of such "large cell variants" of small cell carcinoma may be problematic, but the clinical setting and the usual presence of a characteristic small cell component generally permit their recognition. It was originally noted by Dickersin et al[2] that a characteristic ultrastructural feature of this neoplasm was the presence of dilated cisternae of rough endoplasmic reticulum (RER) filled with moderately electron dense, granular material. This finding has been confirmed in other studies.[4,9] McMahon and Hart[4] have recently reported the ultrastructural findings in six patients with ovarian small cell carcinoma, and they concluded that this dilated RER containing granular material was a sufficiently constant feature to be of differential diagnostic utility.[4] In a controversial case, therefore, ultrastructural analysis would appear to be of diagnostic value. A major pathologic differential diagnosis is the distinction of small cell carcinoma of the ovary from juvenile granulosa cell tumor (JGCT). These neoplasms share many clinical and pathological features, although the cytologic atypism of small cell carcinoma is less common in JGCT. Furthermore, estrogenic symptoms may occur in JGCT and are not a feature of small cell carcinoma.[10] McMahon and Hart[4] propose that ultrastructural analysis may be particularly useful in establishing this differential diagnosis, which is especially important given the usually benign behavior of JGCT and the aggressive, malignant nature of small cell carcinoma of the ovary.

The histogenesis of small cell carcinoma of the ovary remains controversial. The possibilities include a surface epithelial origin, a sex-cord origin, or a germ cell origin. Immunohistochemistry has not proved useful in sorting out these possibilities.[6] Small cell carcinoma of the ovary shows frequent positivity for cytokeratins and vimentin and occasional positivity for epithelial membrane antigen.[6,9] A recent study has also reported frequent positivity for the neuroendocrine markers chromogranin A and neuron-specific enolase (NSE),[6] but the significance of these findings must be tempered by the lack of neurosecretory granules on ultrastructural examination in most series,[2-4,9] with one exception.[11] Furthermore, NSE appears to be quite nonspecific, and most investigators do not believe that small cell carcinoma is a neuroendocrine tumor.[6] It is therefore inappropriate to equate the term "small cell carcinoma of the ovary" with small cell carcinoma of the lung; those who practice medical oncology should be aware of the different connotations of this term when it is applied to these different organs. My own point of view is that, based on the clinical circumstances, the histology, the ultrastructure, and the (admittedly nonspecific) immunohistochemistry, there is more to support a theory of germ cell origin than the other possibilities, but this issue will undoubtedly remain controversial until a more specific marker is identified in these neoplasms, probably through the application of molecular biologic techniques.

The prognosis of small cell carcinoma of the ovary remains dismal, despite a variety of therapeutic approaches including surgery, radiation, and complex, multiagent chemotherapy regimens. Many patients present with their initial disease localized to the involved ovary, but recurrences characteristically develop within a matter of months, and death often occurs within one or two years of diagnosis. Young et al[3] reported a survival rate of 12% in a series of 49 patients with follow-up. With the report of Malfetano et al[1] in this issue of the *Journal*, it becomes increasingly clear that physicians who routinely see children in their clinical practice need to be aware of this diagnostic entity so that surgical treatment and adjuvant therapy can be initiated at the earliest possible point in its natural history. Because of the rarity of this entity, it is also obvious that the clinical experience of many centers will have to be pooled in order to define and refine the best possible therapeutic approach to this very malignant tumor affecting girls and young women.

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Small cell carcinoma of the ovary: A therapeutic challenge

Small cell carcinoma of the ovary, an example of which is reported by Malfetano et al[1] in this issue of the *Journal*, is a relative newcomer to the ranks of ovarian cancer, having been described only within the last decade.[2] This highly malignant neoplasm, which occurs in young women, has many puzzling features.[3,4] It has been encountered exclusively in premenopausal females, but almost never in the first decade, with the patient whose case is reported in this issue being a rare exception. It is the only malignant tumor in the human that is associated in the majority of cases with para-endocrine hypercalcemia, which is of unknown pathogenesis. It has been seen only in the ovary, is of undetermined cellular origin, and unlike most ovarian cancers, has responded poorly to the usually effective therapeutic regimens.

Approximately two thirds of the patients with ovarian small cell carcinoma who have had calcium determinations in the presence of tumor have had elevated levels, although only a few of them have had symptoms of hypercalcemia, as reported in the current case. The small cell carcinoma is with rare exceptions the only type of ovarian cancer that is associated with hypercalcemia in the premenopausal female, with the clear cell carcinoma being the most common form in older women. Therefore, in a young female with an adnexal mass suspected of being an ovarian tumor, a preoperative calcium determination may provide very strong evidence that the tumor is a small cell carcinoma in the absence of another explanation for hypercalcemia. No detailed studies have been performed to elucidate the mechanism of the hypercalcemia in patients with ovarian small cell carcinoma, although in a few cases attempts to identify parathormone or a parathormone-like substance in the serum or in the neoplastic tissue have been unsuccessful.

Microscopic examination of the small cell carcinoma has revealed in most cases small cells with malignant-appearing nuclei and scanty cytoplasm growing in a variety of epithelial patterns that characteristically include distinctive follicle like spaces. In about one third of the cases, portions of the tumor contain large cells with abundant cytoplasm, and in an occasional case the large cells predominate. A curious finding in 10% of the tumors has been focal differentiation of the cells into those filled with mucin.

The origin of the tumor remains a mystery. The great majority of ovarian cancers are of surface epithelial, germ cell, or sex cord origin. Transitions between the small cell carcinoma and tumors of the above lineages have not been encountered. Since the common ovarian carcinomas of surface epithelial origin reach their peak age incidence after the menopause, it appears unlikely that the small cell carcinoma belongs in that category. Primitive germ cell cancers and malignant tumors of sex cord origin occur in the same age group as the small cell carcinoma, although the former neoplasms, unlike the latter, are also encountered during the first decade. An argument against the germ cell origin of the tumor, as suggested by some investigators,[5] is the absence of other forms of germ cell neoplasia in association with the small cell carcinoma and its apparent lack of occurrence in the testis. The occasional differentiation of the tumor cells into mucin-containing cells is not a clue to its histogenesis, since such cells can be seen in surface epithelial, germ cell, and sex cord (Sertoli-Leydig cell) tumors. Although the term "small cell carcinoma" was chosen to designate this neoplasm, it differs markedly in nature from the small cell neuroendocrine carcinoma of the lung and similar tumors of other organs, not only in its high frequency of associated hypercalcemia but also in its light-microscopic and electron-microscopic appearance.[6,7] Ultrastructural studies have revealed nonspecific features,[2,8] the most prominent of which is an abundance of dilated, rough endoplasmic reticulum, and only rarely have a few granules interpretable as neuroendocrine dense-core granules been described.[9,10] Immunohistochemical studies have likewise failed to show a striking similarity to the small cell carcinoma of the lung.[7]

Optimal therapy of the ovarian small cell carcinoma remains a problem. In more than one third of the cases, the tumor has extended beyond the ovary by the time of exploration.[3,4] Although the overall survival rate is only approximately 10%, between 20% and 25% of the patients with Stage 1A tumors have survived after operative therapy alone. (Some of these patients have not been followed for long periods.) Although the tumor is synchronously bilateral in only about 1% of the cases, its rare recurrence in the contralateral ovary after unilateral oophorectomy raises the question whether bilateral removal of the adnexa is indicated even for Stage 1A tumors. Patients with disease apparently confined to the ovary can be followed in the usual manner for patients with ovarian cancer, with the addition of determinations of the serum calcium level, which typically rises with recurrence of the tumor. Effective therapy of patients with higher stage tumors or recurrent disease has not been achieved as yet. A variety of chemotherapeutic agents in combination that have been successful in the treatment of malignant germ cell tumors and surface epithelial carcinomas have been tried.[11,12] They have resulted in temporary complete remissions of disease in some cases.[12] Radiation therapy has occasionally proved effective, but prolonged remissions have been very rare. With greater recognition of this new and devastating form of ovarian cancer it is probable that its cytogenesis and paraendocrine function will be elucidated, and most important, improvements in its management will ensue.

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The public health benefit of increasing tobacco taxes in New York State

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ABSTRACT. The 1989–1990 New York State budget increased the tax on a package of cigarettes from 21 to 33 cents. In this paper we estimate the impact of this tax increase on smoking prevalence and smoking-induced deaths in New York State. Findings show that 115,967 New Yorkers will be encouraged to quit or not start smoking as a result of the increased cigarette tax. The reduced prevalence of smoking attributed to the tax will result in the avoidance of approximately 28,992 premature smoking-induced deaths over the next generation.

(NY State J Med 1990; 90:174–175)

The 1989–1990 New York State budget increased the cigarette excise tax from 21 cents to 33 cents per pack. This 12-cent per pack increase in the cost of cigarettes is expected to have a significant impact on the smoking prevalence of New Yorkers. In this paper, we estimate the number of smoking-induced deaths averted due to the tax-related reduction in smoking prevalence.

METHODS

Price elasticity estimates for cigarettes developed by Lewit et al[1,2] were used to estimate the impact of a 12-cent increase in the price of cigarettes on smoking prevalence. Elasticity, a measure of the degree of responsiveness of demand to changes in price, is defined as the percentage change in quantity of cigarettes purchased divided by the percentage change in price. An elasticity of −0.5, for example, means that a 10% increase in price would reduce by 5% the quantity of cigarettes consumed. A change in the price of cigarettes (due to taxes or otherwise) will affect both the prevalence of smoking and the amount smoked. That is, some smokers will quit smoking, others will continue smoking but smoke fewer cigarettes, and some prospective smokers will not start smoking. Because it is difficult to judge the health benefits associated with reduced smoking, and because previous studies have demonstrated that cigarette prices affect cigarette consumption primarily by reducing smoking prevalence, this analysis is restricted to the impact of increased cigarette taxes on smoking prevalence.[3]

While several studies have attempted to estimate the price elasticity for cigarettes,[3] we have chosen to use the methods developed by Lewit et al,[1,2] because estimates are given separately for several age groups. Responsiveness to price changes will vary by age. This is because, compared to adults, fewer teenagers smoke, and among those who do, the habit is less well established. Furthermore, teens have less disposable income than do adults and are more sensitive to price increases. Thus, responsiveness to price changes is likely to be greater among teenagers than among adults. Because it is rare that a person over age 21 will take up the smoking habit, discouraging smoking in teens will have significant impact on future adult smoking rates.

The average cost of a package of cigarettes in New York State is estimated to be $1.25.[4] Assuming no other changes in the cost of cigarettes except an increase by 12 cents in the state excise tax, the average price of a package of cigarettes would increase by 9.2%. The expected percentage change in the prevalence of cigarette smoking by age was computed by multiplying the price elasticity estimates from Lewit et al[1,2] as shown in Table I, by 9.2%. Elasticity estimates were not available for persons 18–19 years of age and for persons 75 years or older. The price elasticity for those 75 years of age and older was assumed to be the same as for those 36 to 74 years of age. For 18–19 year olds, the percentage change midway between that for 12–17-year-olds and that for 20–25-year-olds was used.

Table II presents estimates of the number of smokers in New York State by age. The overall population figures used for computing the number of smokers by age were based on the 1990 population projections for New York State.[5] Cigarette smoking prevalence figures for 12–17 year olds were estimated from a 1983 household telephone survey of teenagers conducted by the New York State Health Department.[6] Cigarette smoking prevalence figures for adults were based on estimates from the 1987 Behavioral Risk Factor Surveillance System Survey conducted in New York State (personal communication, H. Bzduch, New York State Health Department, February 1989).[7,8]

RESULTS

The third column in Table II provides an estimate of the change in the number of smokers for each age group attributable to a 12-cent increase in the state cigarette excise tax. Overall, approximately 115,967 New Yorkers would be encouraged to quit or not start smoking as a result of the increased cigarette tax.

To assess fully the impact of the proposed tax change on health, one would need to know how many persons would reduce their use of cigarettes, how many would quit smoking, and how many would not start smoking. This information is not available. However, it is possible to estimate crudely the likely health effects that would be expect-

TABLE I. Price Elasticities for Cigarette Smoking Prevalence by Age

Age Group (yr)	Elasticity Estimate for Smoking Prevalence
12–17	−1.20
20–25	−0.74
26–35	−0.44
36–74	−0.15

Source: Lewit et al.[1,2]

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TABLE II. Estimated Impact of a 12-Cent Increase in the Cigarette Excise Tax on the Number of Smokers in New York State by Age

Age Group (yr)	Number of Smokers	Decline in the Number of Smokers Due to the Tax Increase
12–17	205,007	22,633
18–19	136,555	12,181
20–24	249,470	16,964
25–34	936,416	37,831
35–74	1,833,787	25,306
75 and older	76,265	1,052
Total	3,437,500	115,967

ed to result from the reduced numbers of smokers due to the tax increase. Using the conservative estimate that one lifelong smoker out of four dies of smoking-related illness,[9] a 12-cent increase in the cigarette excise tax, maintained in real value over time, would avert approximately 28,992 premature smoking-induced deaths over the next generation.

DISCUSSION

No doubt, the decision to increase the cigarette excise tax in New York State was the result of a need to raise additional revenues. However, a positive side effect of this decision is a decline in smoking prevalence and a corresponding reduction in smoking-induced disease. While the 12-cent per pack increase in the cigarette excise tax will only reduce the overall prevalence of smoking by about 3%, due to the large number of smokers in New York State (over three million), the absolute effect of the tax increase is substantial. It is estimated that approximately 115,967 New Yorkers will be encouraged to quit smoking or not to start smoking as a result of the proposed tax increase. This number in turn translates into a reduction in the diseases caused by cigarette smoking. There are probably few things in this year's state budget that will yield as great a public health benefit as the increase in the cigarette excise tax.

There are, however, some issues that should be considered in the current tax plan. First, the current system for taxing cigarettes, which involves a constant amount on each pack of cigarettes, loses its value over time due to inflation. The average price for a package of cigarettes in New York State in 1987 was actually less than it was 20 years ago when adjusted for inflation. The unit tax on cigarettes should be replaced with an equivalent-yield ad valorem tax which would allow revenues to keep pace with inflation-induced increases in cigarette prices. In this system, the tax is a fixed percentage of the wholesale price. An alternative approach to the ad valorem tax would be to tie the tax change to a price index for cigarettes.[10]

The earmarking of cigarette tax revenues for special purposes should also be considered. Several states have used cigarette tax revenues to finance tobacco-related health programs. In Minnesota, the Omnibus Nonsmoking and Disease Prevention Act of 1985 increased the cigarette excise tax five cents per pack and earmarked one cent of the added revenue to a public health fund.[11] One quarter of the public health fund was dedicated to helping local schools implement tobacco education programs. In California, three quarters of a recent 25-cent per pack increase in the cigarette excise tax was dedicated to support health education, research, medical treatment, and environmental conservation programs.[12] In the first year, it is estimated that this added tax will generate $650 million to support these programs.

In New York State, the 12-cent increase in the cigarette excise tax will generate $194 million of new revenue in the first year. Under the current budget, the revenue generated from cigarette taxes is allocated to the state's general fund.

In 1988–1989, the New York State Health Department spent less than $500,000 on tobacco education programs (unpublished data, Bureau of Adult and Gerontological Health, February 1989). Given the increasing financial pressures on the health department to address the AIDS problem, resources allocated to tobacco education are likely to continue to be minimal. Earmarking 5% of the revenues generated by the cigarette tax increase to support tobacco education efforts in New York State would be an appropriate and wise use of these added resources.

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Abdominal aortic aneurysmectomy without preoperative angiography

A prospective study

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ABSTRACT. **In an attempt to analyze whether routine angiography is necessary prior to elective abdominal aortic aneurysmectomy (AAA), a prospective study was designed in which this examination was obtained only for specific indications. These included significant hypertension, renal dysfunction, symptoms of visceral ischemia, suprarenal extension of the aneurysm or a coexisting thoracic aneurysm, and diminished or absent femoral pulses. A consecutive series of 124 abdominal aortic aneurysms is reported, in which 110 procedures were performed electively. Preoperative angiograms were obtained in only ten patients (9.1%) and in nine of these an alteration in the usual operative strategy resulted. In the remaining 100 patients undergoing elective AAA without preoperative aortography, acceptable morbidity and mortality rates were obtained despite the intraoperative discovery of iliac aneurysms in 25 patients (23%) and accessory renal arteries in three patients (2.7%). In the absence of specific indications for angiography, the mainstay of the preoperative evaluation for abdominal aortic aneurysms should be computed tomography (CT). The preoperative workup can be done entirely on an outpatient basis.**

(NY State J Med 1990; 90:176–178)

As early as 1961, the question was raised whether routine aortography is necessary prior to elective abdominal aortic aneurysm (AAA) resection.[1] Despite numerous advances in the surgical management of this lesion since that time, no consensus has been reached regarding the precise role of angiography in the preoperative period. In a recent review of publications regarding this question, Couch et al[2] noted that authors have been nearly evenly divided between recommending preoperative aortography for all patients or selectively. Most of these studies were retrospective and in the few prospective studies, the protocols entailed the routine use of angiography prior to aneurysm resection. By conducting these examinations in all patients, it is difficult to assess their true worth in a completely objective manner. For this reason we wished to study the contribution aortography makes to preoperative evaluation of patients with abdominal aortic aneurysms by limiting its use to patients with specific indications.

PATIENTS AND METHODS

Between July 1986 and September 1988, 124 consecutive patients with abdominal aortic aneurysms underwent surgery. In 14 cases surgery was performed emergently for expanding or ruptured aneurysms, and these were excluded from this study. In the remaining 110 elective cases, preoperative angiography was performed only for one or more of the following indications: significant hypertension or renal dysfunction, symptoms of visceral ischemia, suprarenal extension of the aneurysm or a coexisting thoracic aneurysm, and diminished or absent femoral pulses.[3]

There were 79 men and 31 women who underwent elective AAA. Their ages ranged from 54 to 86 years with a mean of 69 years. In addition to the usual careful history and physical examination, the routine preoperative evaluation of these individuals included a chest radiograph, electrocardiogram, and computed tomography (CT) of the abdomen. Arteriography, when indicated, was performed by a retrograde transfemoral technique.

RESULTS

Among 110 consecutive elective patients, ten (9.1%) presented with one of the aforementioned indications for preoperative aortography. Diminished or absent femoral pulses were noted in five patients. One hypertensive patient had a creatinine of 6.4 upon admission which prompted angiography. CT findings which led to further evaluation included a pararenal abdominal aortic aneurysm in one patient (Fig 1) and a thoracoabdominal aneurysm in two patients (Fig 2). Angiography was also performed in one patient who had a thoracic aneurysm noted on the preoperative chest film.

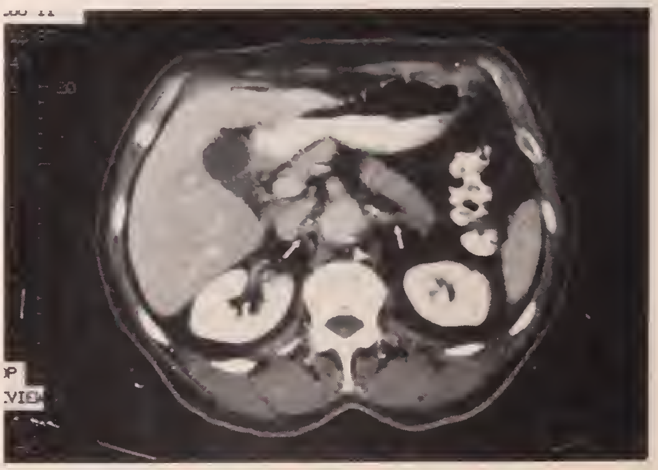

FIGURE 1. Abdominal CT demonstrating pararenal aneurysm (arrowhead) with renal arteries (arrows) emanating from the lesion.

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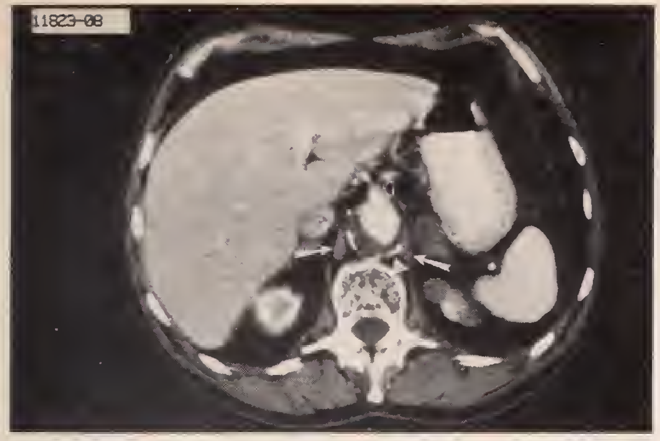

FIGURE 2. Thoracoabdominal aneurysm (arrowhead) originating at the level of the diaphragm (closed arrows). The celiac axis is also seen arising from the aneurysm (open arrow).

Significant findings on arteriography included severe aorta-iliac occlusive disease in four patients and a thoracoabdominal aneurysm extending just above the diaphragm in two patients. The pararenal aneurysm had two normal renal arteries emanating from its origin. One individual had a 6-cm thoracic aneurysm in addition to a 5-cm abdominal aortic aneurysm. The patient presenting with hypertension and an elevated creatinine had complete occlusion of one renal artery and a 95% stenosis of the contralateral vessel.

Associated procedures in all patients included a coronary artery bypass graft ten days to two weeks prior to AAA in three, and thoracic aneurysm resection two weeks prior to AAA in another patient. The individual presenting with renal failure underwent simultaneous renal artery bypass with a 6-mm thin-walled polytetrofluoroethylene graft originating from the aortic graft. One patient underwent simultaneous coronary artery revascularization for unstable angina and a tender aneurysm, and another patient had simultaneous inferior vena caval interruption for recurrent deep venous thrombosis.

Our preference is for straight or tube graft placement in the therapy of abdominal aortic aneurysms whenever possible. This was performed in 81 of the 110 elective cases (74%). Bifurcation grafts were used only in the presence of concomitant iliac artery aneurysms (25 patients) or significant aorta-iliac occlusive disease (four patients). Iliac artery aneurysms were also identified preoperatively in a high percentage of cases by CT. There were two postoperative deaths in this group, for a mortality rate of 1.8%. An 81-year-old man died of a myocardial infarction one week following his AAA and reoperation for bleeding. An 81-year-old woman suffered a fatal myocardial infarction two weeks after surgery.

Three major complications occurred. One patient suffered a myocardial infarction on postoperative day one, from which he recovered completely. A second patient suffered a cerebral vascular accident three weeks following aneurysm resection, resulting in right hemiplegia. One patient, mentioned above, required emergency reoperation for bleeding following a difficult AAA. This patient had received six units of banked blood and upon reexploration was noted to have a coagulopathy. He succumbed to a myocardial infarction one week later. Two superficial wound infections also occurred which responded to local care and oral antibiotics.

Additional noteworthy findings at surgery included six inflammatory aneurysms and one small renal artery aneurysm; most of these were seen on preoperative CT. CT is perhaps the most reliable method for detecting inflammatory aneurysms preoperatively. Three patients had unsuspected accessory renal arteries, which were incorporated within the proximal anastomosis in each case.

DISCUSSION

In addition to the widely accepted indications for angiography prior to AAA mentioned above, numerous other reasons for routine use of this test have been offered. Kahn et al,[4] and later Brewster and his colleagues,[5] noted the importance of defining the size and location of the aneurysm, and its relationship to the renal arteries. The use of conventional and more recently high resolution CT, however, has been shown to be as accurate as and safer than angiography.[6] Other studies also indicate that CT is far more accurate than ultrasonography, and in many instances provides information that angiography does not.[7,8] We too have found CT to be a highly reliable means of judging aneurysm size and its relationship to the main renal arteries. In this series of 110 cases, it was accurate in the vast majority of cases.

Kwann and his coworkers have advocated routine angiography to detect occlusive disease, multiple aneurysms and anomalous vessels.[9] We have found that significant occlusive disease is reliably detected by preoperative examination of the femoral pulses and careful history-taking. Noninvasive arterial Doppler examination provides an additional screening tool for determining the need for angiography. Multiple aneurysms occurred in 23% of the present series and are usually quite apparent at laparotomy. Anomalous vessels are rare, but also easily detected during dissection of the aneurysm. In this group of 110 electively treated patients, accessory renal arteries were found in 2.7%.

In a review of aortography prior to 100 cases of AAA, Baur and his colleagues claimed that it substantially influenced surgical decisions in 75% of cases.[10] The authors noted the importance of angiography in defining visceral arterial abnormalities, and in fact, performed renal artery reconstruction in four patients and visceral artery reconstruction in eight patients. Of the individuals undergoing renal revascularization, all had indications that would have prompted preoperative angiography. However, the authors failed to note the reasons for mesenteric revascularization in this series. If these procedures were therapeutic, then angiography would have been performed based on the appropriate symptoms. Careful physical examination and history taking, therefore, would have identified those cases in which preoperative aortography would have altered surgical strategy. In explaining the large discrepancy between the number of arteriographic findings and fewer resulting procedures, the authors also noted the importance of utilizing clinical judgment.

In a recent prospective protocol of routine preoperative angiography, Bunt and Cropper[11] noted that 18% of 70 angiograms provided decisive information. Four isolated iliac aneurysms, two thrombosing aneurysms, one accessory renal artery, and one arteriovenous fistula within an iliac aneurysm were detected. Each of these lesions, however, would have been seen at surgery. One case each of suprarenal extension of an abdominal aortic aneurysm, renal artery aneurysm, and renal cell carcinoma were diagnosed, yet CT scanning would likely have detected these, as noted by the authors. In the single case of significant renal artery stenosis, the indications for surgical repair were not noted. A case of femoral arteriovenous fistula, resulting from a prior angiogram, represented the single lesion which might have gone undetected in this group, thereby reducing the true angiographic yield to far less than the 18% reported.

An additional advantage of CT over angiography is expense. In our area the cost of an abdominal CT is well below 50% of the cost of an aortogram. Use of only the former examination results in significant savings and appears to be more cost effective. In addition, patient acceptance of CT over angiography is much higher and there is no risk of embolization from thrombus within the aneurysm resulting from CT.

In the present series of 110 elective aortic aneurysms, preoperative angiography was performed in only ten patients with significant findings in nine. In the remaining 100 patients, a variety of associated unsuspected lesions were encountered at surgery, such as iliac aneurysms and accessory renal arteries. These were all managed without serious disruption of the surgical procedure and with acceptable rates of morbidity and mortality.

Angiography prior to AAA, therefore, seems practical only for specific indications. These include significant hypertension or renal dysfunction, symptoms of visceral ischemia, suprarenal extension of the abdominal aneurysm or a concomitant thoracic aneurysm, and diminished or absent femoral pulses. Thoracic and thoracoabdominal aneurysms are reliably diagnosed using chest radiography and CT. The preoperative finding of hypertension or renal dysfunction alerts one to most cases of significant renal artery stenosis. In the subset of patients undergoing AAA (mean age 69 years in this series), the number of individuals who might benefit from a prophylactic renal revascularization is undoubtedly quite low. This is also true for individuals with visceral arterial occlusive disease as the indications for prophylactic revascularization are few. Iliac aneurysms and anomalous arteries are usually readily detected at surgery.

The cost, risk, and low yield of aortography in the absence of specific indications make its routine use prior to AAA unwarranted. In addition to the usual history and physical examination, an adequate preoperative workup of patients with abdominal aortic aneurysms consists of chest radiography, EKG, and abdominal CT. A complete and accurate evaluation of these individuals may be performed entirely on an outpatient basis.

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Children of substance abusers in New York State

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During the 1980s, increasing attention was given to children of substance abusers. This was due in part to the increasing number of female substance abusers; a study of substance use among females in New York State[1] found a dramatic increase of nonmedical substance use for females aged 18 years to 34 years from 1979 to 1984. Based on projections from survey findings, the percentage of females in this age group who had used substances nonmedically (defined as use of illegal substances or the nonmedical use of psychoactive prescription drugs in their lifetime) went from 49% in 1979 to 63% in 1984. Concerns focus on the impact of pregnant women's drug ingestion on the developing fetus, as well as the consequences for children living with parents who are substance abusers. Fears about the impact of the acquired immunodeficiency syndrome (AIDS) epidemic[2] and the effects of cocaine use on the fetus[3-5] are also gaining attention.

The literature on children of substance abusers indicates that these children and their parents are populations in need of help[6]; some of these children have developmental difficulties and require assessment and referral for appropriate counselling and treatment services. Furthermore, many parents who are substance abusers require assistance in becoming more effective parents. Information from the New York City Department of Health (personal communications, Bureau of Vital Statistics, 1984, 1986, 1988) underscores the importance of providing services to this population. For the period 1978–1984, the infant mortality rate for infants whose mothers were substance users (as indicated on birth certificates) was about three times higher than the citywide rate (46.7/1,000 vs 15.5/1,000 live births). The percentage of newborns of low birth weight (under 2,500 grams or less than 5½ lb) born to these women was more than three times the citywide rate (32.8/1,000 vs 9.6/1,000 live births). Recent information indicates that the infant mortality rate may be increasing in New York City, after more than a decade of decline, due to births to mothers who are abusing cocaine or who have AIDS.[2]

The urgency of this problem and the subgroups in need of services can be identified more effectively if the dimensions of the problem are determined. No recent estimates are available regarding the numbers of these children or the numbers of births to drug abusing women. This paper provides information on trends in births to substance abusing women useful in projecting anticipated increases or decreases in such births. Also included are estimates of the number of such births, to identify the magnitude of the problem for newborns, and estimates of the number of children under 18 years of age who constitute the children of substance abusers of New York State, to identify the total number of children in this population. Secondary analyses of several data sources were undertaken to arrive at these estimates. In addition, due to the changing nature of the substance abuse problem and recent increases in cocaine use, data are presented separately for narcotics, for cocaine, and for other substances of abuse, when available.

COLLECTING THE DATA

Several basic sources contributed to the development of trends and the calculation of estimates.

Trends. Data on trends of births to substance using women were obtained from the New York City Department of Health and the New York City Infant Health Assessment Program (IHAP).

The New York City Department of Health maintains a file of birth certificates and accompanying confidential medical reports in which drug use by the mother is cited. The report usually includes the specific drug used and items of information about the parents, the infant, and the delivery. Overall data on the number of such births were calculated for the 10-year period 1978–1987; specifics on the types of drugs used were available for the period 1978–1986.

The Infant Health Assessment Program is a state-funded program administered by the New York City Department of Health, Bureau of Child Health. The program has been in existence since January 1986, and focuses on identifying, registering, and tracking infants at risk of developmental disabilities, using various criteria including maternal substance abuse. (The IHAP criteria are birthweight of less than 2,001 grams, gestational age of less than 33 weeks, maternal age of less than 16 years, inherited metabolic disease, major congenital abnormality, stays of greater than ten days in neonate intensive care units, maternal alcohol abuse during pregnancy, and maternal substance abuse during pregnancy.) Data are collected from 12 New York City hospitals (11 municipal hospitals and one voluntary), estimated to account for about one third of all births in New York City. Although this is a

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statewide program, data on substance abuse are only available for New York City.

Estimates. Estimates of the number of children of substance abusers used in this report are based on the numbers of narcotic and non-narcotic substance abusers in treatment and not in treatment in New York State.

All drug treatment programs funded by New York State are required to submit monthly forms to the Management Information System of the New York State Division of Substance Abuse Services containing information on the demographics of new admissions, including age, race, and sex. Information on the primary and secondary drugs of abuse is also collected. For the purposes of this study, demographic data were obtained for the more than 46,000 patients in treatment in these programs as of August 1987. Data on a sample of patients in nonfunded programs (estimated at approximately 5,000) were also included.

During 1984, surveys were conducted of a stratified, random sample of patients in New York State-funded methadone maintenance[7] and residential drug free treatment programs.[8] The stratification was by program location in the state. Survey instruments, administered by counselors in the programs, included questions on demographics and the numbers and ages of children. These surveys were used to generate estimates of the number of children per patient. For methadone patients, each male patient was found, on the average, to have 1.7 children, and each female patient had 1.8 children; for residential drug free treatment programs each male patient was found, on the average, to have 0.9 children, and each female patient, 1.2 children. An additional correction was incorporated to estimate the number of children under 18 years of age.

A different approach was needed to estimate the numbers of substance abusers not in treatment. The New York State Division of Substance Abuse Services is responsible for assessing the extent of substance abuse in the state population; it conducts statewide surveys of drug use. The most recent survey, conducted in 1986, included a stratified, random sample of more than 6,300 residents 18 years of age and older throughout the state. The results were statistically projected to reflect findings for the 12.7 million adults residing in New York State. An index of severity of substance use was constructed from questions in the survey and served as a basis for estimating the number of adult non-narcotic substance abusers in the state. (Non-narcotic substance abusers include users of substances such as cocaine, marijuana, hallucinogens, and/or the nonmedical use of psychoactive prescription drugs.) The survey also produced data on the number of children under the age of 18 years living in the household.

A second statewide survey conducted by the Division of Substance Abuse Services was a drug use survey among secondary school students between the ages of 12 and 17 years. The most recent such survey was conducted in 1983, with more than 27,000 students participating throughout the state. The results were statistically projected to reflect findings for the 1.5 million students enrolled in public and private schools in grades 7 through 12. Here, too, an index of severity of substance use was constructed from questions in the survey and served as a basis for estimating the number of non-narcotic substance abusing youth in the state.

In addition to estimates of non-narcotic substance abusers, special estimates of narcotic abusers have been generated. Since surveys of the general population of adults and students do not provide an effective basis for sampling narcotic abusers, another strategy for estimating the prevalence of illicit narcotic abuse was devised. This strategy first used regression analysis to relate indirect indicators of narcotic abuse for 1970 through 1974—including opiate arrests, treatment admissions, number of cases of hepatitis, and price and purity of retail heroin—to estimates of the number of narcotics abusers, based on the New York City Narcotics Register data for 1970 through 1974. The New York City Narcotics Register ceased to issue reports in 1974 and ceased operation shortly after. Thus, the ability to make subsequent estimates of the narcotic abusing population became limited. When Narcotics Register data were no longer available, adjustments in estimates over time have been dictated by trends in indirect indicators of narcotic abuse in New York City.[9] Current estimates depend more on recent indicator trends than on the initial data.

Birthrates. For narcotic abusing women, an estimated birthrate of 4% annually was based on estimates of births to New York City women who abused narcotics.[10] For non-narcotic abusing women, birthrates for the general population were used and were derived from 1980 New York City census figures for numbers of women in age categories 12–44 years, and the number of New York City births, by age category, in 1984. All totals are based on a sum of the number of births calculated by age category.

RESULTS

Trends. Data for the state as a whole were not available. Although the data collected were underestimates of the total number of births to substance abusing women, the consistency of the trends in the number of births over time can indicate whether increases can be anticipated. Figure 1 shows the trend of such births from 1978 through 1987 based on data derived from birth certificates. From 1978 to 1983, the number remained below 1,000. There

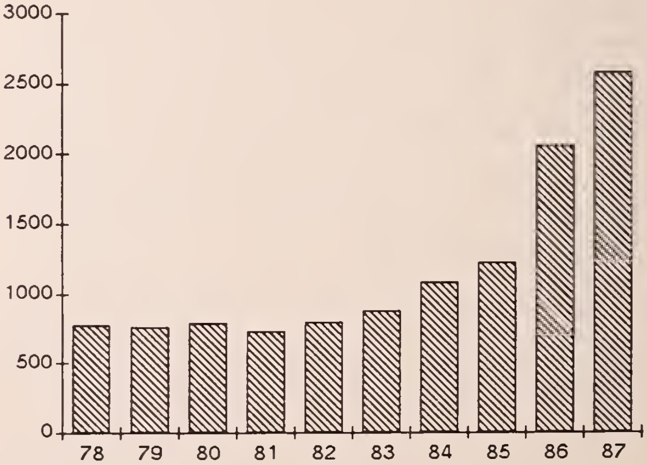

FIGURE 1. Births to substance abusing women in New York City, 1978–1987 (source: NYC Department of Health, Bureau of Vital Statistics).

TABLE I. New York City Births with Maternal Cocaine and Heroin Use, 1978–1986

Year	Number of Births* Cocaine	Heroin
1978	68	206
1979	83	205
1980	106	248
1981	106	266
1982	184	304
1983	266	296
1984	482	287
1985	628	286
1986	1,364	361

* May include other drugs, and these categories are not mutually exclusive.
Source: NYC Department of Health.

has been a steady increase since 1981, with the largest increases occurring in the last two years. In 1985 there were 1,227 such births (a rate of 10.3/1,000 live births), compared with 2,066 such births in 1986 (a rate of 17.0/1,000 live births) and 2,586 in 1987 (a rate of 20.3/1,000 live births). Although some of this increase may be due to increased attention to this issue, the consistency of increase since 1981 probably indicates an underlying trend.

Table I presents a summary of the number of mentions of cocaine and heroin on New York City birth certificates from 1978 through 1986. Although there has been an increase in the number of persons reporting heroin use (nearly doubling), the number indicating cocaine use has increased most dramatically, by a factor of 20, from 68 in 1978 to 1,364 in 1986. In 1986, cocaine was mentioned in two thirds of New York City birth certificates that mentioned maternal substance abuse.

Table II presents data for the first two years of the Infant Health Assessment Program (personal communications, New York City Department of Health, 1987, 1988) which identifies high risk newborns in 12 hospitals in New York City. Maternal substance abuse accounted for about half of all newborns registered (4,572 out of 9,649) out of the 70,523 live births during this period. Thus, these births account for more than 6% of all births in these New York City hospitals. Although these hospitals are not likely to be representative of all hospitals in New York City, what is most important, perhaps, is that within these first two years there has been an increasing trend in the proportion of births identified because of maternal substance abuse, from 43% in the first few months to 61% in the most recent months. Although the magnitude of the increase may in part be due to an increased awareness resulting from the IHAP system itself, the trend is consistent with other indicators.

Estimates of the Number of Births. Estimates of the number of births in 1986 to females who were regular substance abusers were developed by type of drug use (narcotic and non-narcotic) and treatment status (in treatment and not in treatment). Table III presents estimates of the number of births to women in New York State who had a history of regular narcotics use and the number of births to women who were non-narcotic substance abusers.

Of the 300 births to women who were former non-narcotic abusers in treatment, it is estimated that approximately 60% were to women who were cocaine abusers. By far the largest estimated number of births were to women who abused non-narcotic substances (eg, cocaine, cannabis, hallucinogens, and psychoactive prescription drugs taken nonmedically) and who were not in treatment. These numbers—402,000 women and 21,300 births—are further elaborated in Table IV, by age of mother and by type of substance (cocaine vs other nonnarcotic substances). These estimates indicate that almost one quarter of such births are to teenagers.

Overall, the total number of births to substance abusing women in New York State in 1986 is estimated to be more than 24,000, of which approximately 10% were births to narcotic abusers (2,600 births), an additional 10% were to regular cocaine users (2,500 births), and the remainder (80% or 18,800) were to women who were regular users of other non-narcotic substances.

In addition to births to women who are substance abusers, the threat of AIDS transmission to newborns raises the need to estimate the number of births to women who are not themselves narcotics users, but who are the sexual partners of intravenous drug users. It has been estimated that there are 100,000 female sexual partners of intravenous drug users who do not themselves inject drugs.[11] Using a conservative birthrate of 4% (the same rate used for narcotics-using women), there would be an additional 4,000 births per year to women who are sexual partners of intravenous drug users.

Estimates of the Number of Children of Substance Abusers. Data sources were available for estimating the

TABLE II. Births Associated with Maternal Substance Abuse, 1986–1987, in New York City IHAP Hospitals

	Births and Registrations in IHAP Hospitals*				
				% due to Substance Abuse	
Time Period	Live Births	Total	Registered For Substance Abuse	Of all Births	Of all Registered
1986					
Jan–April	11,272	1,301	554	5	43
May–Aug	11,415	1,421	652	6	46
Sept–Dec	11,814	1,408	735	6	52
1987					
Jan–April	11,292	1,475	801	7	54
May–Aug	12,099	1,548	870	7	56
Sept–Dec	12,607	1,519	911	7	60

* Data based on 12 NYC hospitals: 11 municipal and one voluntary.
Source: NYC Department of Health, IHAP Program.

TABLE III. Estimate of Births to Female Narcotics and Non-narcotic Users in New York State*

Substance and Treatment Status	Number of Women†	Number of Births†
Narcotic		
In treatment	12,100	500
Not in treatment	52,900	2,100
Total	65,000	2,600
Non-narcotic		
In treatment	4,700	300
Not in treatment	402,000	21,300
Total	406,700	21,600

* Source: "In treatment" data based on the NYS Division of Substance Abuse Services (DSAS) program reporting system for patients in state-funded and non-funded treatment programs; "Not in treatment" data based on DSAS estimates of narcotics users and statewide surveys.

† Source: For narcotics abusing women, based on 4% rate[10]; for non-narcotics abusing women, based on age-specific birth rates for the general NYC population, combining births across age categories.

TABLE IV. Estimate of Births to Female Nonnarcotic Abusers, Not in Treatment, by Substance and by Age, in New York State

	Regular Users of		
	Cocaine*	Other Non-Narcotics	Total
Number of women	37,000	365,000	402,000
Number of births by age of woman (yr)*			
12–19	400	4,700	5,100
20–29	1,100	11,000	12,100
30 and over	1,000	3,100	4,100
Total	2,500	18,800	21,300

* Regular users of cocaine have used cocaine at least four days during the past month. Regular users of other non-narcotic substances have used two or more such drugs in the past six months or have used marijuana at least ten days in the past month, or any other drug at least four days during the past month.

† Based on age-specific birth rates for the general population of females in NYC, combining births across age categories.

number of children of substance abusers in and out of treatment. Table V presents information on the total number of former substance abusers in treatment programs in New York State, as of August 1987, by primary drug of abuse. The number of children reported are estimates of patients' biological children, aged 17 years and under, some of whom may not be living with their parents. Data are also presented separately for male and female patients, since the number of children varies by sex, and female patients are most likely to have children living with them and/or to be the parents with primary responsibility. As of August 1987, it is estimated that the total number of children of patients in substance abuse treatment was 59,100; more than three quarters were children of former narcotics abusers in treatment. Furthermore, more than one third of these children (35%) were under six years of age.

For substance abusers not in treatment, separate estimates were made for those who were narcotics abusers and for those who abused non-narcotic substances. Information for narcotics abusers was based on estimates of the number of narcotics abusers previously calculated by the Division of Substance Abuse Services. Information on children in households with adults regularly using other substances was based on the most recent household survey conducted by the New York State Division of Substance Abuse Services. Table VI presents the estimated number of children, by age, of individuals with a history of regular narcotics use. It is estimated that there were approximately 205,000 children of narcotics abusers not in treatment, and that nearly half (42%) of these children were less than six years of age.

Table VII presents totals of the number of children, under 17 years of age, residing in households in which at least one adult reported being a regular substance user. In some cases, however, these children may not be the children of the substance abusing adult.

The total number of children of substance abusers, defined here as children of substance abusers in or out of treatment, or children residing with a substance abuser, is

TABLE V. Estimates of Children of Substance Abusers in Treatment, in New York State

Primary Drug of Abuse by Sex of Patient	Number of Adults	Number of Children by Age (yr)*			
		Under 6	6–11	12–17	Total
Narcotics					
Males	25,400	12,800	12,100	9,500	34,400
Females	12,100	5,800	7,300	4,900	18,000
Cocaine					
Males	4,800	1,300	1,600	1,100	4,000
Females	2,300	1,000	1,200	600	2,800
Other					
Males	4,600	1,200	1,500	1,000	3,700
Females	2,400	1,000	1,200	600	2,800
Total					
Males	34,800	15,300	15,200	11,600	42,100
Females	16,800	7,800	9,700	6,100	23,100
Estimated totals†	51,600	20,800	22,400	15,900	59,100

Source: Number of adult patients was based on data from the NYS DSAS program reporting system for patients in funded and non-funded programs.

* Based on survey of patients in treatment.

† Basing the total number of children on the sum of the estimates for male and female patients may in some instances result in double-counting since a child could be attributed to both parents in treatment. This factor was incorporated by reducing the estimated total by 10%.

TABLE VI. Estimates of Number of Children of Narcotics Abusers Not in Treatment, in New York State*

Age of Children (yr)	Sex of Adult Substance Abuser Male	Female	Total†
Under 6	65,300	30,600	86,300
6–11	60,600	20,500	73,000
12–17	38,000	12,600	45,500
Total	163,900	63,700	204,800

* Based on a total of 260,000 estimated narcotic users in NYS, subtracting 37,500 in treatment and 15,500 estimated to be under the age of 18 years. For the number of children per person, the rate developed from the survey of patients in treatment was reduced by approximately 25%, since narcotics abusers not in treatment are likely to be younger than those in treatment, and continued narcotics use has been found to affect fertility levels.

† As in the previous table, the total number of children was reduced by 10% to account for double-counting of children both of whose parents are narcotics abusers not in treatment.

almost a half million, approximately 467,000. Almost half of these children (42%) are younger than six years of age and are with adult substance users who are not in treatment. In addition, there are approximately 24,000 births to female substance abusers annually. Table VIII provides a summary of the estimates presented in this paper for births and children of substance abusers, by treatment status of parent and by type of substance use.

CONCLUSIONS

Based on the data presented, it is conservatively estimated that close to a half million children in New York State aged 17 years and under—about one out of every ten children in the state—are children of substance abusers. Furthermore, during 1986 alone, it is estimated that 24,000 children were born in New York State to women who were regular users of substances (approximately one out of every ten births in the state). Given the fact that the majority of children of substance abusers are under the age of 12 years and are children of parents who are not receiving treatment services, many of these children may need services for many years to come.

In calculating these estimates, there are several possible sources of error, as the note to Table VIII indicates. These were errors resulting in both overestimating and underestimating. First, there is the problem of overestimating due to double counting. Although a correction for double counting was incorporated in estimating the numbers of children of substance abusers of patients in treatment (see footnote to Table V) or of narcotics abusers not in treatment (see footnote to Table VI), many of these children may still be double counted across different categories, such as a child of a narcotic abuser in treatment and of a non-narcotic substance abuser not in treatment. Second, and much more important, is the problem of underestimates. Cross-sectional studies of the population, which were the basis for estimating non-narcotic substance abusers, do not effectively sample some elements of the population who are particularly likely to be substance abusers. Household surveys, for instance, find it difficult to reach those who have unstable households, those who double up with other householders, or those who have no household at all. Also, school surveys cannot assess substance abuse among those who have dropped out of school. Consequently, estimates of substance abusers drawn from these surveys must be considered an underestimate. Furthermore, these surveys were conducted before the impact of "crack" use was fully felt in New York State; the school survey was conducted in 1983, and the survey of adults was conducted in the first half of 1986. Given these considerations, the overall error in the estimates is most probably in the direction of undercounting the number of substance abusers, and therefore underestimating the number of children of substance abusers in New York State. Although it would be helpful to give confidence limits around the estimates presented, the problems of undercounting and survey bias noted above cannot be quantified for this purpose.

TABLE VII. Estimates of Number of Children in Households in which at Least One Adult Is a Regular Non-narcotic Substance User* (Not in Treatment), in New York State

Age of Children (yr)	Primary Substance Cocaine	Other	Total
Under 6	14,000	75,000	89,000
6–11	10,000	43,000	53,000
12–17	6,000	55,000	61,000
Total	30,000	173,000	203,000

* These figures are underestimates of the number of children residing with non-narcotic substance abusers. The survey was based on reported use by one adult, 18 years and older per sampled household, and therefore any substance use by other adults in the household would not be noted.

Also, the estimates presented here reflect overall findings for New York State. The proportion of the estimates represented by New York City, however, deserves comment, since in some ways the situation is very different from the rest of the state, though in some ways it is very similar. Rates of narcotic abuse and crack abuse are much higher in New York City than in the rest of the state. Rates of other non-narcotic abuse and the misuse of psychoactive prescription drugs, though, are very similar; statewide surveys of adults and school surveys conducted by the State Division of Substance Abuse Services show the similarity in rates for New York City residents and residents in the rest of the state. Thus, within the estimates for New York State, New York City, with about 41% of the state's population and 58% of the substance abusers, provides at least half of the children of substance abusers or more.

Irrespective of where in the state these parents and children reside, few are receiving the services needed. There are services available that would both help prevent health and substance abuse problems for the child and would assist the parents in their own rehabilitation.

Some efforts to provide these services have been initiated, but they are few in number, and reach only a small proportion of the affected children. The New York State Division of Substance Abuse Services has funded several services in its treatment programs for parents and their children and has developed guidelines to encourage additional services for parents in treatment.[12] Surveys of treatment populations indicate that parents in treatment are interested in receiving services.[7,8] Resources for increased services are needed, particularly to develop outreach efforts for substance abusing parents who are not in treat-

TABLE VIII. Summary of Estimates of Births to Substance Abusing Women and Estimates of Children of Substance Abusers*, in New York State

Status of Substance Abuser	Births to Females	Age of children (yr) Under 6	6–11	12–17	Total
In treatment					
Narcotics*	500	16,700	17,500	13,000	47,200
Cocaine*	200	2,100	2,500	1,500	6,100
Other non-narcotics*	100	2,000	2,400	1,400	5,800
Totals*	800	20,800	22,400	15,900	59,100
Not in treatment					
Narcotics*	2,100	86,000	73,000	45,000	205,000
Cocaine	2,500	14,000	10,000	6,000	30,000
Other non-narcotics	18,800	75,000	43,000	55,000	173,000
Totals	23,400	175,000	126,000	106,000	408,000
Estimated combined totals	24,000	196,000	148,000	122,000	467,000

* Includes adjustments previously made, within categories, for double counting.

Note: The specific estimates in each category may be under- or overestimates. Although a correction for double counting was incorporated in estimating the numbers of children of substance abusers of patients in treatment or of narcotics abusers not in treatment, many of these children may still be double counted across different categories (eg, a child of a narcotic abuser in treatment and a nonnarcotic substance abuser not in treatment). However, as noted previously, the estimates of children of substance abusers of non-narcotic substance users not in treatment are likely to be underestimates.

ment. As indicated by the data, there are nearly seven times more children of substance abusers who are not in treatment than of substance abusers in treatment. The trends in births to substance abusers indicate that the numbers of these children are likely to continue to increase. Furthermore, research indicates that without intervention, many of the current children of substance abusers are likely to themselves become substance abusers and become parents to another generation of children of substance abusers.[6]

Births to women using intravenous drugs are of particular concern in light of the current AIDS epidemic. It has been estimated that more than 50% of the female intravenous drug users in New York City have already been exposed to the AIDS virus[10] and could therefore transmit the virus to their children in utero or at birth.

A wide range of services is clearly needed to assist these families and break the cycle of drug abuse: services to the parent (parent training); services to the child (assessment and referral); services to the family (family counseling); and services to staff of agencies likely to come in contact with these parents (skills for discussing parenting issues with their patients and skills for identifying children of substance abusers).

The magnitude and urgency of the problem indicate that services must be developed now. In addition, resources devoted to research and evaluation efforts are also needed. Research is needed in many areas, including further assessing the nature and extent of this problem, evaluating the effectiveness of existing programs, and developing novel approaches for reaching and serving affected children and their parents. As a final indication of the urgency of the problem, births to cocaine-using women in New York City have more than doubled since this paper was written.

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Changing concepts of the pathogenesis of diabetic nephropathy

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The diagnosis of diabetes mellitus is often made during childhood or adolescence by the pediatrician. However, this physician rarely continues to see the patient until an age when the long term complications of this disease become clinically manifest. On the other hand, internists frequently become involved with the patient after complications have started to appear, and when it may be too late to institute preventive measures. By these standards, diabetic nephropathy can be considered an "orphan" disease. It is a complication that affects almost half of the diabetic patients in their adult years, inflicting significant morbidity and mortality. Recent results of clinical studies and studies in animals suggest that early identification of those diabetic individuals who will go on to develop the renal complications of the disease may be quite useful. By institution of early preventive treatment in these patients, the pediatrician may then be able to play a role in reducing the incidence of diabetic nephropathy.

The annual incidence of insulin dependent diabetes mellitus (IDDM), or type I diabetes, is 16 cases per 100,000 population.[1] Of these new cases, it is estimated that 30% to 50% will go on to develop nephropathic changes leading to end stage renal disease (ESRD).[2] At present, it is unclear why a significant number of patients with diabetes do not develop renal disease, and why others go on to develop severe renal and nonrenal complications.[3] Diabetic nephropathy now accounts for 25% of all new cases of end stage renal disease in adults,[4] with an estimated medical cost for treatment of more than $1 billion annually.

These disturbing facts, and the severe morbidity that these complications inflict on patients, are compelling reasons why we need to understand better the pathophysiologic process that leads to nephropathy, and other serious complications of insulin dependent diabetes mellitus. Likewise, we need to explore methods to retard or interrupt the progress of these complications. In this article, we review the clinical course of diabetic nephropathy, as well as the present understanding of the pathophysiologic events that result in renal complications. Results of animal studies and findings from recent clinical trials will help demonstrate the types of interventions that are now being explored.

CAUSES OF COMPLICATIONS OF IDDM

There are three major complications of insulin dependent diabetes mellitus: nephropathy, neuropathy, and retinopathy. It is tempting to try to cite a single cause for all three. Three mechanisms have been proposed to explain these complications: non-enzymatic glycosylation of proteins; alterations in sorbitol and myo-inositol metabolic pathways; and changes in microvascular hemodynamics.

The first of these mechanisms is responsible for the production of hemoglobin A_1C (glycosylated hemoglobin).[5] In addition, non-enzymatic glycosylation modifies other proteins and affects their functioning. Non-enzymatic glycosylation may be an important factor in several of the complications, including renal disease, and will be discussed in more detail later in this article. The second mechanism results in modification of the function of the enzyme Na-K ATPase, which is important in energy metabolism.[6] This pathway may be especially important in the neuropathy of diabetes. The third mechanism has been proposed to be the primary disturbance in diabetic nephropathy and will be discussed in detail below.[7] At the present time, we do not know how much each of these mechanisms contributes to long term complications.

CLINICAL COURSE OF DIABETIC NEPHROPATHY

The clinical course of diabetic nephropathy has generally been divided into a preclinical phase and an overt clinical phase. This division is clearly demarcated by the onset of significant albuminuria (Fig 1). However, better diagnostic tools have resulted in identification of an intermediate phase of the disease. This latter phase is referred to as incipient diabetic nephropathy, the hallmark of which is microalbuminuria.[8]

The preclinical phase of the disease is best characterized by renal hypertrophy and hyperfiltration.[8] Evidence from radiographic studies has shown that the kidneys are enlarged[9] but can revert to normal size with proper insulin therapy and glycemic control. Light microscopy of renal biopsy material has shown that gross enlargement of the affected kidney is a reflection of enlargement of the glomeruli.[10]

Functionally, in the early phases of diabetes, the glomerular filtration rate (GFR) frequently is significantly elevated above that of nondiabetic controls (140 mL/min vs 111 mL/min). Renal plasma flow is also elevated, but to a lesser degree.[11,12] This increase in renal function also

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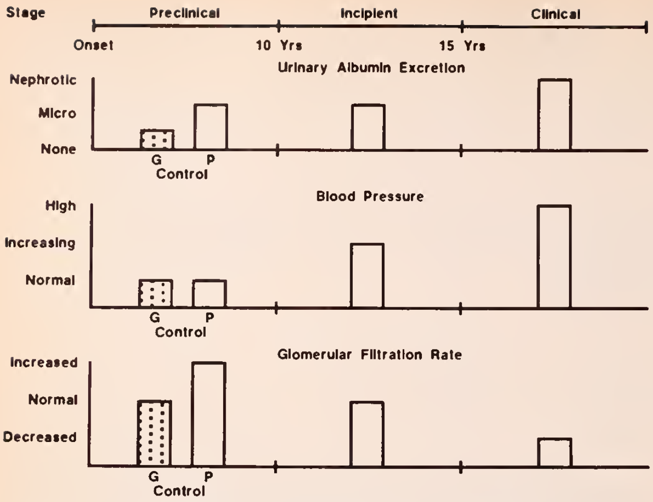

FIGURE 1. The clinical course of patients in whom diabetic nephropathy develops. During the preclinical stage, renal function is usually normal in patients under good control (G), but glomerular filtration rate is increased and microalbuminuria is present when control of blood sugar is poor (P).

returns toward normal with adequate control of the disease. However, not all patients have normal glomerular filtration rates after restoration of a normal blood sugar level. Instead, hyperfiltration continues throughout the preclinical phase of diabetic nephropathy.[13] This latter group of patients with persistently elevated GFR may, in fact, represent an important subgroup of patients who are likely to go on to have progressive renal complications.[14] Systemic blood pressure is generally normal during this early stage.

Studies have also shown that, in patients with diabetes of relatively short duration, exercise provokes an increase in urinary albumin excretion (UAE) above that of nondiabetic patients.[15] This finding again suggests that even in the early phase of diabetes, there are significant functional abnormalities.

There is a transition period between the preclinical phase, in which there is normal urinary albumin excretion, and the overt clinical phase, in which there is significant albuminuria. This period has been labeled incipient diabetic nephropathy (Fig 1), suggesting that the nephropathy is reversible at this time. This phase is characterized by small but persistent elevations in urinary albumin excretion, termed microalbuminuria. Microalbuminuria represents the excretion of from 30 to 300 μg/min (40 to 430 mg/24 h) of albumin. Detection of the small concentrations of albumin in the urine at these low excretion rates has been made possible by new techniques for measuring albumin in urine, including a sensitive radioimmunoassay. It is important to note that microalbuminuria may be present intermittently during the preclinical phase, when a patient's diabetes is not in good control (Fig 1). As with renal hypertrophy and an elevated glomerular filtration rate, in many patients microalbuminuria can be reduced at this time by lowering and stabilizing the blood glucose concentration.

During the phase of incipient nephropathy, other causes of increased protein excretion must be considered. These include poor glycemic control or exercise-induced microalbuminuria, as well as nondiabetic renal or systemic disease.[16] Also in this stage, blood pressure is elevated in many patients. Blood pressure continues to increase in diabetics who progress to the stage of overt clinical nephropathy. In contrast, other diabetics have stable blood pressure, although the pressure is elevated compared to normal controls.[17]

It appears that incipient diabetic nephropathy begins five to seven years after onset of the diabetes and persists for five to ten years.[8] This intermediate phase may be particularly important in identifying those patients who will progress to the advanced stages of the disease. It is unclear whether persistent microalbuminuria represents the onset of progressive renal disease, rather than acting as a marker for predicting the occurrence of future disease. If microalbuminuria truly represents an incipient phase of the disease, it should be reversible. Presently, research is focusing on this phase of the disease, in an attempt to establish it as a prognostic marker for future renal complications.

The final stage of overt diabetic nephropathy is marked by evidence of clinical proteinuria (with loss of albumin of more than 500 mg/24 h). Patients may then develop the florid nephrotic syndrome (Fig 1).[16] This stage rarely occurs within the first ten years of diabetes and most commonly occurs after 15 to 20 years of disease.[18] For patients who survive 30 years of diabetes without clinical proteinuria, there is little chance that renal complications will develop.

For patients who have abnormal renal function and who often have overt hypertension, progression to end stage renal disease is the inevitable course once clinical proteinuria begins. The glomerular filtration rate begins to decline at a rate ranging from 7 to 30 mL/min/year. A correlation between mean blood pressure and the rate of decline in GFR has been found. In patients with hypertension, renal failure develops sooner after the onset of proteinuria than in patients without hypertension. The prevalence of hypertension also appears to rise progressively with advancing stages of nephropathy. In one study of 200 patients with elevated serum creatinine (greater than 1.5 mg/dL), 92% had concomitant systolic blood pressures greater than 160 mm Hg.[19]

Effective treatment of hypertension can retard the rate of progression to end stage renal disease. Long term antihypertensive treatment reduces albuminuria and slows the declining GFR by nearly 60%.[20] Thus, the relationship of diabetic nephropathy to arterial blood pressure appears to be complex: nephropathy leads to elevated blood pressure and hypertension exacerbates the renal failure.

PATHOGENESIS OF GLOMERULAR LESIONS IN DIABETIC NEPHROPATHY

Despite extensive research on the pathogenesis of glomerular injury in diabetes, the exact mechanism of the disease remains obscure. Many researchers have examined the abnormal conditions of the diabetic milieu that might lead to renal complications. Much controversy has surrounded the issue concerning the usefulness of "tight glycemic control" for diabetes. At present, the accumulated evidence suggests that in diabetic patients who are euglycemic, or who have near normal blood glucose levels, renal disease develops less often.[21] This evidence forms the basis for the present goal of clinical management of diabetes, namely, euglycemic control.

Until recently, there has been no adequate method for consistent, frequent, and accurate monitoring of blood glucose. Recent innovations include the introduction and routine use of glycosylated hemoglobin (Hgb A_1C) and home blood glucose monitoring. With these advances, it is now possible to undertake studies to determine the importance of strict metabolic control of glucose, and its relationship to the development of renal disease and other complications.

As already discussed, the hyperglycemic state in diabetes provides a biochemically abnormal environment that may play a role in the pathogenesis of renal disease. One of the most discussed biochemical reactions in diabetes is non-enzymatic glycosylation. In this reaction, glucose combines with a variety of structural and circulating proteins to an extent that depends directly on the blood sugar concentration.[5,21] Measurement of glycosylated hemoglobin is based on this phenomenon in red blood cells. It is not surprising that the glomerular basement membrane (GBM) is also subject to glycosylation. Whether or not the glycosylation of glomerular basement membrane proteins translates into structural alterations is speculative at this time. However, glycosylation of either circulating proteins or glomerular constituents may have a direct effect on the filtering properties of the glomerulus. Alterations in the charge of glomerular basement membrane proteins by glycosylation may interfere with the charge selectivity of the glomerular filtering apparatus. This defect may cause increased permeability of the membrane to albumin.[17]

Recently, abnormal changes in glomerular microvascular hemodynamics have been considered as part of the central pathogenic mechanism in the development of renal injury in diabetes.[22] As already discussed, studies have shown that many young diabetics have supranormal glomerular filtration rates.[7,11] It is now proposed that this state of hyperfiltration leads to intraglomerular hypertension and increased glomerular blood flow. These changes may in turn cause permanent glomerular injury and renal failure.[23]

Renal ultrafiltration is determined by a number of factors. Understanding these factors will help delineate the basis for the proposal that intrarenal perfusion pressure is the mechanism leading to diabetic nephropathy. There are several forces involved in the regulation of single nephron glomerular filtration rate (SNFGR) (Fig 2): the pressure gradient between the glomerular capillary bed and Bowman's space—the transcapillary hydrostatic pressure (P), which favors filtration; the difference between plasma oncotic pressure and the oncotic pressure in the glomerular filtrate (f), which opposes glomerular filtration; the total capillary surface area of the glomerulus that is available for filtration (S); and the effective hydraulic permeability of the glomerular capillary (k). The product of S and f is called the ultrafiltration coefficient (K_f), while the difference between the transcapillary hydrostatic and oncotic pressure is the net ultrafiltration pressure (P_{uf}).

The final factor important in the regulation of the glomerular filtration is glomerular plasma flow rate (Q_A). In some experimental models, variation in the rate of flow of plasma through the glomerular capillary bed will have a proportional effect on the rate of glomerular filtration.[24]

$$\boxed{\mathrm{SNGFR} = k \cdot S \cdot (\triangle P - \triangle \pi) = K_f \cdot P_{uf}}$$

SNGFR = Single Nephron Glomerular Filtration Rate
k = Effective Hydraulic Conductivity
S = Glomerular Capillary Surface Area
$\triangle P$ = Transglomerular Capillary Hydrostatic Pressure
$\triangle \pi$ = Transglomerular Capillary Oncotic Pressure

FIGURE 2. Diagram showing the relationship between single nephron glomerular filtration rate and intrarenal pressure (for explanation, see text).

Finally, it should be noted that the renal glomerulus is unique, in that there is a precapillary resistance both proximal to the glomerular capillaries (the afferent glomerular arteriole) and distal to the glomerular capillaries (the efferent glomerular arteriole) (Fig 3). The pressure and the rate of flow through the glomerular capillary bed depends on the relative relationship between these two resistances. Dilation of the afferent arteriole will result in increased renal blood flow and increased intraglomerular pressure. On the other hand, dilation of the efferent glomerular arteriole will also result in an increase in blood flow rate, but will lead to a decrease in the pressure in the glomerular capillaries.

It has been proposed that changes in the rate of glomerular perfusion and in the intraglomerular blood pressure may be responsible for decreases in renal function seen in a number of forms of experimental and human kidney disease that lead to renal failure, including diabetes. For example, removal of one kidney of a rat and the segmental infarction of five sixths of the remaining kidney results in a doubling of the glomerular filtration rate of the remaining nephrons within a week when compared to that of control animals.[23] In addition, the plasma flow through the remaining glomeruli and the pressure within these glomeruli were increased in the nephrectomized animals.[7,23] The remaining nephrons gradually lost function, and the experimental animals eventually died of renal failure.

In another experiment, an attempt was made to modify the course of renal failure in animals that were five-sixths nephrectomized by changing the protein content in their diet. Reduction of dietary protein results in a decrease in glomerular filtration rate, while increasing protein intake leads to an elevated filtration rate. In this study, a group of animals again underwent a five-sixths reduction in renal

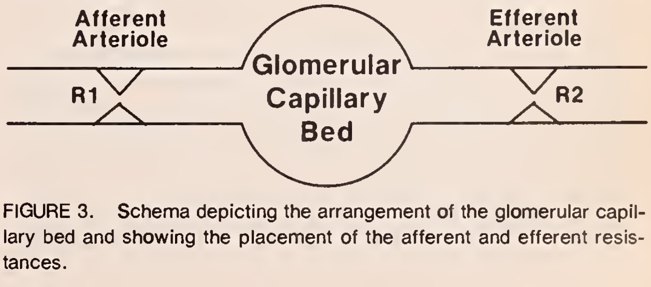

FIGURE 3. Schema depicting the arrangement of the glomerular capillary bed and showing the placement of the afferent and efferent resistances.

mass. Some of the animals were placed on a low protein diet (6%). Animals on this diet had a glomerular filtration rate in the remaining nephrons that was significantly lower than that of animals on a standard diet. The reduced rate of glomerular filtration in the animals on the low protein diet was the result of a lack of increase in the plasma flow to the remaining glomeruli as well as a reduction in the intraglomerular pressure.[25] With the near normal GFR, there was less long term structural damage in the remaining glomeruli and reduced mortality among the animals.[23]

Other studies using a similar animal model have demonstrated that inhibitors of angiotensin I converting enzyme affect renal function in a similar manner as reducing protein intake.[26] Thus, treatment of the partially nephrectomized rat with the angiotensin I converting enzyme inhibitor, enalapril, causes the increased GFR to return toward normal; renal function is then maintained in a manner similar to that seen when the animals are placed on a low protein diet.

The rationale for the use of angiotensin converting enzyme inhibitors in this situation is based on the action of the renin-angiotensin system on the glomerular arterioles.[27] Angiotensin II appears to act by preferentially constricting the efferent glomerular arterioles (Fig 3, R2). The renin-angiotensin system can be blocked using drugs that inhibit the conversion of angiotensin I to angiotensin II; these drugs, such as enalapril and captopril, are angiotensin converting enzyme (ACE) inhibitors. Based on the action of angiotensin on the renal arterioles, inhibition of ACE should lead to dilation of the efferent glomerular arteriole, produce a decrease in intraglomerular pressure, and reduce the GFR.

In order to understand the factors responsible for the hyperfiltration in diabetics, and to determine the importance of this finding in the pathogenesis of diabetic nephropathy, animal models using diabetic rats have been employed. Rats are made chemically diabetic with streptozocin,[7] and the single nephron glomerular filtration rate is determined. Diabetic animals have some renal pathologic changes similar, but not identical, to those occurring in human diabetic nephropathy. If left alone, these animals will develop proteinuria and progressive renal failure and will die.

Diabetic rats have a number of functional renal changes, including an elevated single nephron glomerular filtration rate, which is secondary to both an increase in glomerular perfusion rate and increased transcapillary hydrostatic pressure. These renal abnormalities are similar to those seen in the five-sixths nephrectomized animals.

Two approaches have been taken to modify the changes in intrarenal hemodynamics in diabetic animals. In one study, animals were placed on reduced dietary protein intake,[28] while in another study animals were placed on the angiotensin I converting enzyme inhibitor, enalapril.[26] Both treatments led to reversals in the physiologic abnormalities caused by diabetes. These changes were again correlated with decreases in glomerular plasma flow and intraglomerular pressure.

An interesting clinical counterpart has been reported in a patient with diabetes and unilateral renal artery stenosis and hypertension. The pathognomonic lesions of Kimelsteil-Wilson disease were visible in the unprotected kidney, whereas the stenotic renal artery protected the other kidney from the high blood pressure and prevented the development of these same lesions.[29] This observation supports the concept that intraglomerular hypertension is the primary pathogenic mechanism in the nephropathy.

The exact underlying mechanism of renal injury is still obscure. However, experimental and clinical observations give strong support to alterations in renal hemodynamics as being a promoter of the renal complications of diabetes. Persistently elevated intraglomerular pressures and hyperfiltration, as seen in the hyperglycemic state, appear to damage the functional and morphologic integrity of the glomerulus. It has been suggested that once even a small number of glomeruli become sclerosed, the remaining, unaffected glomeruli respond with compensatory hyperfiltration. This leads to further glomerular sclerosis, which sets up a destructive feedback loop and results in progressive renal damage.[7] What is the critical mass of functioning nephrons below which hemodynamically mediated injury is inevitable remains an unanswered question.[30]

CONCLUSIONS AND SPECULATION: IMPLICATIONS FOR THE FUTURE

The mechanisms involved in the complications associated with insulin dependent diabetes mellitus are complex. A basic summary of the current understanding of the pathogenesis of diabetic nephropathy and other forms of renal disease is shown in Figure 4. Renal disease, which reduces the number of functioning nephrons, and diabetes mellitus both lead to increased blood flow through glomeruli and to increased pressure within glomeruli. These changes in flow and pressure lead to an increase in the glomerular filtration rate. The outcome of this high rate of filtration is a change in the structure of the glomerulus, which produces sclerosis and further reduction in the numbers of functioning units.

In the last few years, we have learned that diabetic nephropathy, as well as other forms of progressive renal disease, takes place on a background of changes in renal hemodynamics. Studies primarily in animal models suggest that the changes can be modified and the progression of the renal failure ameliorated. Studies such as the Diabetes Control and Complications Trial are presently underway

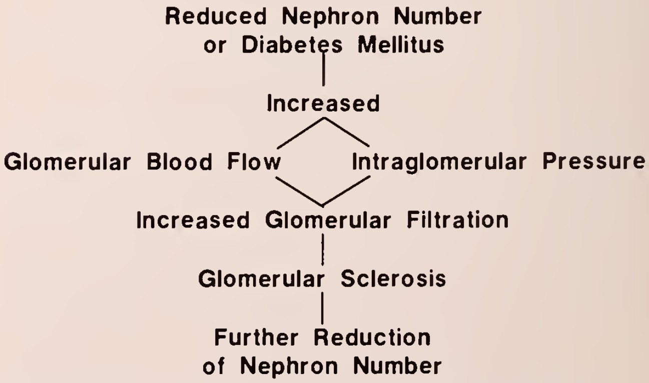

FIGURE 4. Outline of the proposed steps in the pathogenesis of diabetic nephropathy.

to assess whether improved control of the disease can lead to reduced complications.

Based on our present knowledge, we can only speculate on future methods for modifying the course of diabetic nephropathy. There may come a time when we have evidence that drugs such as ACE inhibitors or modification of the protein content of the diet improve the outcome in diabetic humans. That information is not yet at hand.

At the present time, it seems reasonable to attempt to keep diabetic patients under good control. Even in this regard, the definition of "good control" needs to be addressed. However, being sure that patients are educated about their disease, take their insulin regularly, have normal serum lipid levels and blood pressure, and maintain a good activity schedule will all potentially improve the long term outcome.

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Churg-Strauss syndrome

Case Presentation

A 68-year-old man was admitted to the Medical Service of the Manhattan Veterans Administration Medical Center with a one-year history of weight loss, blood-streaked nasal discharge, and blood-streaked sputum. Two years prior to admission, the patient began to experience chest pain and shortness of breath; medical evaluation at that time revealed coronary artery disease, and he underwent a three-vessel coronary artery bypass operation.

One year prior to admission, he was found to have hyperparathyroidism and underwent parathyroidectomy. At that time, the patient also began to complain of difficulty in breathing; nasal polyps were diagnosed and removed surgically. Pulmonary function tests indicated mild obstructive disease, which was treated with anhydrous theophylline sustained action tablets and metaproterenol sulfate. Following the polypectomy, the patient noted intermittent bloody mucous discharge from his nose and mouth, which was treated with tablets and local sprays. Five months prior to admission, the patient moved to Israel and at that time noted the onset of night sweats and weight loss; the nasal discharge continued and a local physician noted the presence of a nasal ulcer. One month prior to admission, the patient experienced intermittent frontal headaches and marked fatigue; diarrhea occurred on two occasions but without subsequent recurrence. He returned to the United States for further medical evaluation. The patient denied arthralgias, myalgias, chest pain, and abdominal pain.

The patient's medical history included peptic ulcer disease 12 years prior to admission, at which time he received seven units of packed red blood cells. He had had hypertension and mild diabetes mellitus controlled by diet. The patient had suffered a skin rash while taking sulfonamides. In addition to those medications mentioned, he was taking beta blockers. A review of all systems was otherwise negative.

The patient was a retired newspaper writer; he denied cigarette smoking and alcohol abuse. His father had died of a myocardial infarction and his mother of leukemia. Two brothers had emphysema.

Physical examination disclosed a well-developed man in no acute distress. His blood pressure was 120/98 mm Hg; pulse rate, 88/min; respiratory rate, 16/min; and temperature, 99° F. The head, eyes, ears, nose, and throat were normal, except for edematous nasal mucosa with bloody crusted nasal discharge. The neck was supple; a well-healed parathyroidectomy scar was present; there was no increase in jugular venous pressure and no arterial bruits were audible. The anteroposterior diameter of the chest was increased; the breath sounds were diminished at the lung bases. The heart sounds were normal; a grade II/VI early systolic murmur was audible along the left sternal border. The abdomen was obese with normal bowel sounds; the liver and spleen were not palpable. There was no spinal or costovertebral angle tenderness. The rectal examination was negative; the prostate was normal to palpation; the stool Guaiac test for blood was negative. The extremities showed slightly tender subcutaneous nodules on several fingertips and hemorrhagic crusted nodules on the soles of the feet. There was no clubbing, cyanosis, or edema.

The neurologic examination revealed good orientation; cranial nerve function and sensory functions were intact. Motor strength was well preserved in all extremities, except for slight weakness (4/5) in the right arm.

Urinalysis demonstrated amber urine with pH 6.0, specific gravity 1.020, 3+ protein, and a trace of blood. The microscopic examination showed 6–10 white blood cells and 6–10 red blood cells per high power field, and moderate amounts of bacteria. The hematocrit was 34%; mean corpuscular volume, 82 femtoliters; white blood cell count, 24,600/μL, with 13% mature polymorphonuclear leukocytes, 15% immature forms, 70% eosinophils, and 2% basophils. The platelet count was 479,000/μL; the prothrombin time and partial thromboplastin time were at control levels. Serum electrolyte levels were as follows: sodium, 139 mmol/L; chloride, 106 mmol/L; potassium, 4.4 mmol/L; bicarbonate, 23.5 mmol/L; and serum calcium, 9.2 mg/dL. The blood urea nitrogen was 15 mg/dL; serum creatinine, 1.1 mg/dL; blood glucose, 174 mg/dL; serum lactate dehydrogenase (LDH), 195 U/L; alkaline phosphatase, 101 U/L; serum albumin, 3.6 g/dL; serum globulin, 2.7 g/dL; direct bilirubin, 0.5 mg/dL; and serum amylase, 62 U/L. The arterial blood gas determinations indicated a pH of 7.47; $P\text{CO}_2$, 33 mm Hg; $P\text{O}_2$, 60 mm Hg; and O_2 saturation, 93%. The serum iron level was 42 μg/dL (normal range, 60–150 μg/dL); iron binding capacity, 174 μg/dL (normal range, 270–380 μg/dL). The serum rheumatoid factor and antinuclear antibody titers were negative; serum tests for hepatitis B surface antigen and surface antibody were negative. The serum level of the third component of complement (C3) was 234 mg/dL (normal range, 86–244 mg/dL).

The chest radiograph showed sternal surgical clips, borderline cardiac enlargement, a small patchy infiltrate in the left lower lobe, and a 2 × 2-cm nodular density in proximity to the pleura in the left upper lobe. The electro-

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Based on a clinicopathologic conference held at New York Infirmary-Beekman Downtown Hospital, New York, NY, on April 11, 1989.

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cardiogram showed normal sinus rhythm, Q waves in leads II, III, and aV_F, and generally low voltage.

On hospital day two, the patient's temperature rose to 102°F; blood cultures were drawn. Urinalysis and repeat chest film added no new information. On hospital day three, the dermatology consultant suggested that the hand lesions were consistent with microemboli. The white blood cell count was 23,000/μL, with 16% mature polymorphonuclear leukocytes, 7% immature forms, 12% lymphocytes, 64% eosinophils, and 1% monocytes.

On hospital day four, the computed tomographic (CT) scan of the paranasal sinuses revealed thickened mucosa in the maxillary, sphenoid, ethmoid, and frontal sinuses, compatible with chronic sinusitis; there was no expansion or erosion of adjacent bone.

On hospital day five, a diagnostic procedure was performed.

Muthiah Sukumaran, MD (Attending Pulmonologist): To summarize, this patient had obstructive airway disease not related to smoking, marked eosinophilia, pulmonary infiltrates with nodules, and multisystemic involvement suggestive of vasculitis.

The patient's eosinophilia can be classified as marked, that is, a total eosinophil count of over 10,000/μL.[1] The CT scan of the chest disclosed a patchy infiltrate in the left lower lobe, a nodule in the left lower lobe laterally (Fig 1), a nodule in the left upper lobe, and a third nodule in the posterior segment of the right lower lobe. None of these nodules showed cavitation or calcification.

I would like to review the various diseases and conditions that include significant degrees of eosinophilia with pulmonary infiltrations, and then to try to select the one that most closely fits the clinical facts of this case.

The combination of eosinophilia and pulmonary infiltrates is called the PIE syndrome (pulmonary infiltrates with eosinophilia). The syndrome has several identifiable as well as idiopathic causes, which I will discuss. First I will describe the identifiable causes.

Induction by drugs. Nitrofurantoin is the most common offender. The patient under discussion developed a rash following the use of sulfonamides, though the time of this event was not stated, and the presence of eosinophilia as well as radiographic evidence of pulmonary infiltrates at that time were not mentioned. So we have some degree of suspicion that his more recent illness may have been related to sulfonamides.

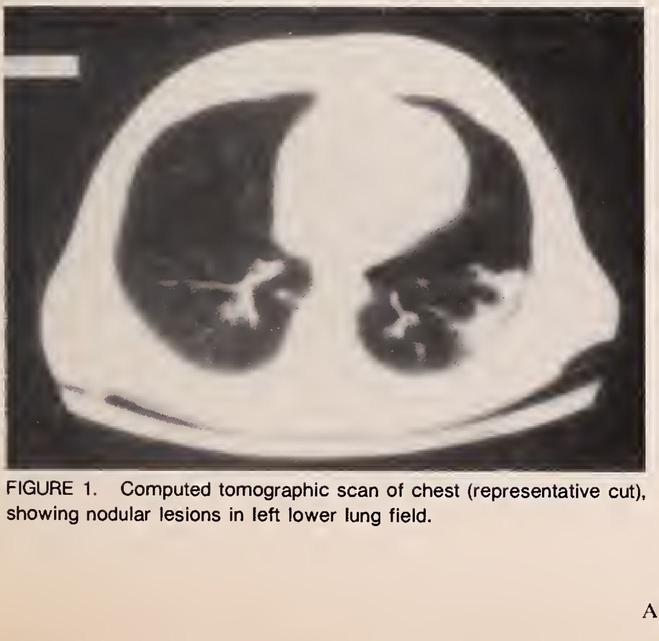

FIGURE 1. Computed tomographic scan of chest (representative cut), showing nodular lesions in left lower lung field.

Parasitic causes. The patient travelled to Israel, which is a locus for common parasitic infestations. Infection with *Ascaris lumbricoides* can produce pulmonary manifestations by the migration of larvae through the lungs, giving rise to an allergic response with eosinophilia, sometimes as high as 70% of the total white blood cell count. Patients with such infections commonly present with cough, difficulty in breathing, and pulmonary infiltrates. The diagnosis is usually made on examination of the stool.

Infection with *Strongyloides stercoralis* causes symptoms following migration of the larval stages of the worm from pulmonary capillaries into the alveolar spaces. The patient experiences dyspnea, cough, and wheezing, with significant eosinophilia.

Ankylostomiasis very rarely produces lung manifestations.

Tropical pulmonary eosinophilia is a condition seen in asthmatics, caused by the nematode worm *Wuchereria bancrofti*. The patients have eosinophilia and pulmonary infiltrates, with dyspnea. In the appropriate setting, the condition should be considered in patients who have asthma unresponsive to conventional treatment.

Pulmonary larva migrans infection includes a tissue migration stage of the larvae of *Toxocara canis*, a parasite of dogs, commonly observed in children who swallow contaminated soil. Though not usually a disease of adults, it has been described in adults; these patients do not have systemic manifestations.

Schistosomiasis is seen in the United States, most commonly in Puerto Rico, and can present with the PIE syndrome. Symptoms include gastrointestinal and genitourinary manifestations, and pulmonary hypertension due to obliterative endarteritis. The eosinophil count is usually not high.

Fungal infections. Bronchopulmonary aspergillosis may present with dyspnea, cough, wheezing, and pulmonary infiltrates, usually perihilar; the eosinophilia is usually not marked. One instance of bronchopulmonary aspergillosis was reported to have progressed to allergic angiitis and granulomatosis, which I will discuss a bit later.[2]

Occupational causes. Hairdressing is an example of an occupation at risk. (The materials used in that line of work are suspect.) In practice we see patients presenting with dyspnea, eosinophilia, and pulmonary infiltrates similar to the PIE syndrome.

Neoplastic diseases. Hodgkin's disease, bronchogenic carcinoma, and lymphosarcoma may be seen in connection with eosinophilia, which, however, may be only a coincidental finding and not caused by these diseases. But cases of bronchogenic carcinoma and Hodgkin's disease with the PIE syndrome have been reported.

There are a few idiopathic diseases associated with eosinophilia and pulmonary infiltrates. They include Löffler's syndrome, for example, for which no cause can be identified in one third of cases.

Chronic eosinophilic pneumonia is an idiopathic condition seen in women. It has a clinical presentation similar to that of Löffler's syndrome, but is characterized by a very protracted course, systemic symptoms such as fever, weight loss, and malaise; and pulmonary symptoms with hemoptysis and dyspnea. Chest films typically demonstrate bilateral peripheral infiltrates. These patients do not exhibit involvement of other organ systems.

The hypereosinophilic syndrome is also known as eosinophilic leukemia, Löffler's fibroplastic endocarditis, and disseminated eosinophilic collagen disease. Some 50 cases of this condition reported by Fauci et al[3] in 1982 met three criteria: persistent eosinophilia, up to 90% of the white blood cell count at times, for a period of at least six months or more; lack of evidence of parasitic or allergic or other known causes of eosinophilia; and signs and symptoms of organ system involvement. Presenting manifestations in the hypereosinophilic syndromes may include fatigue, dyspnea, fever, and rhinitis. Skin manifestations were seen in about 56% of these patients, varying from rashes and angioedema to embolic manifestations most probably arising from the heart.

The cardiac manifestations are the major source of morbidity and mortality. The patient is always at risk of developing a thrombus over an area of endocardial fibrosis. Mitral insufficiency and progressive pump failure have been noted in patients with these conditions. Infrequently, bacterial endocarditis may develop.

The pulmonary manifestations are usually mild, again presenting as a PIE syndrome. The gastrointestinal manifestations are hepatosplenomegaly, abdominal cramps, and diarrhea. Neurologic manifestations are seen in 64% of these patients, presenting as a peripheral neuropathy, or with focal deficits, and sometimes as a mononeuritis multiplex. The patient presented here also had manifestations of altered behavior. Hematologic manifestations include anemia, eosinophilia, thrombi in the lower extremities, and increased serum levels of vitamin B_{12}.

Sinusitis is very rare in patients with the hypereosinophilic syndrome, and renal involvement is almost unknown. Urinalysis is usually normal.

Though the diagnosis of hypereosinophilic syndrome needs to be considered here, I am not convinced that this patient had such a condition. His clinical manifestations, with the PIE syndrome and multiple organ system involvement, suggest some type of vasculitis. It would be helpful to review some of the syndromes that include such a combination.

The first of these is Wegener's granulomatosis, either a limited form or a complete form. My first review of the protocol immediately suggested the possibility of Wegener's disease, since the patient presented with symptoms of upper airway disease, renal disease, and pulmonary involvement. Wegener's granulomatosis was initially described in 1939 by Wegener as a necrotizing granulomatous process with vasculitis, manifested by a triad of involvement of the upper airway, pulmonary disease, and glomerulonephritis.[4]

The full-blown picture of all these may not be seen; thus the term "limited Wegener's granulomatosis" is used in certain cases. Most of these patients have upper airway symptoms, such as nasal discharge, epistaxis, cough, and hemoptysis. Seventy percent of patients exhibit abnormalities on lung radiographs, either patchy infiltrates or nodules which may be single or multiple, sometimes growing into mass-like lesions. The nodules and mass lesions can cavitate and destroy the lung fields; sometimes these nodules pack the lung fields in a manner suggestive of metastatic carcinoma. Skin involvement, seen in about 13% of cases, presents as papules, nodules, ulcers, ecchymoses, or palpable purpuric lesions, such as the patient under discussion demonstrated.

Renal involvement in Wegener's granulomatosis is very common, occurring in about 85% of the cases; many of the patients go on to develop renal failure, which was not present in this patient. Neurologic symptoms include mononeuritis multiplex, which can produce various manifestations, either motor or sensory, but usually a combination of these in one extremity. Eosinophilia has been described, but it is usually mild.

It is true that this patient had upper and lower airway symptoms, pulmonary infiltrates and nodules, skin lesions, and possibly mononeuritis in the right arm. However, against the diagnosis of Wegener's granulomatosis is the lack of renal involvement here, as indicated by normal renal function. This patient had marked eosinophilia, which is a rare finding in Wegener's granulomatosis. I could not find one case in which a 70% eosinophilia occurred. So I do not believe that this patient had Wegener's granulomatosis by itself; if he did have it, it was probably part of a so-called "overlap" syndrome.

Lymphomatoid granulomatosis is a disease that deserves brief consideration. The symptoms are quite similar to those of Wegener's disease, but the patients rarely have upper airway symptoms. Lymphomatoid granulomatosis is largely limited to the thorax, very rarely is skin disease evident, the kidneys are not involved, and no eosinophilia is described. So I must exclude that diagnosis.

The next diagnostic possibility is polyarteritis nodosa (PAN). This diagnosis was difficult to confirm or reject. It is a multisystemic disease dominated by vasculitis, though with less tissue involvement than found in Wegener's granulomatosis. The etiology is not yet understood. PAN has been known in the past by the term periarteritis nodosa, unfortunately a wastebasket term, since many instances of vasculitis of unknown cause are lumped into this group.

The clinical manifestations depend on the system involved. Hypertension is very common, and significant renal involvement is seen in well over 80% of patients. The upper airways are very rarely involved. The pulmonary manifestations are non-specific and include cough and occasionally hemoptysis. The radiographic changes in the lungs are non-specific, either similar to those of Löffler's syndrome or depicting occasional nodules. The central nervous system manifestations are those of mononeuritis multiplex; the severity of various brain stem manifestations depends on the amount of vasculitis within the cranium. This patient's frontal headaches may have been related to a vasculitis involving the central nervous system.

The skin in PAN is very rarely affected, but if involved, it displays subcutaneous nodules, not of the type described in this patient. Eosinophilia in PAN is very rarely as impressive as was seen in this patient, but it can occur.

Because the patient under discussion had prominent upper and lower respiratory symptoms, systemic involvement, and an abnormal urinary sediment, PAN is a difficult diagnosis to establish or to rule out here.

Other causes of vasculitis should be mentioned, including hypersensitivity angiitis due to drugs such as sulfonamides, various connective tissue diseases, infectious disease such as tuberculosis with vasculitis and eosinophilia, and necrotizing sarcoid granulomatosis. However, all of these are rare situations, and I do not believe that any of these illnesses are represented by the patient under discussion.

To be complete, I must mention that vasculitis has been reported to occur in the presence of the human immunodeficiency virus (HIV).[5,6]

Finally, we must consider the condition known as allergic angiitis and granulomatosis, that is, the Churg-Strauss syndrome. In 1951, Drs Churg and Strauss[7] at Mt Sinai Medical Center reported 13 cases of patients who had varying degrees of asthma, nasal and dermatologic symptoms, pulmonary infiltrates, and marked eosinophilia, with vasculitis and/or granulomatosis involving many organ systems. In this group of patients, age of onset varied from seven years to 59 years; the patients had chronic sinusitis, nasal polyps, intermittent fever, weight loss, diarrhea, normal urinary sediments, and a variety of skin manifestations including erythema, maculopapular rashes, palpable purpuric lesions mimicking embolic lesions, and subcutaneous nodules. These patients also manifested several neurologic symptoms, but most commonly those of mononeuritis multiplex.

Since the original description, two subsequent reports, by Dr Chumbley et al at the Mayo Clinic[8] and by Dr Lanham and colleagues in London,[9] have described an additional 46 patients. Three stages in the development of this disease have been described:

1. Predominantly upper respiratory symptoms, along with symptoms of asthma. Allergic rhinitis and nasal polyposis were very common. Patients required repeated surgery for the nasal polyps, and continued to have nasal symptoms. Epistaxes and nasal ulcers were commonly noted. Eighty-eight percent of the patients had radiologic manifestations of chronic pansinusitis without any bone involvement. This patient had a CT scan showing chronic sinusitis but no bony erosions.
2. Eosinophilia of the blood, usually in the marked range, sometimes as high as 90%. Pulmonary infiltrates are consistent with Löffler's syndrome as mentioned earlier; pulmonary nodules have been described with this syndrome, but no cavitation.
3. Multisystem involvement, as described by Drs Churg and Strauss. There was no clear-cut relationship between the asthma and the activity of vasculitis: asthma could precede or start simultaneously with vasculitis, but the shorter the interval between the onsets of these two symptom complexes, the worse the prognosis.[8]

Based on these comparisons, I believe that this patient had Churg-Strauss syndrome. Before concluding, I wish to review the polyangiitis overlap syndrome.[10] This is characterized by systemic vasculitis, with features that overlap several distinct vasculitis syndromes already discussed. This syndrome must be considered for the patient under discussion.

Löffler's syndrome combines eosinophilia with tissue involvement. The Churg-Strauss syndrome and Wegener's granulomatosis combine eosinophilia, tissue involvement, and vessel involvement. In PAN, the findings are predominantly eosinophilia and vasculitis, with little tissue involvement. Temporal arteritis, hypersensitivity angiitis, and rheumatic carditis present various combinations of either vasculitis, or tissue involvement, but no eosinophilia.

I have had much difficulty in ruling out PAN and Wegener's granulomatosis, but I feel that this patient had the Churg-Strauss syndrome, that is, allergic angiitis and granulomatosis. I presume that he had asthma, though it is not mentioned in the protocol. He had obstructive airway disease severe enough to require metaproterenol and sustained-action theophylline. He did not smoke, so we will presume that he had asthma. He had pulmonary and upper airway symptoms, skin involvement, abnormal urinary sediment, and systemic manifestations such as fever, weight loss, headaches, and marked eosinophilia. All these findings fit the diagnosis of Churg-Strauss syndrome. I would suggest that the appropriate diagnostic procedure would have been a biopsy of the skin lesions on the extremities.

Norbert Brau, MD (Medical Resident): On the CT scan of the lung, a portion of the liver was visible. Were there any necrotizing areas within the liver?

Dr Sukumaran: The radiologic report commented only on the lungs. No mention is made of any changes in the liver in the case protocol. In addition, I assume that the nasal polyps in this case might be only the common type of nasal polyps, such as might be seen in any allergic rhinitis, and have no special relationship to the final diagnosis.

Melvin Rosenzweig, MD (Medical Attending): Was there any aspirin sensitivity? You postulated that he might have asthma. He might have polyps related to aspirin sensitivity.

Dr Sukumaran: We have no information along those lines.

CLINICAL DIAGNOSES

Allergic granulomatosis (Churg-Strauss syndrome).

PATHOLOGY PRESENTATION

Gurdip S. Sidhu, MD (Attending Pathologist): The parathyroid specimen removed at surgery before the onset of the patient's more serious symptoms proved to be a chief-cell adenoma and is more or less irrelevant to the main disease which subsequently developed. This specimen from the parathyroidectomy did not show any vasculitis.

The polyps were removed in another institution, and we did not have the resected tissue for pathologic study. However, the patient had crusted bleeding lesions of the nose, and a biopsy from the nose revealed squamous mucosa with ulcerations and a mixed infiltrate of polymorphonuclear leukocytes, fibrin, and eosinophils, but no evidence of vasculitis in this biopsy.

A skin biopsy from the hand was performed, as Dr Sukumaran predicted. Portions of the epidermis showed necrotic lesions characteristic of infarction (Fig 2) when the blood supply has been cut off. Cellular infiltrates in the deeper dermis were related to the blood vessels. The ves-

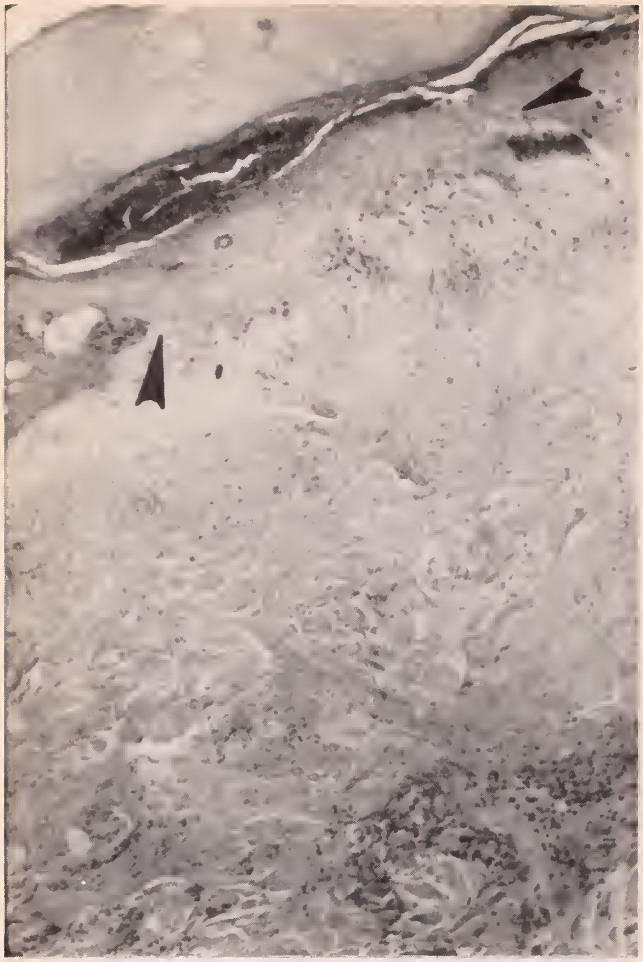

FIGURE 2. Biopsy of skin lesion on right palm, showing a superficial and deep dermal infiltrate, with ischemic necrosis of the epidermis in area delimited by arrowheads (hematoxylin phloxin saffron stain; original magnification × 65).

sels in some instances appeared necrotic, with a fibrinous wall, debris, and a large number of inflammatory cells, many of which were eosinophils. Thus, it is evident that we are observing a vasculitis.

In the deeper vessels, there were great numbers of eosinophils. The infiltrate in some areas is vaguely granulomatous, not definitively so but strongly suggestive (Fig 3).

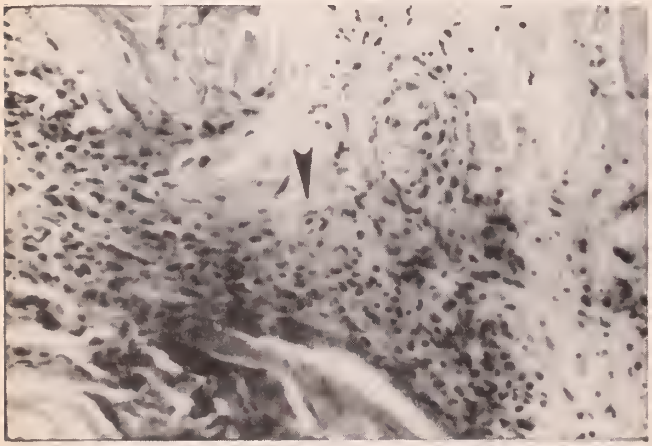

FIGURE 3. Deep dermal vascular plexus, showing infiltrate of eosinophils and epithelioid histiocytes (arrow) (hematoxylin phloxin saffron stain; original magnification × 400).

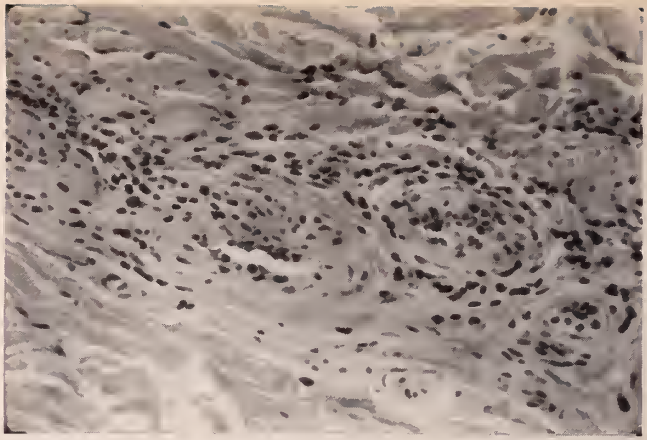

FIGURE 4. Small veins and venules in deep dermal plexus, with eosinophils in the act of margination, and infiltrating the intima and perivascular tissues (hematoxylin phloxin saffron stain; original magnification × 400).

Some of the damaged vessels had thin walls, with many eosinophils migrating into the wall. The veins are infiltrated by eosinophils, involving the intima and portions of the vessel wall (Fig 4). A variety of blood vessels are involved in this process—arterioles, venules, and veins, for example.

Before giving the diagnosis, we should consider the differential diagnosis from the pathologic point of view. This patient obviously did not have polyarteritis nodosa (PAN). Sections of vessels in polyarteritis can show a thrombus in the lumen, with inflammation extending throughout the vessel wall. The term periarteritis is incorrect, since in PAN the inflammation involves not only the adventitia of the vessel, but also the entire thickness of the vessel wall. It is thus a panarteritis.

Polyarteritis is a more rational term, since it includes the important fact that the inflammation involves multiple arteries, albeit segmentally. Eosinophils may be seen in the cellular infiltrate, but also included are lymphocytes and polymorphonuclear leukocytes. However, the important fact in PAN is that the process exclusively involves medium-sized and small arteries. This patient clearly did not show such exclusivity. Also, a healed lesion of PAN shows that internal elastic lamina is partly destroyed, and aneurysms may result.[11]

In Wegener's granulomatosis, there are large necrotic lesions with a granulomatous inflammatory response, multinucleated giant cells, and other mononuclear cells in the infiltrate.[12,13] Here again, one can see the involvement of vessels with necrosis, which is generally very marked. However, the presence of eosinophils is very uncommon. In Wegener's granulomatosis, a necrotizing glomerulonephritis is also observed.

The present case demonstrates a necrotizing vasculitis with very marked eosinophilia, chronic asthma, and involvement of the skin and lungs. The upper airway involvement is not really necrotizing in the sense that the bones and other structures are not involved, except for the lining of the sinuses.

Putting all the facts together, we made a diagnosis of allergic angiitis and granulomatosis—Churg-Strauss disease. The patient was given prednisone, 60 mg/day, with a dramatic remission within three days. Thereafter, the medication has had to be reinitiated from time to time, and the patient has experienced side effects, primarily because of his cardiac disease. But at present, now two years after the diagnosis, he is doing well.

Over the long term, most such patients are able to discontinue steroid therapy, so Churg-Strauss disease is very often a relatively benign condition, compared to the other vasculitides. Wegener's granulomatosis requires treatment with cyclophosphamide, and the ultimate prognosis is much worse. PAN of course has an even dimmer outlook.

Churg-Strauss disease[7] is an idiopathic condition, and I know of no reasonable hypotheses to explain the onset of this particular entity in this patient.

Dr Sukumaran: Churg-Strauss syndrome has no known precipitating factors. Patients do respond well to steroids. Many of these patients go on to develop involvement of various organs, and the outcome can be fatal. The cardiac manifestations in this particular patient were confined to low voltage in the electrocardiogram; pericarditis is described in about 15–20% of patients. Myocarditis may also occur. Since this patient responded well to the treatment, we cannot be sure how many organs were involved in his case. If the patient does not respond to steroid therapy, treatment with azathioprine (Imuran™) and other chemotherapeutic agents has been tried with some positive response.

Dr Sidhu: There seems to be no particular correlation of this condition with any immunofluorescent studies. That is true for all of the necrotizing vasculitides.

Dr Sukumaran: Studies of such factors have been limited, since Churg-Strauss syndrome is not a very common disease. The original cases at Mt Sinai Medical Center were not studied with immunofluorescent techniques. The study from the Mayo Clinic included measurement of serum IgE, suggesting an allergic response not directly causing the syndrome. There are no immune studies described, as far as I know.

Lester Blair, MD (Director of Pulmonary/Critical Care Medicine): What do you believe the nodules in the lung represent—a vasculitis or some other type of lesion?

Dr Sidhu: They could very well be evidence of a vasculitis with associated pneumonic reaction or infarction. In this case, the nodules cleared up as a result of the treatment.

Dr Sukumaran: The pulmonary lesions in Wegener's granulomatosis also may show a fairly good response, not to steroids, but to the chemotherapeutic agents.

Dr Sidhu: The vessels involved in Churg-Strauss disease are smaller; and the necrosis in the lung is never as severe as that seen in the lung in Wegener's granulomatosis. The infiltrates in the lung can shift from one part of the lung to another, which implies that they can resolve spontaneously. It is hard to say whether one is seeing pneumonic or infarctive lesions, since we have so little opportunity for pathologic examination of the lung.

Dr Sukumaran: The infiltrates in the lungs are similar to those seen in Löffler's syndrome. They are migratory in nature, not necessarily related to lesions of the vessels. The nodules are probably due to vasculitis with infarction.

Dr Sidhu: A good deal of the necrosis may be due to the eosinophils. When you find a very dense infiltrate of eosinophils, you must remember that the granules within the eosinophils are fairly toxic both to human tissue and to parasites. Any tissue that has a severe infiltration of eosinophils will be subject to necrosis. A great deal of necrosis could be due to just the eosinophils without ischemia. That would explain why the condition may spontaneously resolve and move from one area to another, and does not imply necessarily a necrotizing vasculitis, though the latter will be found in some locations in the body, since Churg-Strauss syndrome is classified as a necrotizing vasculitis. The liver, in response to Dr Brau's question, is usually not a site of such necrosis; while the skin, lungs, and neurologic tissues may be.

FINAL DIAGNOSES

Allergic angiitis and granulomatosis of Churg-Strauss syndrome, with involvement of upper and lower respiratory tract, and of skin.

Chief-cell adenoma of parathyroid; status, post-surgical resection.

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SPECIAL ARTICLE

Passover matzoh and chocolate Easter eggs

PASCAL JAMES IMPERATO, MD

When I was growing up in the Tudor Village area of Ozone Park, New York, few families owned automobiles. World War II had halted automobile production, and gasoline rationing rendered many existing cars idle. Unfortunately, the transmission of our 1935 Chevrolet gave out as the war began. Although my father took the car to several mechanics, all of them pronounced it beyond repair. The last of these was not only unable to repair the transmission, but also gave the poor Chevy a death blow by ruining the engine pistons. My mother never ceased muttering maledictions, even years later, whenever she walked past this mechanic's garage that stood under the Boyd Avenue station of the BMT Fulton Street el. This el and the Green Bus Lines became our major means of moving beyond the neighborhood once our car had been towed off to a final resting place on a friend's nearby farm. It stood there on blocks for several years, my father ever hopeful that new pistons and a functioning transmission might be found.

Ozone Park was well served by small shops, delicatessens, and groceries, but the nearest Bohack and A&P were a mile from where we lived. Weekly food shopping was a major chore for which young children had to be recruited, usually with much cajoling, threats, entreaties, and promises of reward. Extra pairs of arms were always needed to carry cloth shopping bags filled with fruits, vegetables, meats, fish, and staples.

Fortunately for us children, milk and bread were delivered to the doorstep by familiar trucks driven by men who faithfully showed up year after year, some for two decades. Renkens, Sheffield Farms, and Maplewood Farms were motorized, but the Borden's milk truck was pulled by a horse which knew every stop on the route. Krug's and Dugan's delivered bread in the morning from their rectangular-shaped trucks, attaching the loaves to the doorknobs with string. In the afternoons, they announced their arrival with distinct horn sounds which brought housewives to the front doors. Soda and bottled water were also home delivered, and a stream of peddlers hawked vegetables, fruits, and fish. Their prices were much higher than those in the stores, and many families, including mine, were unwilling to pay for the convenience, except on occasion. We children were grateful for the milk and bread men and the peddlers, since they made our lives easier. But perhaps our greatest thanks went to Sears Roebuck, whose telephone directory-sized catalogues featured everything from jewelry to live poultry. Operator 19 at Sears and my mother became good friends over the years. I recall that only a small fraction of their telephone encounters were taken up placing the orders. Their conversations covered family, friends, politics, and the war. Even my latest school report card didn't escape comment and was also shared with the United Parcel Service delivery man who carried in the Sears Roebuck orders a few weeks later. He and the Fuller Brush Man regularly drank coffee in our kitchen, encouraged us to work harder in school, and witnessed my transformation from a fourth grader to a college senior.

The locus of grocery shopping was often influenced by sales advertised in the local newspapers. As far as I was concerned, it was all the same since the Bohack was a mile west in City Line, and the A&P a mile east on Cross Bay Boulevard. I had carefully verified the distances on my bicycle speedometer several times and irritated my mother no end by challenging her claim that the Bohack was closer. Her claims of this store's proximity were really a cover, I thought, knowing that she preferred going there because of its being in a shopping area. City Line, located on the Brooklyn side of the Brooklyn-Queens line beneath the el on Liberty Avenue, had a wide variety of stores. It had grown up in the early part of the century around the then Grant Avenue terminus of the el. However, as big as the variety was, it wasn't sufficient to satisfy my mother's needs. Regular trips were made to Jamaica via the Q8 bus of the Green Bus Line for purchases of shoes and clothing. And on occasion we took the el to Borough Hall in Brooklyn to shop at Abraham & Straus and Loesers. All of these expeditions were full-day affairs since it was a mile to the el and slightly more to the bus stop, after which the rides took close to an hour. As tedious as all these trips were for a young boy, by far the worst ones were to the produce pushcart market on Blake Avenue in the New Lots section of Brooklyn. The variety of produce there and the reasonable prices eroded my mother's usual restraint. She invariably purchased far more than I thought we could humanly carry, a problem she unhesitatingly resolved by asking for porter assistance to and from the bus and el stops from willing passersby.

As much as I disliked the round trips to Blake Avenue, I thoroughly enjoyed myself while there. It was an exciting and alien world to me where much of life was lived on the street. Canopied pushcarts loaded with produce lined one side of the avenue stretching for a few blocks east and west of the intersection of Cleveland Street. The buildings

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along the avenue housed kosher butcher shops, bakeries, dry goods stores, and novelty shops whose wares spilled out onto tables set up on the sidewalk. At the heart of the market stood the Cleveland Fish Market, on the southeast corner of Blake and Cleveland. Live fish swam in tile-lined tanks at the fish market and the ice covered counters displayed an unbelievable variety including cod, mackerel, and carp. I used to stand on the store's tall round stoop and look up and down the avenue. From there I could see hawkers pacing the sidewalk in front of their stores, enticing customers to come in, while from the curbside the produce peddlers chanted their low prices and the quality of their goods. There was an operatic quality to some of their chants which rose to high decibels in Yiddish and accented English. Their aim was to attract a potential customer's attention through sound, but they didn't let their fortunes ride on their chants alone. They would call out to people walking by, compliment women on their dresses or coats, extol the beauty of a child, and in so doing produce guilt if a purchase weren't made.

The produce peddlers who dominated the life of the market either stood high up on wooden crates behind their pushcarts or else worked the crowd from the sidewalk in front. In fierce competition with one another, their aim was to build up customer loyalty. When quality was lacking, they accommodated on price and threw in extras in the form of a few free apples or plums. Posted prices meant little since they would come down with intense bargaining, the purchase of quantity, or for a good customer, or else go up for someone the peddler didn't like. The prices were written in bold black letters on brown paper bags that were placed upside down on wooden crate slats vertically inserted behind the piles of fruits and vegetables. Like the stock market, prices fluctuated during the course of the day, and so it was not unusual that a few layers of bags covered each slat. The fluctuations recorded on these bags were relatively small, and it was rare that a peddler set off a sell-off panic by dramatically lowering his prices. Success lay in maintaining price equity with competitors and in giving bargains to customers who then spread the word to other buyers in the market.

Few people who lived in our neighborhood knew about the Blake Avenue market, and scarcely any of them shopped there except my mother and Mrs Rose Gruber. Henry and Rose Gruber, our only Jewish neighbors, had lived in East New York near New Lots and were familiar with the pushcart markets in the area. My mother, who had grown up in Ohio, lived in New Lots for a year before she married. The lure of this market was great for both of them, and only distance and the absence of a car limited them to several excursions a year. One year when I was around eight years old, Passover and Easter fell close together, and Mrs Gruber and my mother decided to go to the Blake Avenue market together for holiday shopping. As an eight-year-old, I was able to protest conscription for this shopping expedition but unable to alter my mother's firm decision that I comply. I set off in a sulking mood, trying to hasten them along toward the el by walking at a fast pace. This strategy didn't work since they had allowed the entire day for this excursion, enjoyed stopping to emphasize points raised in their conversation, and seemed to go out of their way to chat with neighbors along the way. The ride west along the BMT Fulton Street el lifted my spirits, especially as the weather was warm enough for me to look out the open window. Beyond Hudson Street where we got on, the el structure was narrower and more rickety, having been built in the 1880s. The stations here were constructed of wood as were the waiting rooms with their potbellied coal stoves in the center. Whenever the trains stopped or started up, the stations swayed along the direction of the tracks. Beyond the Crescent Street station, the el made a sharp turn south down Euclid Avenue. As the train rolled over the curve it slowed, pitched toward the inner side of the curve, and made a tremendous screeching sound. Below, the Sunrise Highway stretched east into a horizon of farmland and small clusters of houses. Two blocks beyond, the el curved again to resume a westerly course along Pitkin Avenue. Again, the train tilted over and screeched as it completed the curve and rolled into the Chestnut Street station. It was only on these curves that my mother and Mrs Gruber suspended their conversation. My mother grabbed on to me as I stuck my head out of the window trying to peer down at the street below. She told me that el trains had been known to fall to the street on sharp curves like these, and Mrs Gruber added that she knew of a boy who had fallen out of an el train window on a curve. I didn't believe any of it, of course, and even fancied what fun it might be to be caught in a dangling el train.

We got off the el at Linwood Street and walked three blocks south to the Blake Avenue market. Mrs Gruber, who had a bunion on her right foot, decided to stop in at a podiatrist's office on the way. Although she went to a neighborhood podiatrist, she was a firm believer in second opinions long before they became a formal part of medical care. In fact, her philosophy of medical care was one of discontinuity based on the belief that reliance on one physician was unwise. Better to consult several was her constant advice, and visit chiropractors, podiatrists, and herb doctors as well. She and my mother asked some women sitting on a stoop what they thought of the podiatrist whose ground floor office was across the street. They said he was a "shoemaker," which in those years was a kiss of death recommendation given to doctors, dentists, and others thought to be incompetent. My mother and Mrs Gruber listened attentively to these women who shredded this poor podiatrist's reputation with an eagerness and rationale that must have struck them as suspect. Thanking the women, they headed for the podiatrist's office, much to my amazement. He was a young man with an empty office and a new practice. While the podiatrist tended to Mrs Gruber's foot, she deftly uncovered that one of the stoop women had an unattractive daughter whose hopes of marriage had been rebuffed. It seemed that the mother and her cronies took advantage of good weather and the stoop to inflict revenge.

The market was more bustling than I had remembered it, crowded with holiday shoppers from the surrounding neighborhoods. Most were Jewish, descendants of 19th century Eastern European immigrants who worked in the needle trades. However, there were also many Italians, who primarily lived north of Pitkin Avenue but who were within easy walking distance of the market. As was their habit, Mrs Gruber and my mother first walked the five-block length of the produce pushcarts to assess quality and prices, making mental notes as they went. They always left the produce purchases to last because of their weight. We first headed for the kosher bakery where pumpernickel bread was the specialty. Odd pieces of bread were laid out on a tray on the counter for customers to taste. In fact, test tasting potential food purchases was the norm, negating the need for carrying lunch along or eating in restaurants which didn't exist anyway. Since it was close to noon, my mother gave me a piece of pumpernickel bread to eat from the loaf she had purchased. Next door at a grocery store I was given matzoh as Mrs Gruber negotiated their price. This grocery was well stocked for Passover with raisins, nuts, horseradish, noodles, matzoh, and bottled gefilte fish, all of which found their way into our shopping bags. Our next stop was Greenberg's dry goods store, where Mrs Greenberg always referred to her husband as Mr Greenberg, even when addressing him in person. As soon as we walked in, I knew that something was very wrong because the shelves and countertops that were usually smothered with high piles of goods were bare. Mrs Greenberg told us that Mr Greenberg had been taken to Kings County Hospital. My mother and Mrs Gruber reacted with shock, and I was surprised when Mrs Gruber then asked when he was buried. I didn't understand as these adults did that an unelaborated declaration that someone had gone to the hospital also meant that they had gone there and died. It wasn't merely a euphemism they employed, but an empirically determined figure of speech. Hospitals were still places where people went to die, and as such they were both feared and held in low esteem. Mrs Greenberg described her husband's medical history in detail. He had developed pain in his chest and the doctor had diagnosed a heart attack. He was taken to Kings County Hospital, said Mrs Greenberg, as my mother and Mrs Gruber shook their heads, indicating that he was thereby doomed.

Mrs Gruber held to a dietary cause of heart disease. This was remarkable for a lay person in the late 1940s, well before convincing experimental and epidemiologic data on the subject had been accumulated. Although Mrs Gruber kept a kosher kitchen, she also imposed dietary restrictions and exclusions based on her intuitively derived knowledge. She vividly told Mrs Greenberg and my mother how she had first learned about the relationship between diet and health. She attended a lecture in Manhattan given by an herb doctor. He dropped various foods together into pails. On some occasions, fumes rose from the pails, probably from dry ice slipped in along the way, I suspect. However, this enthralled and convinced his audience and won over converts to his low-fat diet program, including Mrs Gruber.

I wasn't surprised when Mrs Gruber probed Mrs Greenberg for her husband's dietary history. As young as I was, I realized that he had eaten too much meat, a major culprit as far as Mrs Gruber was concerned. Her conclusion was that a fat-rich diet had killed him, but of course fate was fate, too. My mother was unconvinced, and to me it all sounded like a lot of hocus pocus. Yet, years later I came to appreciate that Mrs Gruber's intuition and empirical observations had validity. With condolences expressed more than several times, we left and went across the street to see Rosie, the fruit and vegetable peddler. She was a short, squat woman whose leathery face and hands had been textured by the sun and wind, and gnarled by time. She stood atop a wooden crate behind her pushcart from where she hawked to passersby and drove hard bargains with those she didn't like. Rosie was argumentative, and at times unreasonable. I remember once how she raised the price of green peppers for a woman by giving back less change. When the woman protested being shortchanged, Rosie simply told her that the displayed price wasn't for her. Rosie lost in the end because the woman grabbed an extra pepper and ran off into the crowd.

Rosie's reputation in the market rode not so much on the quality of her produce, which was good, but on her being a gossip. As we made our way over to her pushcart, it struck me that my mother and Mrs Gruber were more interested in what she knew about Mr Greenberg's death than in her apples and oranges. Between haggling with customers, Rosie spewed out her disdain for dietary explanations and a heart attack diagnosis. It was aggravation that had killed Mr Greenberg, and his wife was the source of it as only a few knew, including her. According to Rosie, she never gave Mr Greenberg a moment's peace, had grand ideas about moving to Long Island and buying a car, and thought herself superior to everyone. Now that he was dead, she had sold the business, and worse still, had cashed in a life insurance policy with which she was going to buy a house in Valley Stream. It was no secret, according to Rosie, that Mrs Greenberg had even thought about taking out another life insurance policy on Mr Greenberg, but he died before she could.

Rosie's story was clearly at variance with the image of the poor widow we had seen in the dry goods store. My mother and Mrs Gruber were far too experienced to take Rosie at her word, and so their quest for both bargains and medical truth propelled them and me up the line of produce pushcarts. Hymie, the banana man, plied us with free over-ripe stock as he dismissed everything as malicious gossip. Greenberg had obviously died from overwork. Why he, Hymie, was in perfect health, despite a nagging wife and eating everything Mrs Gruber said he should avoid. Mrs Gruber would never concede rhetorical defeat, and wagging her finger at Hymie, warned him that his diet would eventually do him in. A few years later, her remorse was tinted with satisfaction as she told my mother that Hymie had died of a heart attack.

By early afternoon, we were midway along the line of pushcarts. Each of us had two shopping bags apiece, mine being stuffed with an ecumenical assortment of groceries and vegetables for both Passover and Easter. By this time, I was both bored and tired, and began to complain. My mother's first line of response to this was always a disciplinary one, verbal in public, physical at home. When this failed, which it invariably did under my incessant badgering, she resorted to promises of reward, in this case, chocolate Easter eggs. Her goal was to hold off the reward as long as possible, mine to have it at once so as to use nagging to get more or get us on our way home.

We finally arrived on the block where a few stores sold wonderful assortments of kosher chocolates and chocolate Easter eggs. The eggs came in all sizes, and some were decorated with pink and green sugar designs. Loose chocolate was sold in these stores and filled them with a wonderful aroma only second best to taste. I carefully watched my mother's purchase. We were six children at home, and I wanted to see what I could look forward to on Easter Sunday. Although I no longer believed in the Easter Bunny, I still found a basket full of chocolates and candy next to my bed on Easter Sunday morning. As we left the store, my mother reached into the bag and handed me one of the smallest chocolate eggs she had purchased. Trudging behind her and Mrs Gruber, I devoured it while plotting how to get another egg, or even more. I wasn't exactly starving, since Rosie had given me an apple, Hymie bananas, and Mrs Gruber matzoh. But here was an opportunity not to be missed. To be so close to these chocolate Easter eggs and not to be able to eat them was pure torture. I reasoned that I deserved more of them than my siblings since I was serving as the pack animal for this trip. Surreptitious theft certainly crossed my mind, but that carried great risk if I were caught. Anyway, my mother and Mrs Gruber had wisely put the kosher chocolates and the chocolate eggs out of my reach in their own shopping bags.

Our next-to-last stop was Benjamin's, whose low one-story tailor shop stood at the eastern end of the market. There was no sign over the door or on the dingy plate glass window. Yet everyone knew Benjamin and where his store was. He always stood at the rear of the store, his stooping figure silhouetted against a mosaic of frosted glass windows that covered the back wall. Benjamin always wore a black fedora and peered out from behind his workbench through bifocals that rested on the tip of his nose. He was probably only in his sixties then, but to an eight-year-old he looked ancient. The entire store seemed from the long ago with its sculpted metal ceiling, creaking floor boards, and brown patinated counter tops. Yet, it was functional and inviting, and a place where the problems of life could be discussed with someone whose years and wisdom provided insight and solace.

The tragedy of Benjamin's life, according to my mother and Mrs Gruber, was that he had five daughters and no sons. To have so many daughters and no sons was bad enough. But for Benjamin it was compounded by the fact that he was a tailor who only made men's pants. What a shame it was, my mother would say, that poor Benjamin couldn't use his talents to make clothes for his own children.

The counter tops on either side of the store's only aisle were stacked high with dry goods. It was miraculous to see Benjamin measure someone, cut the cloth from a bolt from the counter, and in a short time produce a pair of pants with pleats, cuffs, and pockets. Two old men worked with him hunched over their sewing machines. After Benjamin had cut the cloth, the two old men would sew it into rough form. Benjamin would examine their work against the light of the frosted glass panes, suggest changes, and during a fitting mark up the pants with white soap. Then he would rip and tear them, re-sew, and finally press them with one of the great irons that rested on his work table. They were heavy irons connected with long cords that snaked and looped their way over supporting wires to ceiling plugs.

We sat down on the chairs near Benjamin's workbench, propping our shopping bags against the counter. This time my mother spared me the ordeal of a fitting. I always found it an ordeal because the dressing room was the space behind one of the counters where I would stand in my underwear while the pants were adjusted. Peering out between the heaps of dry goods, I was very self-conscious of the stream of customers and visitors who always seemed to notice me. Benjamin had told my mother a short time before that my left leg was a half-inch shorter than my right one. As a meticulous tailor, he always measured both legs, not just one as many others did. He would make me walk up and down the aisle, scrutinizing the movement of the pleats and cuffs, and adjusting them until the pants were a perfect fit. He thought that my short leg might eventually give me back problems and suggested to my mother that she take me to a bone doctor, and have a wedge put in the heel of my shoe. Over the years, I have thought of Benjamin's unheeded advice, especially when radicular pain radiates down my left leg.

Our visit with Benjamin was purely social, and inevitably the conversation turned to Mr Greenberg's death. The two old men and Benjamin listened silently as Mrs Gruber poured out all the versions we had heard up and down the line of pushcarts. When pressed for an opinion, Benjamin didn't rush to give one. He calmly said that the stories the peddlers told were like the produce they sold, some being of good quality, others being very poor. He evaded specific judgment of anyone, and showed great wisdom in effortlessly shifting the conversation in another direction.

Rest and a social visit with Benjamin gave Mrs Gruber and my mother the renewed energy for their last stop at the Cleveland Fish market. They had decided to return home on the Green Bus Lines' bus that started at the New Lots station of the IRT el and ran to Howard Beach along Linden Boulevard. Getting to the bus stop with all our purchases posed a problem, which Benjamin solved by having the boy who lived next door meet us at the fish market and help us out.

The bus was crowded but we managed to get seats near the rear door knowing we would be the first to get off. The bus rolled out east on Linden Boulevard, into a landscape that progressively became more rural. It lurched from side to side with the undulations of the road and only a firm grasp on the shopping bags prevented the produce from rolling out and down the aisle. Eventually we crossed the county line into Queens where a cool breeze blew in over the marshlands and farms that dotted the horizon to the south. There were no buildings along this stretch of the road by which to gauge our exact location. Only the lonely farmhouse off to the right told us we were approaching Cedar Lane where we had to get off. My mother pulled the cord and the buzzer sent the bus into a lower gear. We slowed and jolted a few times and finally came to a dead stop in front of a clump of tall trees that marked the entrance to Cedar Lane. Passengers helped us off the bus and handed down some of the shopping bags through the rear door.

The problem now was to get all the shopping bags home. Mrs Gruber and my mother always had contingency plans for this leg of the journey. There was a nursery stand at the junction of Cedar Lane and Linden Boulevard where excess shopping could be stored and later retrieved. To facilitate the cooperation of the owner, my mother decided to buy an Easter lily. As she was discussing the price, I saw a woman walking towards us up Cedar Lane. From her dress, I knew that it was one of the Dolan sisters who lived on their family farm a short distance away. The three Dolan sisters were eccentric, and dressed in the mode of the 1920's. Their farm had been idle for years, and a large faded sign painted on the gray clapboard of the house, reading, Farm for Sale, was a poignant reminder of their plight. They rented their barns to horse owners, and this plus other meager sources of income helped them eke out a living.

Further on down Cedar Lane and beyond its westerly bend, Herman Brockmann and his family successfully farmed over fifty acres, and Mr Van Syse's cows produced Cedar Lane milk which was sold at the market. However, the three spinster Dolan sisters didn't have the skills to farm their land which had yet to become valuable for development. They were religious women, and two of them attended church every day. Estelle Dolan, who slowly made her way up the hill, was heavily made up with rouge and lipstick, and wore a black hat with a black "chicken wire" veil. Although she represented salvation since she could help us carry the extra shopping bags home, I feared that she would delay us since my mother and she hadn't spoken in a long time. But fortunately, Estelle was on her way to church for a Holy Week service and couldn't spend too much time talking. The extra pair of arms obviated our need for the nursery stall and for buying the quid pro quo potted lily. I breathed a sigh of relief since I had already been fingered to carry the lily, a fate worse than death for an eight-year-old boy, especially if he met his friends.

My mother stood firm in her refusal to give me another chocolate Easter egg despite my badgering. However, after we crossed the nearby Sunrise Highway, Mrs Gruber paused, and while motioning to me to stop, told my mother and Estelle Dolan to go on ahead while she adjusted the contents of her bags. With my mother several paces in front, she dug down into one of her shopping bags and surreptitiously produced two kosher chocolates. She sealed our conspiracy with a wink and a smile and whispered that I should dawdle behind, hoping that my mother wouldn't notice my moving jaws. As we entered Tudor Village, the conversation turned to health, Mr Greenberg and diets. Estelle Dolan's view that a healthy diet consisted of meat, eggs and dairy products was no doubt shaped by her life on the farm. However, it did not go over well with Mrs Gruber. But that is another story.

Henry Keller, MD, of New York and the Alyn Hospital in Jerusalem

MYER MAKIN, MD, SIMON T. WINTER, MD†

Although early hospitals were mostly of ecclesiastic origin, many later hospitals owe their founding to the impetus and devotion of individual public-minded physicians.[1] Among the latter is the Hospital for Sick Children in Great Ormond Street, founded in London in 1852, which resulted from the labors of Dr Charles West.[2] Similarly, the renowned orthopedic hospital at Oswestry in England was opened in 1900 through the efforts of the pioneer orthopedic surgeon, Sir Robert Jones, and his eminent head nurse, Dame Agnes Hunt, after both of whom the hospital is now named.[2]

Henry Keller, MD, was one of the first orthopedic surgeons in New York City. He contributed significantly to his specialty in both academic and clinical fields. Outside of medicine, his interests were numerous. In 1932, he was instrumental in the establishment of services for crippled children in Jerusalem, Israel. In 1936, when there was not one orthopedic department in the whole of Palestine, Keller was the guiding force behind the opening of the Alyn Hospital, the first children's orthopedic hospital in the country.[3,4]

THE SETTING

The historical pull of their original homeland on the Jewish people is well known. Ever since their dispersion from the Holy Land, an intermittent stream of Jews had returned there, some to live and some to die. After the creation of the Zionist movement at the end of the 19th century, the return flow of Jews to Palestine slowly began to increase.

When the first World War broke out in 1914, Palestine had been in the hands of the Turks for nearly four centuries, and poverty, illness, and malnutrition were prevalent. During the war, conditions so deteriorated that the population slowly decreased by half. The common illnesses of neglect, typhus, typhoid, and cholera were frequent, and trachoma was prevalent. There was little to hold the Jewish population in Jerusalem, so that by 1916 there remained only half of the original population of 50,000.

After the outbreak of war, communications became more difficult but were still possible between Turkey and the United States, which remained neutral until 1917. In 1915, medicines and food were sent by merchant ship from the US to the Jewish population of Jerusalem, but the future still looked grim.

In November 1917, however, the British government issued the Balfour Declaration. This gave hope of early resettlement by Jews in Palestine, a hope reinforced by the capture of Jerusalem by the British general Edmund Allenby a month later. This was the background to the entrance of Dr Henry Keller on to the scene in 1918.

EARLY LIFE AND EDUCATION

Henry Keller was born in 1877 in Cracow, then in Austro-Hungary but now in Poland. In 1893, at the age of 16 years, after a brief sojourn in London, he moved with his parents to New York.[5] There he attended a theological seminary and then Senftner's Preparatory School[5,6] before enrolling, in 1898, at the School of Medicine of New York University, then known as the University and Bellevue Hospital Medical College.[6,7] In 1903, he graduated with the degree of MD and received a license to practice medicine in New York State.

Dr Keller's postgraduate training included posts in orthopedic surgery at the Roosevelt Hospital at the New York Post-Graduate Medical School in 1909–1910, and then at the New York Polyclinic Hospital.[8] Further orthopedic experience was gathered by visits to London, Berlin, and Vienna.[8] On his return he opened a private office on West 110th Street in New York, and held posts as attending surgeon at the New York Polyclinic Hospital, the University and Bellevue Dispensary, Roosevelt Hospital, and Harlem Dispensary.[6]

FIRST TRIP TO PALESTINE

In June 1916, during the first World War, but ten months before the United States declared war on Germany, an appeal from the Zionist Head Office in Copenhagen reached the Women's Zionist Organization of America, asking for medical aid to be sent urgently to Palestine.[9] There, we know, the population was suffering from a critical and deteriorating breakdown of the already meager medical services.[10] While epidemics of acute infectious disease prevailed and malnutrition was rife, hospitals had either closed or reduced their services. Most of the Jewish doctors had been trained in Czarist Russia. The majority of the variegated group of local physicians were either recruited into the Ottoman Army or expelled as enemy

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FIGURE 1. Dr Henry Keller.

aliens. An American Zionist Medical Unit (AZMU) was therefore planned in order to alleviate this situation.

Keller volunteered and was appointed orthopedic surgeon to this unit; no other surgeons joined. The staff for the unit was quickly organized, but wartime formalities and restrictions on travel led to considerable delay in its departure. After America entered the war, in April 1917, and with the sailing of massive American military contingents to Europe, permission was granted for the unit to go to Palestine. In 1918, the AZMU, with equipment and stores, sailed from New York in a naval convoy carrying 30,000 American troops to Britain.[9] After arrival there they had to travel over land via France and Italy to reach Alexandria in Egypt. They arrived in Palestine on August 18, 1918, where the AZMU unit was brought under British Army command.[11]

The AZMU was a strictly professional team of 44 members. There were 11 specialist doctors, 20 nurses, a dentist, a pharmacist, sanitary engineers, and laboratory staff. It soon became fully active. Allenby's offensive to capture the north was underway, and at the clearing station set up in Tel Aviv, casualties were treated by the AZMU staff. Keller also conducted regular orthopedic clinics in Jaffa.[12]

After hostilities ended in November 1918, the AZMU team was kept busy dealing with civilian diseases. There were acute outbreaks of cholera, typhoid, typhus, plague, and relapsing fever, together with the endemic problems of tuberculosis, malaria, trachoma, and severe malnutrition. The abandoned buildings of the Rothschild Hospital in Jerusalem were turned over to the AZMU and were rapidly put back into service.[13] Smaller units became active, dealing with epidemics in Safed, Tiberias, and Jaffa, with clinics and small local hospitals being set up in these towns. These modest beginnings, including the opening of a nursing school in Jerusalem, were the precursors of the Hadassah Medical Organization, with its later internationally recognized network of clinical, preventive, teaching, and research institutions.[14]

RETURN TO NEW YORK

Keller served with the AZMU until 1919 and then returned to New York. He was appointed to the staff of the Neurological and Community Hospital, the West Side Hospital, the Rehabilitation Institute and General Hospitals, and a number of institutions for elderly citizens.[3,7,8] However, his zeal and his sense of personal involvement in Palestine had been kindled by his war service and remained deep within him. He never forgot those experiences that contributed to his resolve ultimately to settle there.

Keller was active in academic fields, as well as being a prolific writer and a member of the American Medical Editors and Writers' Association. Academic recognition was accorded by the New York University School of Medicine with his appointment as Instructor and, from 1929, Clinical Professor of Orthopedic Surgery.[8] Acknowledged as an effective lecturer, he was a member of both the American and British Associations for the Advancement of Science.[7]

More than 40 publications demonstrate the breadth of his involvement both in orthopedics and in other aspects of disease. His basic investigations related to epiphyseal ossification and the influence of climate on this process.[15,16] His clinical observations were exemplified by publications on the complications of tabes dorsalis[17] and typhoid fever,[18] injuries of the acetabulum,[19] skeletal deformities related to the muscular system,[20] primary vertebral involvement in adolescent rickets,[21] and mobility of the human spine.[22] At the time of their publication, his papers on the psychological aspects of orthopedics were innovative; he stressed the need to evaluate the patient's mental state as well as the physical.[23-25]

FIGURE 2. The American Medical Zionist Unit in Palestine in 1918. Dr Keller, left, back row.

His articles also dealt with techniques in orthopedic diagnosis and treatment. Keller addressed such subjects as arthrography,[26,27] a device for overcoming knee contractures,[28] and laboratory tests in surgical tuberculosis.[29,30]

Keller also concerned himself with organizational matters such as contract practice,[31] the medical aspects of a five-day work week,[32] and pay night hospital clinics in New York, which were then being contemplated.[7]

COMMITMENT TO JUDAISM

The involvement of Henry Keller was far-reaching, as he was clearly a ready "joiner."[6,7] He was a member of many medical societies, including the Medical Society of the State of New York, the New York County Medical Society, the Medical Association of New York, the Eastern Medical Society, Harlem and New York Physicians' Association, Yorkville Medical Association, Physicians Square Club of Greater New York, New York Academy of Medicine, and the Riverside Medical Society.

As an orthodox Jew, Keller was active in the religious Mizrachi Zionist Movement. He was a founder and first president of both the Jewish Academy of Arts and Sciences and the Hebrew Speaking Medical Society.

History was clearly a major field for his scholarly mind, and Keller was a member of the History of Science Society. This may have accounted for his frequent quotation of the Old Testament phrase (Ecclesiastes i,), "there is no new thing under the sun." He studied medieval personalities including Maimonides ("Rambam"), 1135–1204, the medical and religious author and court physician to Saladin in Cairo, for whom, in 1198, Maimonides composed a "Guide to Good Health"; and Judah Halevi (1805–1140), the philosopher-poet and physician in Toledo, Spain, apparently in the services of the king and his nobles. These studies led him to publish biographical essays on these historical personalities.[33]

In Boston, in 1931, Keller published *Modern Hebrew Orthopedic Terminology and Jewish Medical Essays*.[33] This book was published during a period of rapid expansion of the Hebrew language. Over thousands of years Hebrew had adapted and changed, yet many aspects of modern scientific experience and development could not be adequately covered without verbal innovations. The acceptance or rejection of such new words had been put on a systematic basis through the pioneering efforts of Eliezer Ben Yehuda and the National Language Academy. Keller's book proposed original Hebrew nomenclature to meet the needs of orthopedic surgery. His proposals were based on Biblical and Talmudic sources and met ready acceptance.

SETTLING IN JERUSALEM

Henry Keller, as a consultant to the Hadassah Hospital, visited Jerusalem on several occasions after World War I.[3] He treated the sick and undertook clinical investigations.[33] During these visits he noted with concern the number of children with inadequately treated crippling conditions in all sectors of the population—Jews, Moslems, and Christians. Cases of skeletal tuberculosis were common, and specific treatment was then not available.[34]

Malnutrition was possibly the basis of the high incidence of osteomyelitis, a major problem prior to the introduction of chemotherapy. The incidence of poliomyelitis was high, as it was in the surrounding Arab countries. Congenital malformations received little attention.

Keller made his home in Jerusalem in 1932. Nevertheless, he continued to spend part of each year in New York, where he maintained his private orthopedic practice. In the downtown area of Jerusalem, he opened a private orthopedic office and also a clinic for handicapped children. This "mini-clinic" expanded rapidly. Poor children were charged no fee, and he himself paid out of his own pocket for medical appliances and drugs for many children. He rapidly became well known in Jerusalem because of his experience and achievements.

It became clear to Keller that the total problem of the care of the crippled child required a communal approach. He was concerned by the lack of facilities for such children and dedicated his skills to providing the children with both medical care and education. None of the Jerusalem hospitals provided long term facilities for children with musculoskeletal diseases. In the western world, the 1930s was a period of rapid and extensive progress in orthopedics, but these new techniques could not be introduced in Palestine because no hospital had suitable facilities. Care within the family was usually quite inadequate because of poor living conditions.

The possibility of opening an orthopedic department in one of the country's hospitals was denied him, so in 1935 Keller took the initiative to found the Society for Crippled Children of Palestine, its Hebrew title being "Agudat Lemaan Yeladim Nehim," the initials of which made up the name ALYN. From its initiation, this society was nonsectarian and nonprofit-making. Early members included the High Commissioner for Palestine, Sir Arthur Wauchope; Mustafa Bey Khalid, the Arab mayor of Jerusalem; Daniel Auster, the Jewish deputy mayor; British Mandatory officials; Moslem physicians; Christian church leaders; the US consul general; the Vester family from the Jerusalem American Colony; and many active volunteers from the growing Jewish community of Jerusalem.[35] An extra apartment on the roof of the building where Keller practiced and lived was adapted with facilities for the hospitalization of 12 children, and rapidly expanded to provide for 18 children in the same modest surroundings. Keller paid the rent and the electricity and provided his medical services free. The needs of the country eventually led to the opening of outpatient clinics in Tel Aviv and Haifa.

Half of the children hospitalized in Jerusalem were Arabs, including many from smaller towns and villages. Other patients came from distant Moslem countries, including Syria, Trans-Jordan, Libya, Egypt, and Persia.

Income for the society was provided through donations and the proceeds of social functions, including garden parties, dances, and concerts.[35] On the inception of the society, the High Commissioner Sir Arthur Wauchope was elected patron. The manager of the Barclays Bank in Palestine, Mr A. P. S. Clark, became president and turned out to be an excellent fund raiser among wealthy Arabs and Jews.

Despite severe restrictions of space, growing demands led to the expansion to 38 beds. Clearly the hospital had reached the bursting point. In 1937, Keller was joined by Edgar Heilbronner, an experienced orthopedic surgeon who had recently left Nazi Germany for Palestine. Following his service in the German Army during the first World War, he had gained further orthopedic experience in Munich, Frankfurt, and Stuttgart.[36]

Henry Keller suffered a cerebrovascular accident in 1939. A further stroke in 1942 led to his confinement to a wheelchair, but he continued to direct the hospital. He worked until a few days before his death on November 22, 1944, at the age of 67 years, and was buried in Jerusalem on the Mount of Olives.[3,5]

ALYN'S FURTHER DEVELOPMENT

After Keller's death, Edgar Heilbronner took over the direction of the hospital and continued to develop services for crippled children. An x-ray unit was installed, and school classes and rehabilitation facilities were expanded. Yet, despite the almost intolerable problem of accommodation, the need for further beds constantly increased.

The Society for Crippled Children had some almost puritanical conventions. From the start, it imposed the rule that administrative costs be limited to 5% of income. This, while limiting expense, meant that money could not be invested for any public relations activities that would aid the raising of further money to cover expenses. The Society was always in financial difficulties. Another of the original rules was that children should only be admitted whose physical condition would improve with treatment.

With the outbreak of the War of Independence in 1948, Alyn underwent a crisis. Children who were sufficiently fit were sent to their homes, but there remained a core of Jewish and Arab children who could not be discharged.[35]

During the summer of 1949, an epidemic of poliomyelitis erupted, with 128 new cases during that year; in 1950 there were more than 1,600 new cases, and 917 during 1951. Ninety percent of the patients were under the age of four years. A total of more than 4,000 cases was reported between 1949 and May 1954.[14] Patients were kept in pediatric wards in general hospitals for about three weeks and then discharged home. Because of the large number of survivors with residual paralysis, the need for rehabilitation and orthopedic services, paramedical care, and clinics became tremendous. The epidemic burnt itself out following the exposure of most of the population to the virus and the introduction of the Salk and Sabin vaccines.

As the sole hospital for crippled children in Israel, Alyn was expected to cope. Larger premises were sought as a top priority. In Katamon, on the boundary of Western Jerusalem, was the neglected and vacant Greek Monastery of San Simon, over 300 years old and reputed to stand on the site of the house and tomb of the Elder, Simeon.[37] It was a picturesque building among old cypress trees, pines, and catacombs. The Greek Orthodox Church willingly rented the large rambling building to Alyn for a children's rehabilitation hospital although, in the long term, it was clearly unsuitable. The building comprised small rooms, winding staircases, and narrow corridors. However, it was the only building available, it was a start, and Alyn moved there in 1950.

By 1957, 100 children were living in San Simon for long term residential rehabilitation. Close connections were developed with the academic Orthopedic Department of the Hadassah University Hospital, where major surgery was performed on selected Alyn patients. A brace shop was created jointly between the Alyn and Hadassah hospitals. Continual developments in special education, paramedical services, social work, and psychological units created demands for more work and recreation space. It was realized that the old building at San Simon could no longer adequately serve the children as a hospital, particularly with so many wheelchair cases.

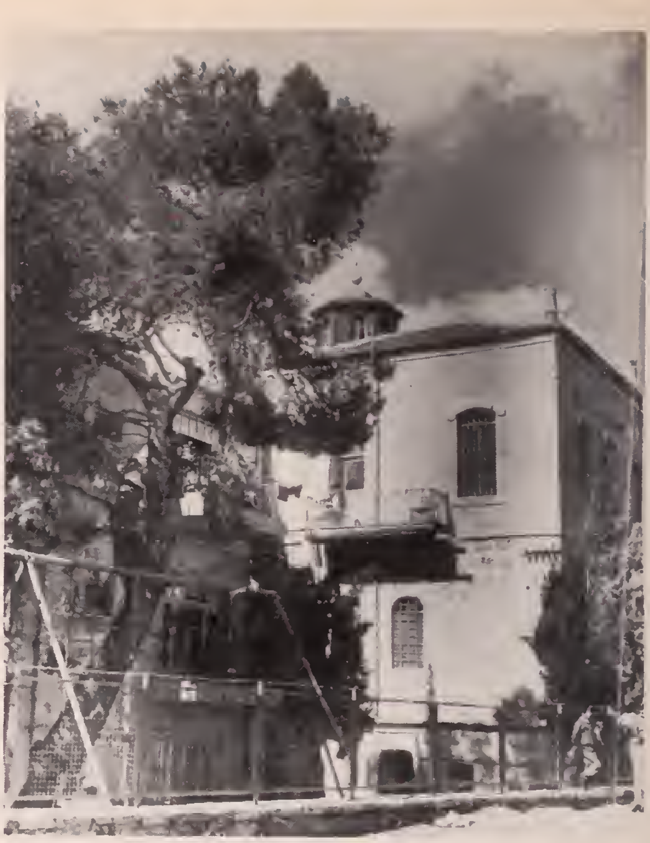

FIGURE 3. The old Alyn Hospital in San Simon, Katamon.

ALYN TODAY—A LIVING MEMORIAL TO HENRY KELLER

A specially built hospital and school were needed, and plans were drawn up. The Jerusalem Municipality donated a plot of about four acres in Kiryat Yovel, not far from the Hadassah Hospital, with a magnificent view overlooking the village of Ein Karem (Luke i:39). A new building was designed based on the principle that all four wards, plus treatment and education facilities, including an indoor pool, were to be on a single floor to enable the easy movement of children on crutches, wheelchairs, or trolleys.

There was a splendid worldwide response to a fundraising campaign, championed by Malcolm Woldenberg of New Orleans, Louisiana, and the new building was opened in 1971. It provided improved care, freedom of movement for the children, and convenience. A variety of outpatient consultation and treatment clinics were opened for children from all over the country. There has been a marked change in the pattern of disabling disease among the children hospitalized at Alyn. The survivors of the po-

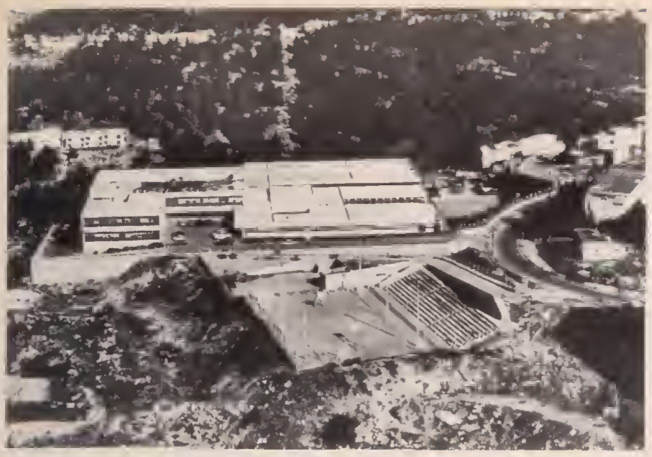

FIGURE 4. The new Alyn Hospital opened in 1971 in Jerusalem.

liomyelitis epidemics of the 1950s left Alyn to return to their homes or to modified special apartments for independent living. In their place, the 93 beds are occupied by children and adolescents up to the age of 18 years with severe musculoskeletal disorders such as osteogenesis imperfecta, arthrogryposis, spina bifida, muscular dystrophies, and spinal muscular atrophy. The unique treatment and residential facilities available are essential for children with such conditions.

Both Arabs and Jews attend the hospital, and at present about 15% of the hospital population is Arab. This presents unique difficulties. Long term hospitalization of young Arab children may lead them to forget Arabic and speak only Hebrew. It becomes easy for an Arab child to turn into a "Jewish" child when he or she forgets his Arabic, speaks Hebrew, and lives in a Jewish environment. After three to six months of hospitalization, such patients may refuse to converse with their parents. Such problems are prevented by continuous Arabic studies and conversation, and frequent visits to family surroundings, even if this is difficult and expensive.

Today, Alyn occupies a position of merit within the medical horizon of Israel. As the only children's orthopedic hospital in the country, it is the best tribute to the memory of Dr Henry Keller, the American surgeon who founded it. Truly the children of Israel have much cause to be grateful for the life and work of Henry Keller.

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Para-endocrine hypercalcemia and ovarian small cell carcinoma

John H. Malfetano, md; Edward Degnan, md; Rudolfo Florentin, md

Small cell carcinoma of the ovary, often associated with hypercalcemia, was described in 1982 by Dickersin et al.[1] Since this original description of 14 cases, an additional 11 have been reported.[2-4] Currently, there is an ongoing review of 80 cases of small cell carcinoma, which one hopes will be reported on in the future.[5]

The majority of patients are in their second or third decade of life and often present with asymptomatic hypercalcemia and a unilateral adnexal mass. This disease is aggressive and usually fatal within 15 months of diagnosis despite therapy. The occurrence of this ovarian neoplasm must be considered when dealing with hypercalcemia that has no other known etiology. This report details the clinical course of the youngest known reported patient with ovarian small cell carcinoma, and the natural history of the disease after surgical removal of the lesion, since no additional therapy was administered.

Case Report

A nine-year-old girl was initially seen by her pediatrician for evaluation of progressive lethargy, malaise, poor appetite, and a 12-pound weight loss spanning two weeks. Laboratory data revealed a calcium level of 15.4 mg/dL, and the patient was admitted to the hospital for diagnosis and treatment.

Physical examination revealed mild generalized abdominal tenderness with no masses initially detected. Results of laboratory studies performed on admission were as follows: calcium, 17.2 mg/dL; sodium 125 mEq/L; potassium, 2.8 mEq/L; creatinine, 1.3 mg/dL; blood urea nitrogen (BUN), 39 mg/dL; uric acid, 12.0 mg/dL; and PO_4, 2.2 mg/dL. Initial therapy with hydration, diuretics, and calcitonin was prescribed.

A renal ultrasound was obtained and showed an incidental finding of a large pelvic mass. The mass measured 11 × 8.5 × 7.5 cm and appeared to be arising from the left ovary. A bone scan was unremarkable, and parathormone levels were within normal limits. A gynecologic oncology consultation was obtained. The patient underwent exploratory laparotomy, and a left 12 × 18-cm encapsulated, ovarian tumor and 100 cc of amber ascitic fluid was noted. A left salpingo-oophorectomy and surgical staging were performed.

The postoperative course was unremarkable except for the rapid return of the patient's calcium to within normal levels (8.9 mg/dL). The patient's family refused any adjuvant treatment despite knowledge of the unusual and highly aggressive nature of this malignancy.

Six months after surgery, disseminated recurrence was noted on computerized axial tomographic (CAT) scan. Metastases were present in the lung parenchyma, mediastinum, paraaortic nodes, and left supraclavicular nodes. The serum calcium was 19.6 mg/dL at the time of the recurrence. The family still refused treatment, and the patient died eight months after her original surgery. A request for an autopsy was refused.

The gross pathologic examination of the ovary showed a solid, encapsulated, yellow, nodular mass with focal hemorrhagic areas and empty cystic spaces. On microscopic evaluation, the tumor consisted of uniform cells, with vesicular nuclei arranged in nests or trabecular patterns. These cells had little cytoplasm and were rounded in appearance. There were numerous pseudofollicles present (Fig 1). The tumor was actively undergoing mitosis. The ascitic fluid contained malignant cells; all other staging biopsies were negative for metastatic disease. The patient was diagnosed as having a Stage IC small cell carcinoma of the ovary.

Discussion

Small cell carcinoma of the ovary is a rare and highly malignant tumor frequently associated with paraendocrine hypercalcemia—in 70% of reported cases when calcium was measured.[1-4] This patient is the youngest known to be reported. Previous reported ages have ranged from 12 to 35 years, with a median age of 20 years. Presenting symptoms are generally nonspecific. Rarely do patients present with symptomatic hypercalcemia as did this patient. More commonly, hypercalcemia is noted on the routine admission laboratory data. Germ cell malignancies are the most common ovarian neoplasm seen in this young population, and small cell carcinomas must now be added to the list of potential malignancies, especially if there is associated hypercalcemia. The hypercalcemia associated with this neoplasm is relieved by surgery in two thirds of the cases and becomes elevated with tumor recurrence. This makes the serum calcium level an important marker for treatment response and identifying potential recurrence. Patients usually experience recurrence within ten months and die from the disease in an average of 15 months. Of 60 patients with one year of follow-up, only seven were alive, and all had Stage I disease.[5] This disease has a propensity for extensive intra-abdominal spread, retroperitoneal spread, and distant metastases. The course is generally rapid and progressive despite therapy.

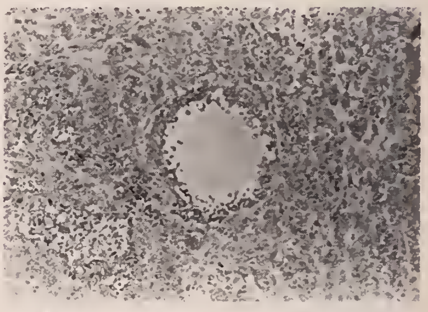

FIGURE 1. Sheets of uniform small cells arranged in nests surrounding a follicle-like structure with eosinophilic fluid (hematoxylin-eosin stain; original magnification × 200).

Controversy exists over the cell type of origin for this malignancy. A germ cell origin has been suggested by Ulbright and colleagues,[3] with the recom-

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mendation that this tumor be recognized as "poorly differentiated ovarian carcinoma of probable germ cell type." Dickersin and coworkers[1] believe that this neoplasm is epithelial in nature. However, they were unable to ascertain the cell type of origin and designated these tumors nonspecifically as small cell carcinomas.[1] In a series of 15 cases of small cell carcinoma incorporating histologic, immunohistochemical and electron microscopic investigations, Aquirre et al[6] were unable to evaluate the cell of origin of this neoplasm. The term "small cell carcinoma of the ovary" adds additional confusion, since this neoplasm is unlike small cell carcinomas seen in other sites. On histologic examination, there are no neurosecretory granules, and consideration of better terminology seems appropriate.

Multiple treatment regimens, including radiation therapy and chemotherapy, have been utilized but have not been successful. Recently, Senekjian et al[7] utilized a six- or seven-drug regimen (vinblastine, cisplatin, cyclophosphamide, bleomycin, doxorubicin, etoposide, with and/or without vincristine) in five patients with small cell carcinoma of the ovary. Three had no evidence of disease after six courses, and two had a partial regression of their tumor recurrence. Despite this drug combination showing some activity in this malignancy, only one patient remained without evidence of disease at 29 months.[7]

Even though the patient described here refused adjuvant therapy, it appears unlikely that her prognosis would have been altered had it been employed. At the present, the origin of this malignancy remains unknown, and there appears to be no effective treatment regimen even in early stage disease.

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Endometrial carcinoma in five patients with breast cancer on tamoxifen therapy

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The nonsteroidal antiestrogen agent tamoxifen is commonly employed in the treatment of breast cancer.[1] Patients whose tumors are both estrogen- and progesterone-receptor positive have a response rate of almost 80%, whereas only a 5% response rate is seen in patients with negative receptor status. Tamoxifen has been associated with only minimal side effects; these include nausea, vomiting, hot flushes, and visual disturbances. Vaginal bleeding may also result from tamoxifen's stimulatory effect on the endometrium of postmenopausal women. Endometrial cancer has been reported in three patients receiving tamoxifen.[2–4] We report here five additional cases in which endometrial cancer developed in postmenopausal patients with breast cancer who were treated with tamoxifen, suggesting a possible causal relationship.

CASE REPORT

Case 1. A single, nulliparous, hypertensive, 65-year-old woman underwent a right modified radical mastectomy in February 1978 for adenocarcinoma. Approximately one year later, an asymptomatic nodular density was noted in the right lower lobe of her lung on a chest film. A needle aspiration biopsy showed clusters of atypical cells. Further tests to identify metastases were negative, and the patient was placed on tamoxifen, 10 mg twice a day, in January 1979. A chest radiograph in March 1979 revealed a complete disappearance of the nodule, and the patient was maintained on the tamoxifen regimen for almost eight years with no evidence of recurrent carcinoma.

In October 1986, at the age of 73, vaginal bleeding led to a dilation and curettage procedure. The endometrial scrapings showed an infiltrating adenocarcinoma with focal areas of squamous differentiation. Histoimmunologic analysis for estrogen receptors was negative. A total abdominal hysterectomy with bilateral salpingo-oophorectomy was performed six weeks after radiation was administered with an implant. Residual infiltrating endometrial adenocarcinoma involving half of the myometrial wall was found.

Case 2. A single, nulliparous, obese woman who had diabetes and hypertension underwent a right modified radical mastectomy at the age of 53 in January 1967. Eleven of 46 lymph nodes were positive for tumor at the time. Twelve years later, in June 1979, at the age of 65, low back pain radiating down the left thigh was noted. Radiographic studies revealed blastic metastases to the L3 lumbar vertebra, and the patient was started on tamoxifen, 10 mg orally, twice a day. Within one month the pain disappeared entirely. Four and a half years later, at the age of 70, while the patient was still taking tamoxifen, vaginal bleeding led to a diagnosis of endometrial adenocarcinoma. The patient had a radiation implant followed by a hysterectomy in November 1983. Pathologic studies revealed well-differentiated adenocarcinoma.

The patient was continued on tamoxifen until a recurrence of pain at L3 radiating down both legs was noted in October 1986. At that time, megestrol acetate, 40 mg orally four times a day, was substituted for tamoxifen, and the pain disappeared entirely within one month. She continues to do well on this medication.

Case 3. An 80-year-old woman who had

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two children underwent a hysterectomy in October 1987 for well-differentiated adenocarcinoma of the endometrium. The pathologic description included cystic hyperplasia, squamous metaplasia, and marked glandular atypia as well. Eight years previously, the patient had undergone bilateral mastectomies for breast carcinoma, and she had received tamoxifen, 10 mg twice a day, which she continued to take until June 1987.

Case 4. An 80-year-old married, nulliparous woman had undergone a modified radical mastectomy in 1980 and had been placed on tamoxifen therapy. Nearly eight years later, a hysterectomy revealed endometrial carcinoma.

Case 5. A 72-year-old woman who had two children had undergone a right modified radical mastectomy in 1971 and a left modified radical mastectomy in 1983. She was placed on tamoxifen after the second operation; the medication was continued for more than four years, when a hysterectomy revealed endometrial carcinoma.

DISCUSSION

This report describes five postmenopausal women with breast carcinoma in whom endometrial carcinoma developed while they were taking tamoxifen. Three other cases of endometrial cancer that developed in women treated with tamoxifen for metastatic breast cancer have recently been reported.[2-4] In those cases, endometrial carcinoma developed 12, 7, and 14 months after tamoxifen was begun, a much shorter duration of treatment than in the patients reported here.

Three of the five patients reported here were nulliparous, a well-known risk factor for endometrial carcinoma. In addition, patient 2 was obese, diabetic, and hypertensive as well. Unfortunately, none of the patients discussed here had had endometrial sampling prior to the diagnosis of endometrial carcinoma. As the length of time the patients were on tamoxifen before the diagnosis of endometrial cancer was made varied from four to eight years, perhaps premalignant changes might have been detected in time to reverse the process. If, indeed, the premalignant changes were reversible by discontinuing tamoxifen, a strong case could be made that tamoxifen induces endometrial carcinoma.

The mechanism by which tamoxifen may induce endometrial carcinoma in some women is not clear. In one study of the endocrine effects of tamoxifen in postmenopausal patients with breast cancer, the drug caused a weak stimulatory effect on the endometrium as well as “estrogenization” of vaginal smears, indicating an agonistic effect on these targets. Simultaneously, levels of circulating estrogen did not increase, suggesting the endometrial changes were not caused by changes in estrogen levels.

An alternative possibility that tamoxifen treatment and the development of endometrial carcinoma are not related causally must be considered. In a study involving 60,065 women in Sweden with breast cancer, there was a significantly increased incidence of endometrial cancer than was expected. In the patients presented here, other risk factors were present as well.

Tamoxifen is widely used in the treatment of metastatic breast cancer, and the drug is generally well tolerated with minimal side effects. It is now recommended in the adjuvant setting for postmenopausal patients as well, which will probably increase its use. If indeed it stimulates the postmenopausal endometrium and at times may induce carcinoma, as in the case with estrogens, one should observe this association more frequently in the next few years. Physicians should be alerted to the possibility of endometrial cancers developing in women on tamoxifen. Endometrial screening should be part of the follow-up treatment for patients on tamoxifen. If premalignant changes are found, it would be of great interest to know if the addition of progestins can reverse the process.

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Superior vena cava syndrome associated with a Hickman catheter

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Superior vena cava (SVC) syndrome is usually caused by the local effects of a tumor of the mediastinum. The development of this syndrome may be the presenting feature of the tumor. It is important to recognize that other causes of the superior vena cava syndrome do exist and may confuse management, particularly in patients with known malignancies.

Described below is a patient in whom SVC syndrome developed during a one-week period, due to thrombus associated with a Hickman catheter.

CASE REPORT

A 58-year-old man came to the emergency department complaining of a one-week history of intense shortness of breath each time he would lie down. He noted that after “finding the right position” (usually decubitus), he would be able to sleep normally. There had been no orthopnea, cough, chest pain, or paroxysmal nocturnal dyspnea. The episodes also occurred in daylight hours. During the same one-week period, he also reported dyspnea on exertion, aching in both shoulders, and heaviness in his arms, neck, and face. He and his wife noted swelling of his face and neck, especially in the morning.

Four years prior to admission, carcinoma of the colon had been diagnosed. Metastases involving the peritoneum had been discovered eight months prior to admission. At that time, an indwelling silastic, Hickman catheter was placed in order to administer 5-fluorouracil infusions. A dose of 400 mg daily had been given for several months by con-

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stant infusion, with heparin added to the infusion.

The patient had never smoked and had no history of pulmonary or cardiac diseases.

Physical examination revealed a well-developed, well-nourished man in no distress. His temperature was 36.5°C; pulse, 72/min; blood pressure, 140/86 mm Hg; and respirations, 16/min and unlabored. The chest was clear to auscultation and percussion. The heart examination revealed no murmurs or gallops, and no rub. The point of maximal impulse was palpable in the fifth intercostal space, 9 cm left of the mid-sternal line. The abdominal examination was normal. The neck was enlarged, and the face was plethoric. The neck and arm veins were dilated at 45°. The rectal examination was normal. There were no palpable lymph nodes.

While supine during examination, the patient developed cyanotic facial coloration and marked dyspnea. He improved immediately on assuming the left lateral decubitus position.

The chest radiograph was normal and showed the left-sided Hickman catheter with the tip near the junction of the left innominate vein and the superior vena cava. Prominence of the right paratracheal area was noted. The electrocardiogram (ECG) showed incomplete right bundle branch block and normal sinus rhythm. Arterial blood gases on room air showed a pH of 7.41; P_{CO_2}, 32 mm Hg; P_{O_2}, 76 mm Hg; and oxygen saturation, 95%. The hemoglobin was 11.8 g/dL; hematocrit, 35%; and white blood cell count, 7,200/mm^3. The sodium was 140 mEq/L; potassium, 4.2 mEq/L; bicarbonate, 21 mEq/L; and glucose, 101 mg/dL.

A computed tomographic (CT) scan of the chest did not show a mass or mass compression of the SVC, and no abnormal lymph nodes were noted. The SVC was opacified, of small caliber, and appeared patent. Contrast venography revealed extensive thromboses involving the SVC and the right and left innominate and subclavian veins. The azygos vein was patent and uninvolved.

Urokinase was infused for 12 hours at 4,400 IU/kg/hr, following an initial 2,000-IU bolus. Heparin was then infused to prevent recurrent thrombus formation. Nine days later, the patient was discharged on warfarin, and his condition was markedly improved. The catheter was left in place, and chemotherapy was continued.

DISCUSSION

William Hunter[1] provided the first description of SVC obstruction in 1757 in a patient whose gigantic aortic aneurysm eventually eroded through the chest wall. Aneurysms prevailed as the most common cause of SVC syndrome until the decline of syphilis in the latter half of this century. Malignant tumors are now responsible for the majority of cases, the most common being bronchogenic carcinoma (85%) or lymphoma (10%).[2]

The patient described presented with symptoms typical of SVC syndrome. Suffusion, a feeling of fullness, is a commonly associated symptom, as is dyspnea. Other symptoms may include headache, vertigo, or decreased mentation. These reflect increased intracranial pressure due to obstruction of venous drainage. Edema of the head and neck may also result in laryngeal edema and airway compromise. Presentation may be subacute. One series involving 107 patients noted that the average duration of symptoms prior to presentation to a physician was 15 weeks.[3]

Although discussions of the SVC syndrome are found in all major medical and surgical textbooks, the disorder is uncommon. Only 86 cases were noted in a 20-year comprehensive review of all cases seen at the Mayo Clinic.[4] Of those, 78% were due to malignancies, and 64% of the patients died. Three cases (4%) resulted from thrombosis associated with central venous catheters. A study involving 66 patients with the syndrome seen at the University of Iowa over a ten-year period noted that 97% of cases were due to a malignant tumor.[5]

The advent of the silastic rubber Hickman catheter[6] has spared many patients with malignancies the pain of frequent venipunctures and intravenous placements for medication administration. These catheters are normally left in place for extended periods of time after fluoroscopic placement in the lower superior vena cava.

Overall complications with the Hickman catheter occur at a rate of about 0.3 per 100 catheter days, with the infection rate reported as 0.14 per 100 catheter days.[7] Complications include infiltration with extravasation of infusate, infections, hemorrhage, major thrombus formation, catheter severance, catheter malfunction, and air embolism. Thrombus formation within the catheter is commonly seen[8] and is minimized by the addition of heparin to chemotherapy infusions and by the use of fibrinolytic enzymes to restore catheter patency if occluded.[9]

SVC syndrome associated with Hickman catheters has rarely been reported.[8,10-12] There have been a number of other reports, however, of thrombus formation and complications involving other major veins (especially the subclavian). Tricuspid obstruction and pulmonary embolus development have been noted. A recent report noted catheter related thrombosis in 17% of a series of 168 patients with solid tumors.[13] In addition, SVC thrombi have been noted in association with SVC syndrome caused by tumor compression and are attributed to stasis effects.[14] However, patients with malignancies are at increased risk of thrombus formation due to tumor effects on the coagulation system.[15] Chemotherapeutic agents can irritate vascular structures and result in thrombophlebitis and thrombus formation.

Silastic and polyurethane catheters are much less thrombogenic than stiffer catheters made of polyvinylchloride or polyethylene. Major thrombus formation is more common with prolonged catheterization and when very hyperosmolar solutions are infused. The latter predisposes to intimal damage.

Superior vena cava syndrome requires urgent diagnostic studies to determine the cause. The chest radiograph will often show a mediastinal mass, but radiographic studies may be normal. For example, nine (13%) of 67 patients in the Mayo Clinic study who had malignant tumor compression of the SVC presented with normal chest radiographs.[4] When thrombus is suspected to be the cause of SVC syndrome, contrast venography of both upper extremity venous systems will adequately demonstrate obstruction.[16]

Palliative radiation is usually the treatment of choice for compression by a malignant tumor,[4] but chemotherapy may be preferred for some lymphomas and for small cell carcinoma of the lung.[17] Patients may improve within three to four days of treatment. The overall prognosis is guarded in many patients and depends on the cause of the syndrome. Although diuretics and steroids are often used, no controlled trials have established the efficacy of either of these agents. Balloon angioplasty to maintain SVC patency has been used in a patient with critical compression due to a tumor.[18]

In patients with central catheters, especially if there are known malignancies, it is essential to consider thrombus formation as a possible cause of the SVC syndrome, since the available treatment is effective, and the patients will likely die without it. In the few reported cases and in the patient described above, thrombolytic medications and heparin have been used successfully for treatment.[10,11,19] This underscores the importance of broadening clinical suspicions regarding the causes of this syndrome.

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Isolated adrenocorticotrophic hormone deficiency presenting as chronic hyponatremia

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We report a case of isolated adrenocorticotrophic hormone (ACTH) deficiency associated with autoimmune thyroid disease that was undiagnosed for two years, during which time the patient had chronic hyponatremia. The manifestations of isolated ACTH deficiency are reviewed and are compared with those of primary adrenal insufficiency, and the mechanisms that cause abnormal water balance in this disorder are discussed. This case emphasizes the need to consider disorders of pituitary ACTH secretion in the differential diagnosis of unexplained hyponatremia.

CASE REPORT

A 58-year-old man had hypothyroidism, which had been diagnosed in 1976 by a low serum T4 level and elevated thyroid stimulating hormone (TSH) level. He was given L-thyroxine, 50 μg daily, but was lost to follow-up. For two years prior to the current presentation, he had hyponatremia, for which he was treated as an outpatient and inpatient with serum sodium ranging from 124 mEq/L to 134 mEq/L. He did not respond to fluid restriction or demeclocycline, and no specific diagnosis was made.

Seven months prior to presentation, he was admitted for gangrene and underwent a left femoral popliteal bypass. A rapid ACTH stimulation test was performed to evaluate the hyponatremia. This was interpreted as normal based on doubling of the baseline value from 4 μg/dL to a maximal stimulated value of 8 μg/dL 30 minutes after ACTH infusion. The graft thrombosed, and he was readmitted for amputation. Both surgical procedures were well tolerated without hypotension. An endocrine consultation was requested for the chronic hyponatremia.

The review of systems was significant for stable angina, chronic fatigue, and generalized weakness. He had no symptoms of hypoglycemia, hypothyroidism, sexual dysfunction, fever, arthralgias, abdominal pain, nausea, vomiting, or hyperpigmentation. The family history was positive for Addison's disease in his mother and Hashimoto's thyroiditis in a sister. His medications included nitrates, thyroxine, and diltiazem. On physical examination he appeared younger than his stated age, with nonhyperpigmented, somewhat carotenemic skin. He was neither febrile nor hypothermic. There were no orthostatic changes in blood pressure or pulse, and perfusion appeared normal. Axillary and pubic hair were normal. The testes were normal in size.

The laboratory evaluation included sodium, 128 mEq/L; potassium, 3.9 mEq/L; blood urea nitrogen (BUN), 10 mg/dL; creatinine, 0.6 mg/dL; plasma osmolality, 241 mosm/kg H_2O; urine osmolality, 269 mosm/kg H_2O; urinary sodium, 64 mEq/L; hemoglobin, 10 g/dL; and hematocrit, 30% with normal red blood cell indices. The T4 level was 2.9 μg/dL (normal range, 4.9-10.1 μg/dL); T3 resin uptake, 0.82; and TSH, 17 μU/mL. A rapid intravenous ACTH stimulation test was abnormal, with a baseline cortisol level of 4 μg/dL and a peak of only 14 μg/dL at 30 minutes after infusion with 250 μg synthetic ACTH.

Insulin-induced hypoglycemia was not used to test pituitary function because of the patient's coronary disease. Pituitary testing included arginine infusion, standard overnight metyrapone test (30 mg/kg at 2400 hours), a combined gonadotropin releasing hormone (GnRH) and thyrotropin releasing hormone (TRH) infusion, and a three-day ACTH stimulation test. Standard water loads, including radioimmunoassay of plasma arginine vasopressin (AVP) levels, were performed with and without cortisol replacement and were repeated after the patient was chemically euthyroid.

With the exception of the pituitary-adrenal axis, pituitary function was normal. Basal TSH was elevated to 17 μU/mL, with a stimulated level of 54 μU/mL after TRH. A computed tomographic (CT) scan of the head demonstrated a normal pituitary without sellar or suprasellar lesions. The metyrapone test indicated absent ACTH response, with a baseline plasma compound S level of 0.2 μg/dL and a stimulated value of only 0.8 μg/dL. A three-day ACTH infusion (250 μg infused over eight hours on each day) showed low baseline serum and urinary cortisol values, but a normal stepwise increase during the infusion (Table I).

Water loads were performed before and after acute corticosteroid replacement (Fig

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TABLE I. Three-Day ACTH Infusion Test

Study Day	8 AM Plasma Cortisol (μg/dL)	Urinary 17 Hydroxy-corticosteroids (mg/24h)	Urinary Free Cortisol (μg/24h)	Urinary 17 Keto-steroids (mg/24h)
1 Baseline	7.0	1.2	NDA	4.8
2 Baseline	11.0	1.0	NDA	4.4
3 ACTH, 250 μg	19.0	10.5	490.0	8.4
4 ACTH, 250 μg	21.0	23.4	1188.0	12.7
5 ACTH, 250 μg	14.0	22.6	1196.0	15.4

NDA = no detectable amount.

1, A). The patient excreted 38% of the water load, with maximum urinary dilution to 275 mosm/kg H_2O and a decrease in plasma osmolality (posm) from 279 to 269 mosm/kg H_2O. Basal plasma vasopressin (AVP) was elevated at 2.2 pg/mL, with maximal suppression to only 1.6 pg/mL. After four days of corticosteroid therapy (hydrocortisone 30 mg/day), the water load was still abnormal, with 49% excretion; maximal urinary dilution was 272 mosm/kg H_2O, and there was a decrease in posm from 282 to 275 mosm/kg H_2O. Plasma AVP suppression was still incomplete—from 2.8 pg/mL to 1.3 pg/mL.

Four months after thyroid and cortisol replacement therapy, when T4 and TSH levels were within normal limits, the water loads were repeated (Fig 1). The patient was markedly improved, with excretion of 80% of the water load, and urine dilution to 107 mosm/kg H_2O. Baseline AVP was 1.2 pg/mL, with suppression to 0.7 pg/mL (limits of assay detection = 0.5 pg/mL). However, four days following cortisol withdrawal, the water load was again markedly abnormal, with only 23% total excretion, maximal urinary dilution to only 270 mosm/kg H_2O, and a decrease in posm from 279 to 270 mosm/kg H_2O. Plasma AVP suppression was noted, though less than initially, from 1.4 to 1.0 pg/mL. Antithyroglobulin and antimicrosomal antibodies were positive at 1:1600 and 1:2500 dilutions, respectively. Adrenal, parietal cell, pancreatic islet cell, and testicular antibodies were all absent.

The patient was maintained on 30 mg/day of hydrocortisone and 100 μg/day of thyroxine, with complete resolution of hyponatremia and anemia. He remains well after more than one year of medical follow-up.

DISCUSSION

Isolated ACTH deficiency accounted for 8% of adrenal insufficiency diagnoses for a period of 14 years in a general hospital population.[1] Figure 2 summarizes the signs and symptoms found in the patient described here, along with the characteristics of 46 other reported cases of isolated ACTH deficiency,[2-37] and contrasts these with signs and symptoms of 264 reported cases of primary adrenal insufficiency.[38-40] The average patient age was 48 years and ranged from 2 to 74 years. There was a male to female ratio of 2:1. The clinical presentation varied from asymptomatic to critically ill. The classical features of primary adrenal insufficiency may be seen, with two major exceptions: hyperkalemia, since mineralocorticoid secretion remains intact with ACTH deficiency; and hyperpigmentation, since ACTH is absent rather than elevated. The most common presentations are malaise, lethargy, and weakness (54%), and hypoglycemia (including fasting, postprandial, and alcohol induced) (57%). Gastrointestinal symptoms are also fairly common (40–50%). Hyponatremia and anemia are less common (20–30%) and, as in this case, when these have been the

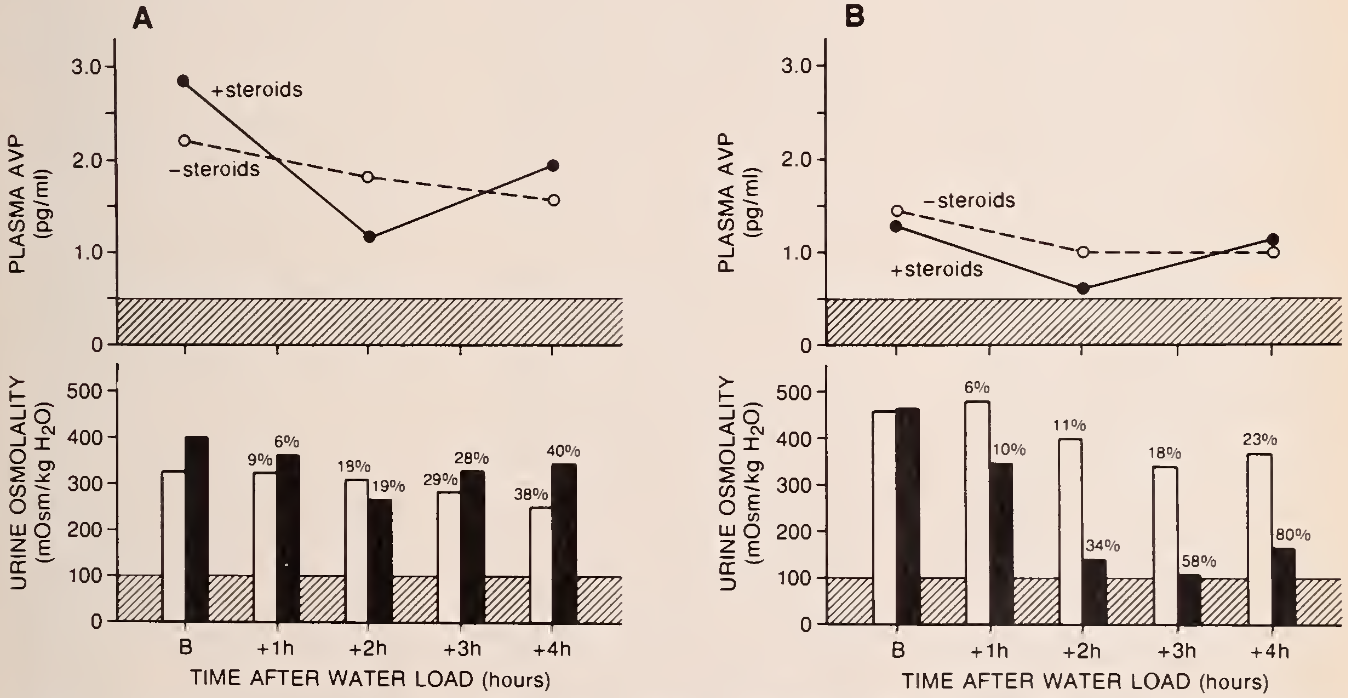

FIGURE 1. Water load tests with (+) and without (−) steroids. Results of plasma AVP levels (upper panel) and urinary osmolalities (lower panel) during 20 mL/kg water load tests are plotted: Left, at the time of the initial evaluation; Right, after four months of thyroid and hydrocortisone replacement. B represents basal values before water loading. The shaded areas represent the minimum detectable plasma AVP in the assay (upper panels) or the minimum urine osmolality required for classification of a normal test (<100 mosm/kg H_2O) (lower panels). The percentages above the bars represent cumulative percent excretion of the water load after each hour. Left, open circles and bars represent initial values and filled circles and bars indicate results after three days of hydrocortisone replacement. Right, the solid circles and bars represent results following four months of replacement with thyroid hormone and steroids whereas open circles and bars represent results of a repeat test done after three days of hydrocortisone withdrawal.

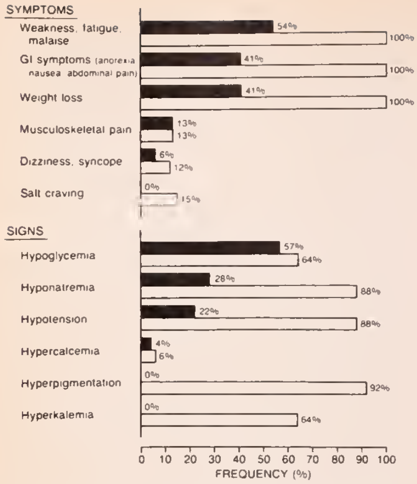

FIGURE 2. Comparison of frequency of reported symptoms and signs of isolated ACTH deficiency and primary adrenal insufficiency. Reported frequency of symptoms (upper panel) and signs (lower panel) of isolated ACTH deficiency (solid bars, present case plus 46 reported cases) and Addison's disease (open bars, 264 reported cases) are shown.

major features, the diagnosis has frequently been overlooked for a period of time. Hypotension, commonly seen in primary adrenal insufficiency, is less frequent with isolated ACTH deficiency (22%).

In general, the manifestations of hypocortisolism are less frequent and less severe in isolated ACTH deficiency than with primary adrenal insufficiency. Although the manifestations should be similar in panhypopituitarism, the obvious advantage of this comparison is its focus on signs and symptoms attributable to hypocortisolism alone in the absence of contributions from other anterior pituitary hormone deficiencies.

There are several likely reasons for the less severe symptoms with ACTH deficiency. First, mineralocorticoid secretion remains normally regulated by the renin angiotensin system even in the absence of ACTH, thus patients are not as prone to marked salt and volume depletion. Indeed, this patient underwent two major surgical procedures without crisis. Second, ACTH deficiency causes only relative hypocortisolism. Plasma cortisol levels are low or low normal, but still measurable, in contrast to a total absence of circulating cortisol in many cases of Addison's disease.

When ACTH deficiency is suspected, the initial screening test should be a standard rapid ACTH stimulation test.[41] While criteria for normality vary, an absolute plasma cortisol value greater than 18 μg/dL at 30 minutes would be unequivocally normal. Attempts to interpret the relative cortisol increment (eg, doubling of baseline values) in patients with low basal values should never be used as the sole criterion for a normal test.[42] Subnormal responses require further study via a three- to five-day ACTH infusion. A normal or stepwise increment in urinary steroids, as demonstrated in this patient (Table I), confirms secondary rather than primary adrenal disease. A low ACTH level prior to the infusion would also represent supportive evidence, but was not done in this case. Dynamic pituitary function testing must be done as well to assess secretion of other pituitary hormones. The diagnosis of isolated ACTH deficiency is established by a failure to stimulate cortisol or ACTH secretion with insulin-induced hypoglycemia or metyrapone, as in the present case, accompanied by normal adrenal response to prolonged ACTH infusion and intact secretion of other pituitary hormones.

Proposed mechanisms for the defect include reduced hypothalamic corticotropin releasing factor (CRF), insufficient ACTH secretion and release, secretion of an abnormal ACTH molecule, circulating antibody to ACTH, and ACTH receptor defect. In cases in which these possibilities have been studied, most patients have not responded to CRF[32,33,35] or arginine vasopressin,[20,32-34] suggesting complete pituitary ACTH deficiency. However, this area remains under active investigation. The association of ACTH deficiency with autoimmune thyroiditis, as in this case, has been reported,[26,27,36] as has TSH deficiency, pernicious anemia, hypoparathyroidism, diabetes mellitus, and lymphoid hypophysitis.[10,14,21,24,28,32] There have similarly been reports of other isolated pituitary hormone deficiencies with polyglandular autoimmune disorders. Such provocative findings continue to suggest that autoimmunity may contribute to the pathogenesis of some cases of isolated ACTH deficiency.

Hyponatremia is a common electrolyte disorder in the United States, with incidences of 15–22% in hospitalized patients if a criterion of plasma sodium less than 135 mEq/L is used, although the incidence is 1–4% if only patients with plasma sodium less than 130 mEq/L are included.[43,44] Hyponatremia is a recognized cause of morbidity and mortality.[45] While severe hyponatremia may cause neurologic dysfunction and seizures, less marked degrees are associated with as much as 60-fold higher mortality rates when compared to normonatremic hospitalized patients.[46] Thus, even mild hyponatremia should be investigated in order to treat potentially correctable causes, as well as to identify occult malignancies associated with ectopic AVP production and the syndrome of inappropriate antidiuretic hormone secretion (SIADH).[47]

Despite the well-known association of hypocortisolism and hyponatremia,[48] this diagnosis was missed in this patient for two years. Delay in diagnosis is unfortunately not uncommon in cases of isolated ACTH deficiency. The patients do not generally have a striking clinical presentation, and the recognition of hypocortisolism is more difficult. Additionally, since the ACTH deficiency is "isolated" with otherwise normal pituitary function, patients do not have stigmata of panhypopituitarism such as growth failure, hypogonadism, and secondary hypothyroidism; in addition, head CT scans are normal. Finally, because serum and urinary cortisol levels are not absent, and relative responses to rapid ACTH infusions are not uncommon, clinicians may overlook the diagnosis, as happened in this case.

Due to similar fluid and electrolyte patterns, patients with hypocortisolism are frequently diagnosed as having SIADH; however, it should be remembered that the final criterion necessary to establish a diagnosis of SIADH remains normal renal and adrenal function.[49] The etiology of hyponatremia in this patient was likely multifactorial. Hypothyroidism has been the sole cause of hyponatremia in many patients.[50] Although this may be caused by abnormal AVP secretion,[51] impaired distal delivery of glomerular filtrate appears to be the major factor causing impaired water excretion in most cases.[52,53] Hypothyroidism may have worsened this patient's hyponatremia initially, however it clearly was not the only cause of water retention, since the water load remained abnormal in the euthyroid cortisol deficient state.

The mechanism of abnormal water excretion in the cortisol depleted state has been controversial. Previous animal and human studies[54-56] have emphasized the contributions of either inappropriate AVP secretion or tubular effects of cortisol. It is significant that in this case the data clearly support the importance of both AVP excess and tubular effects of cortisol in maintaining impaired water excretion. Furthermore, these data also demonstrate that these mechanisms are not immediately reversible, since four days of corticosteroid replacement were not sufficient to normalize plasma AVP levels and water excretion. In addition, it is apparent that complete absence of circulating cortisol is not necessary to impair free water excretion sufficiently to produce clinically significant hyponatremia, since this patient manifested significant hyponatremia despite only relative deficiencies in cortisol levels.

In summary, isolated ACTH deficiency is a rare but known cause of cortisol deficiency in adults and children. Although the clinical presentation is not dramatic, as in primary adrenal insufficiency, the presence of unexplained hypoglycemia or hyponatremia should always warrant consideration of the diagnosis. As in this case, the entity can often be misdiagnosed as SIADH. Other autoimmune endocrine disorders may be associated with this disorder and should be evaluated. The mechanism of abnormal water balance in this disorder appears to be multifactorial, and both tubular effects of cortisol and abnormal AVP secretion contribute to hyponatremia. Finally, the hyponatremia is completely correctable with glucocorticoid replacement, but periods of treatment greater than four to seven days may be required to produce a clinical response.

Acknowledgments. The authors thank the nurses of The Clinical Research Center for their assistance with the clinical studies, and Dr Alan G. Robinson for his ongoing support and critical review of the manuscript. This work was supported by the Clinical Research Center. The first author is currently supported by the E. Clarence Rice Fellowship of the American Diabetes Association Washington, DC, affiliate.

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LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Paraesophageal hernia

TO THE EDITOR: I should like to comment on the excellent article entitled "Paraesophageal hernia," published in the December 1989 issue of the *Journal.*[1]

I would like to elucidate certain points and offer certain alternative suggestions based on my experience and my reading of the current literature.

I agree, based on the long experience of Skinner and Belsey[2] and my own 20 years' experience, that these patients should be operated on as soon as possible after discovery of a paraesophageal hernia because of the high rate of complications. However, I do not feel that the abdominal approach is preferred. Four things, in my opinion, favor the thoracic approach.

First, in elective reductions, it is often necessary to mobilize the entire distal esophagus, which is more easily done through a transthoracic incision, in order to do an appropriate reduction and antireflux repair, as commented on in the article in DeMeester's series.[3] I concur that one can prevent the conversion of Type II to a Type I hernia. One can also prevent reflux and recurrence by loosely wrapping the esophagus with cardia and anchoring the repair in the abdomen to avoid the high incidence of recurrence when a simple Allison repair is done.

Second, the procedure from below should not be termed a Mark IV Belsey since the repair is only done appropriately through the chest, as stated by Belsey.[4]

Third, in an acute case, the incarcerated and twisted bleeding organ in the chest may be extremely fragile and tear or separate on pulling down. Furthermore, previous incarcerations may result in intense inflammatory reaction on the superior surface of the crus fixing these organs in the chest and, again, making blind dissections from below quite dangerous. A simple Nissen repair which is a wrap and suturing to the junction is inadequate when there is both reduction and removal of the intrathoracic sac. This is not a Nissen but a modified wrap.[4(p593)]

Fourth, in struggling with an acute situation with the stomach incarcerated with or without the colon in the chest, there is an extremely good chance of injuring short gastric vessels or splenic vessels in the dissection from below, necessitating splenectomy. This is a major complication of the abdominal approach. It is avoided by going transthoracically where all the structures at risk can be directly visualized. With this approach the spleen is protected by the diaphragm. Parenthetically, with current thoracic anesthesia, the mortality from a transthoracic procedure may be as low as 0.3%.[4(p590)]

With these four annotations, I thoroughly agree with the basic thrust of this article and commend the authors' conclusions.

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1. Vitelli CE, Jaffe BM, Kahng KU: Paraesophageal hernia. *NY State J Med* 1989; 89:654–657.
2. Skinner DB, Belsey RH: Surgical management of esophageal reflux and hiatus hernia. Long term results with 1,300 patients. *J Thorac Cardiovasc Surg* 1967; 53:33–54.
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In reply. We would like to thank Dr Sicular for his comments. His points with regard to a thoracic rather than an abdominal approach all merit consideration in the repair of paraesophageal hernias. In situations where a longstanding, large, strangulating paraesophageal hernia is present, a thoracic approach may prove to be advantageous. However, in our own limited experience, inability to reduce the hernia has not been encountered. Although mortality rates for transthoracic procedures may be as low as that cited by Dr Sicular, this figure reflects mortality in the best of circumstances. Since the typical presentation of a paraesophageal hernia is in the older patient, often after the development of a life threatening complication, the morbidity of a transthoracic approach may be much more significant.

With these considerations in mind, the selective use of a transthoracic approach may be most appropriate. For smaller paraesophageal hernias without evidence of strangulation, the routine use of an abdominal approach can be recommended; however, for those that are very large and presenting with strangulation, a thoracic approach should be strongly considered (TR DeMeester, personal communication, February 1990).

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Animal experimentation for medical research

TO THE EDITOR: The attack against antivivisectionists, as illustrated in the report by Rosner et al,[1] represents a "straw man" argument that avoids the real issues regarding animal research. The real threat to animal researchers has never been antivivisectionists, who have never had the numerical and financial strength to achieve their goals. The real danger to animal researchers has always been the dissent of clinical physicians who challenge the scientific validity of animal models.[2]

Today, there are more than 2,000 physicians in the Medical Research Modernization Committee (MRMC) and the Physicians Committee for Responsible Medicine who are highly critical of animal research practices. Rosner et al, dismissing animal research critics as "a small but very vocal and energetic minority of the public,"[1] failed to acknowledge that many physicians encourage wider use of nonanimal methodologies, particularly modern clinical research technologies such as non-invasive scanning, epidemiology, and post-marketing surveillance.

Before accepting the call of Rosner et al to join the campaign of the Foundation for Biomedical Research and the American Medical Association, doctors should recall their own experiences involving animal research. Were the studies scientifically sound? Were animals treated in a humane, responsible manner? Many physicians, such as myself, were troubled by personal experiences, and this encouraged a closer examination of animal research practices. We have concluded that the historical and contemporary value of animal research has been grossly overstated, and we will gladly supply interested physicians with our findings.

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1. Rosner F, Bennett AJ, Cassell EJ, et al: Animal experimentation for medical research. *NY State J Med* 1989; 89:671-676.
2. *A Critical Look at Animal Research*. New York, Medical Research Modernization Committee, 1990.

In reply. Dr Kaufman is seriously mistaken when he writes that antivivisectionists "have never had the numerical and financial strength to achieve their goals." The animal rights movement has become increasingly vocal and violent during the past decade, with break-ins at research institutions, destruction of property, and the stealing of animals.

On August 15, 1988, animal rights activists staged a pre-dawn raid on an animal holding facility at Loma Linda University and stole a variety of animals and records, dumped red paint on equipment, smashed television sets, and painted graffiti on the walls. On November 11, 1988, a long-time animal rights activist was arrested while in the act of planting a radio-controlled pipe bomb near the parking place of the president of a company involved in medical research. A search of this activist's apartment yielded three more pipe bombs, a bazooka-like weapon, and ammunition. A long campaign of protest by animal rights activists forced a professor at Cornell University Medical College to give up a $600,000 federal grant. On December 12, 1988, several groups including the Physicians Committee for Responsible Medicine held a demonstration at Emory University to kick off another campaign against the use of animals in drug addiction research.

These are just a few examples of the power of the extremely well financed and politically influential animal rights groups. The trend is so alarming that Nobel laureates are speaking out on the need for animals in research,[1] and the American Medical Association's Group on Science and Technology[2] and the Council on Scientific Affairs[3] have recently written major articles in defense of animal research. There is no question that animal experimentation is being threatened by animal rights groups.[4]

The medical community is always open to constructive criticism from fellow physicians and others. Physicians and scientists are also dedicated to treating animals in a humane and responsible manner. A variety of safeguards to insure ethical conduct in the use of animals for medical research are in place. Let us reconfirm our commitment to use animals in a humane and responsible manner in the achievement of the dual goals of finding cures for human diseases and relieving human suffering.

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1. Goldsmith MF: Nobel laureates call for speaking out on need for animals in research. *JAMA* 1989; 262:2647-2648.
2. Loeg JM, Hendee WR, Smith SJ, et al: Human vs animal rights: In defense of animal research. *JAMA* 1989; 262:2716-2720.
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4. Rosner F: Is animal experimentation being threatened by animal rights groups? *JAMA* 1985; 254:1942-1943.

Lead poisoning during home renovation

TO THE EDITOR: The case report by Drs Curran and Nunez, "Lead poisoning during home renovation" was interesting, but unfortunately not unique.[1] Every year, some children develop lead poisoning from renovations. We do not know how many. Since most of these children are from middle class families, their physicians usually assume that they are not at high risk of developing lead poisoning. Thus, they are not tested.

Questions on possible renovations at home or in homes visited are not usually a part of the pediatric medical history. Thus, one cannot rely on data from the history alone to determine whether a child is at increased risk and therefore in need of screening.

For this reason, the New York City Department of Health recommends annual lead screening for all children up to six years of age, regardless of social class or the presence (or absence) of data on specific high risk exposure. Such screening is now required for children in day care, nursery school, and kindergarten in New York City.

Screening is no substitute for proper precautions during renovations. However, it can pick up problems and heighten the community's awareness of the hazard.

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1. Curran JP, Nunez JR: Lead poisoning during home renovation. *NY State J Med* 1989; 89:679-680.

An alma mater by any other name is an alma mater

TO THE EDITOR: As we all know, the older you get, the more attention you pay to the obituary column. This is natural, because this is where many of one's friends and classmates of bygone years appear from time to time. It also provides an answer to "the question, "Whatever happened to so-and-so?"

As a faithful reader of the column I admit to being disturbed by the reference to the medical school from which I graduated (the Long Island College of Medicine, Dec '43) as: the "State University of New York Health Science Center at Brooklyn." Not only did I not attend the latter school, I would have never applied to an institution with a name that long.

I also wonder what two physicians recently featured in your column, ages 91 and 92, must have thought on reading their obituaries over coffee in the next world. The air must have been blue as they fumed to each other.

Enough already of rewriting history. We all know that the former Long Island College of Medicine was taken over and is now that other place. That does not, however, alter the name of the school from which many of us graduated.

Long Island College of Medicine, *Sic Transit Gloria Mundi;* but in the end, *Vincit Omnia Veritas!*

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In reply. Two events that strike at the heart of alumni are when an alma mater closes and when it changes its name. Not infrequently, the latter is also accompanied by a change in physical location. For some, extinction is preferable to a change in name because one is left with happy memories which provide comfort in place of the steady irritation of a new name for which no sentiment can be aroused.

Dr Harvey's point is well made. However, there is no easy solution to the problem he presents. His alma mater has had five different names in the 134 years since it was founded in 1856. It began as the Long Island College Hospital (1856–1930), then became the Long Island College of Medicine (1930–1950), and in 1950 was absorbed by the State University of New York (SUNY) as the College of Medicine at New York City. While two names spanned the school's first 94 years three have been used in the last 40. SUNY College of Medicine at New York City soon became SUNY Downstate Medical Center, College of Medicine, which was replaced in 1986 with the longest name to date: SUNY Health Science Center at Brooklyn, College of Medicine.

Although this medical school has had five name changes, from the legal perspective it is the same institution because it has consistently functioned under its original charter. The present SUNY Health Science Center at Brooklyn, College of Medicine, views itself as the lineal descendant of its predecessors and considers their histories its own.

The issue of whether or not one should be identified as a graduate of an institution under one of its former names can be cogently argued from either side. Sentiments pull to one side while legal arguments and common sense persuade otherwise. It would be an impossible task for medical directories and journals to identify physicians by the names by which their medical schools were once known. This would also not be very useful, as with the passage of time, former names become increasingly unfamiliar to successive generations of both physicians and the public who may not necessarily link them to the current name.

Dr Harvey might take heart in the knowledge that when SUNY Downstate Medical Center, College of Medicine, changed its name to SUNY Health Science Center at Brooklyn, College of Medicine, in January 1986, the graduating class insisted that the name Downstate Medical Center appear on their diplomas. The remaining three classes also made this request, arguing that they had entered the institution when it was known as Downstate Medical Center. However, in due course they will be listed in medical directories as graduates of the SUNY Health Science Center at Brooklyn, College of Medicine, and in the fullness of time their obituaries will carry the same listing, unless of course the name is changed again.

PASCAL JAMES IMPERATO, MD
Editor

Tumor metastasis to an infusaport site

TO THE EDITOR: Implantable venous access devices have become a popular technique for providing long term venous access for the oncology patient. They may be used for chemotherapy, antibiotic infusion, blood component replacement, and sampling. Since they are foreign bodies, complications may arise due to their presence. The following is a case report of metastasis to an infusaport site.

Case Reports. The patient was a 60-year-old woman who underwent a total hysterectomy and bilateral salpingo-oophorectomy for cervical squamous cell carcinoma Stage I in December 1986. In August 1987, she was diagnosed with recurrent cancer in the pelvis. She was to receive 5,000 CGy of radiation therapy, but the radiation therapy was stopped when a rectovaginal fistula developed. In December 1987 she had a colostomy performed for bowel obstruction secondary to metastatic disease in the pelvis. Postoperatively, she received a further dose of 3,000 CGy. An infusaport was placed under local anesthesia via a right cephalic vein cutdown in February 1988 for chemotherapy.[1] In March 1988 cellulitis developed around the infusaport site and the patient was begun on oral antibiotics. Since there was no response to antibiotic therapy, the infusaport was removed. A mass was also noted at the infusaport site. Biopsy was then taken and pathological examination demonstrated metastatic squamous cell cancer of cervical origin encasing the infusaport site. The tumor was not removed. Cultures revealed *Staphylococcus aureus.* The patient was continued on chemotherapy and died two months later from metastatic disease.

Discussion. Clinical and experimental data provide support that tissue trauma and inflammation enhance the ability of a tumor to be metastatic to a given site.[2–4] Fisher et al[5] found that when rats were injected with tumor cells and subjected to mechanical, chemical, and surgical trauma, a significantly greater number of tumor cells lodged in sites of trauma. Inflammatory oncotaxis has been proposed as the mechanism by which tumor cells are attracted to a point of inflammation. Whether there is an actual oncotaxic substance similar to those that attract leukocytes, or whether the neoplastic cell may be an innocent bystander brought passively to these sites by increased blood flow and capillary permeability, is currently purely speculative.[6]

A review of the literature reveals two other reports of metastasis to implantable venous access devices. These consisted of metastasis of the site of multiple myeloma and Burkitt's lymphoma to broviac catheter sites.[7] Both patients in this series died from metastasic disease. With the increasing role of these access devices in the care of the oncology patients, further clinical presentation of this complication can be expected in the future.

Management should consist of biopsy of the site, if tumor implantation is suspected. When tumor is found, the venous access device should be removed. The patient should be treated for metastatic disease in the appropriate fashion.

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Visual hallucinations as grief reaction without the Charles Bonnet syndrome

TO THE EDITOR: Visual hallucinations as grief reaction can occur in the absence of the Charles Bonnet syndrome. The latter involves hallucinations in the elderly with sensory deprivation such as ocular pathology but without indications of "a dementiform process." Patel, Keshavan, and Martin[1] described the Charles Bonnet syndrome in an 86-year-old woman. Alroe and McIntyre[2] described its occurrence in three women, two of whom were 75 and one 60 years old. All episodes occurred following the death of the patients' spouses. Adair and Keshavan[3] noted its appearance in an 85-year-old woman after the death of her son. The neurologist and neuropsychiatrist S. Weir Mitchell (1829–1914) described his own experience at age 63. He suffered no cerebral or ocular pathology. He hallucinated the face of his close friend, clergyman Philips Brooks, over a ten-day period following the shock of suddenly learning about his death.[4] The present case report involves a middle aged, unmarried woman whose visual hallucinations as grief reaction were not associated with the Charles Bonnet syndrome.

Case Report. A 41-year-old woman undergoing psychotherapy for panic attacks occurring during travel by train revealed that her father had suddenly died of a heart attack nine months previously. She had been closely attached to him and regretted that their contact had not been more frequent despite weekly telephone conversations. She disclaimed any connection between her father's death and the panic attacks that started three months afterwards.

During the six weeks following the death, when walking through a park her father had frequented, she suddenly and clearly saw him sitting on a bench as had been his custom. Three such episodes occurred. She stopped, looked at him, turned, walked away briefly, looked back, and found him gone. Her reaction was one of "sadness." She realized at the time that the visions were hallucinatory and avoided that path thereafter. Two months prior to her account in therapy she had had a general physical examination with normal findings and a routine ophthalmological examination which revealed normal visual acuity and no ocular pathology. Her visual hallucinations evidently constituted a compensatory attempt to cope with a significant sense of loss.

Discussion. This clinical observation and Mitchell's experience probably indicate more widespread occurrence of visual hallucinations in the absence of the Charles Bonnet syndrome. They raise the question of the frequency of such hallucinatory experience in individuals, with and without the syndrome, as well as their appearance and significance at times as grief reactions.

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Benzodiazepines: Prescribing declines under triplicate program

The addition of benzodiazepines to the New York State Triplicate Prescription Program has resulted in a 27–53% decrease in the number of benzodiazepine prescriptions written since the law took effect January 1, 1989. This is the first of a two-part series describing the decrease in prescriptions, as well as the effect on substitute drug prescriptions, physician participation, pharmacy practices, drug abuse emergency room incidents and diversion.

Triplicate prescriptions have been required by the New York State Department of Health (DOH) for the past 11 years for the prescription of all Schedule II drugs, such as narcotics, barbiturates, and amphetamines. These drugs must be prescribed on a New York State Triplicate Prescription. One copy of the triplicate prescription is forwarded to DOH. The pharmacist and prescribing physician retain the other copies. On January 1, 1989, benzodiazepines were added to the triplicate program, becoming the first Schedule IV drugs to be added to the program.

Under the Triplicate Prescription Program, except for certain approved conditions, prescriptions can only be written for a maximum 30-day supply; refills are not permitted. Patients must receive a new prescription for additional drugs, although continued office visits are not required and are left to the physician's judgment in accord with sound medical practice.

New York State's experience with the program has demonstrated that abuse and misuse of licit controlled substances are reduced and prevented while appropriate use remains unaffected. Analysis of the program information shows physician and pharmacy participation has increased.

Benzodiazepines, used primarily for the short term treatment of anxiety, insomnia, and muscle spasms, have also been found to produce dependency with long term therapeutic use. These drugs are also found to be widely abused, diverted into illicit use by some practitioners, and involved in high levels of overdose emergency room admissions. Research has further indicated that patients taking benzodiazepine experience impaired driving skills, are at elevated risk for accidents and increased hip fractures among the elderly and, for long term users, cognitive impairment.

Methodology

The primary objectives for inclusion of the benzodiazepines into the Triplicate Prescription Program were to reduce the diversion of these drugs into illicit use and to reduce inappropriate prescribing that can compromise patient well being. In addition, the objectives were to be accomplished without interrupting legitimate use.

In order to measure the success in achieving the objectives, benzodiazepine prescribing practices were analyzed both pre- and post-regulation (January 1, 1989). Benzodiazepine prescribing practices were evaluated by analyzing prescription claims and payments processed through the following third party payor sources:

- Medicaid, a federal, state, and locally supported program that provides health care to the poor. Administered by the New York State Department of Social Services, Medicaid serves 1.2 million New Yorkers annually.
- Empire Plan, which provides insurance for over 423,000 state and local government employees, retirees, and dependents. Administered by the State Department of Civil Service, the pharmacy portion of the plan is carried by a private company.
- Elderly Pharmaceutical Insurance Coverage (EPIC) Program, a relatively new and growing state cost-sharing program that provides prescription drug insurance to more than 70,000 low- and moderate-income individuals aged 65 or older.

The Medicaid, Empire Plan, and EPIC programs were used to provide an analysis of the impact of the regulation because these programs offer an opportunity to compare prescribing patterns both before and after the triplicate requirement took effect.

In the Medicaid and Empire Plan analyses, a quarterly claims average was calculated from the four quarters of 1988 and from the two quarters of 1989. The averaged quarter of 1988 was then compared to the averaged quarter of 1989. In the Medicaid and Empire Plan, first and second quarter 1989 data include claims paid for prescriptions filled prior to the regulation's implementation and claims for refills of prescriptions written in 1988. Subsequent quarters will be less affected by late claims and refills.

For the EPIC program, data were similarly averaged from three quarters in 1988, but compared individually to each of the first two quarters of 1989. Because the number of EPIC participants has been growing steadily, changes in the percentage of participants with claims and the percentage of all claims that were for benzodiazepines were assessed, rather than comparing numbers of claims.

Findings

Medicaid claims for benzodiazepines fell 55%, from the average number of claims in 1988 to the average number of claims for the first half of 1989. The average number of Medicaid benzodiazepine claims per quarter in 1988 was 389,250 as compared to 173,000 in 1989 (Fig 1).

In addition, a special review was conducted of all Med-

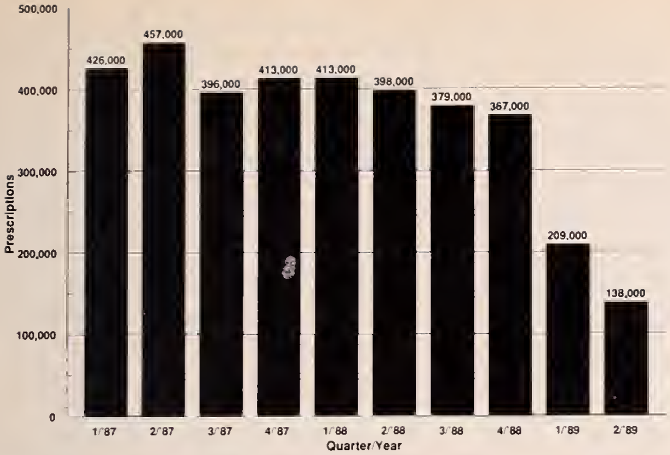

FIGURE 1. Prescriptions for benzodiazepines, paid by New York State Medicaid (data derived from MR-53 Quarterly Reports, NYS Department of Social Services).

icaid recipients who used more than $500 in pharmacy services in at least one month between September and November of 1988. This group was selected and monitored because of its predisposition to divert prescription drugs into illicit use. Of the approximately 6,000 people in this high volume pharmacy use group, an average of 3,439 had benzodiazepine prescriptions filled during the last quarter of 1988. As shown in Figure 2, the number of prescriptions for September 1988 for these benzodiazepine users was 19,200. This number increased slightly to 19,900 in October and then began to decrease. By May 1989, the number of monthly prescriptions had declined to 1,070, a drop of 95% from the number in October 1988. In addition, the number of individuals in this group receiving benzodiazepine prescriptions declined from 3,609 to 763, a 79% reduction between October 1988 and May 1989.

The average number of benzodiazepine prescriptions written for participants in the Empire Plan in the first half of 1989 decreased by 27% from the average number in 1988 (Fig 3). The average number of benzodiazepine prescriptions written quarterly in 1988 was 70,625 compared to 51,450 in 1989 quarters.

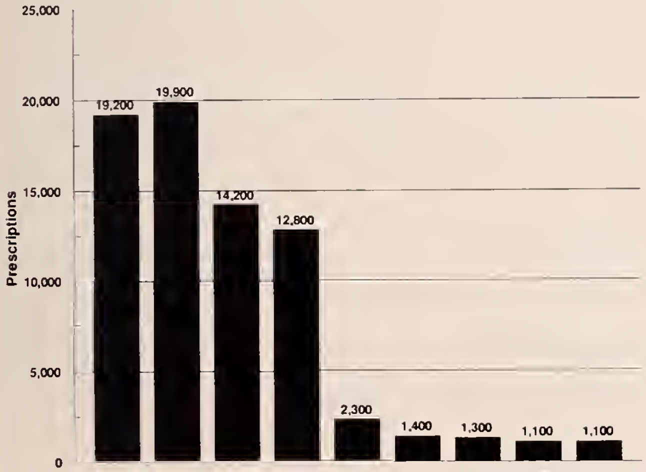

FIGURE 2. Recipients with high volume of pharmacy services benzodiazepine prescriptions, paid by New York State Medicaid (data from NYS Department of Social Services).

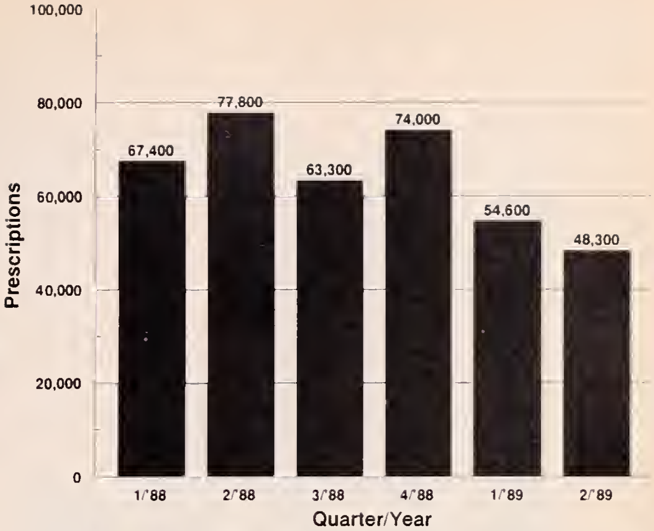

FIGURE 3. Prescriptions for benzodiazepines under the Empire Plan.

While the total number of program participants with drug claims in the EPIC program rose by 47% between the second quarter of 1988 and second quarter of 1989, the number of participants with benzodiazepine claims fell by 23%. As a percent of all claims, benzodiazepines fell from 5.4% in the second quarter of 1988 to a low of 3.2% in the second quarter of 1989, a 41% decrease. While the percentage of EPIC participants receiving benzodiazepine prescriptions each quarter had remained constant at 21% throughout 1988, it dropped to 13.8% by the second quarter of 1989 (Fig 4).

Not All Benzodiazepines Affected Equally

While data from Medicaid and the Empire Plan showed benzodiazepine prescriptions declined overall, not all members of this family of drugs were affected equally. For example, the drug clonazepam, which is used to treat epilepsy, did not experience the declines seen in other benzodiazepines. Under the Medicaid program, clonazepam prescriptions in the first and second quarters of 1989 were within 2% of the average quarter for 1988. In the Empire Plan, the quarterly average of clonazepam prescriptions increased an average of 12.5% compared to 1988. However, Medicaid data indicated that Valium prescriptions were reduced since January 1, 1989, more than any benzodiazepine, down 65% per average quarter in 1989.

Physician Participation

In the first seven months of 1989, data indicate that 25,942 physicians (of the approximately 30,000 physicians in private practice in New York City)[1] actively used the Triplicate Prescription Program. This represents an increase of approximately 50% compared to all of 1988. During the same period, the number of health care institutions with physicians using the triplicate prescription forms rose from 323 to 785, a 41% increase.

Discussion

Benzodiazepines are one of the most frequently used groups of drugs in the state and nation. The addition of benzodiazepines to the 11-year-old Triplicate Prescription Program has produced a decrease, ranging from 27%

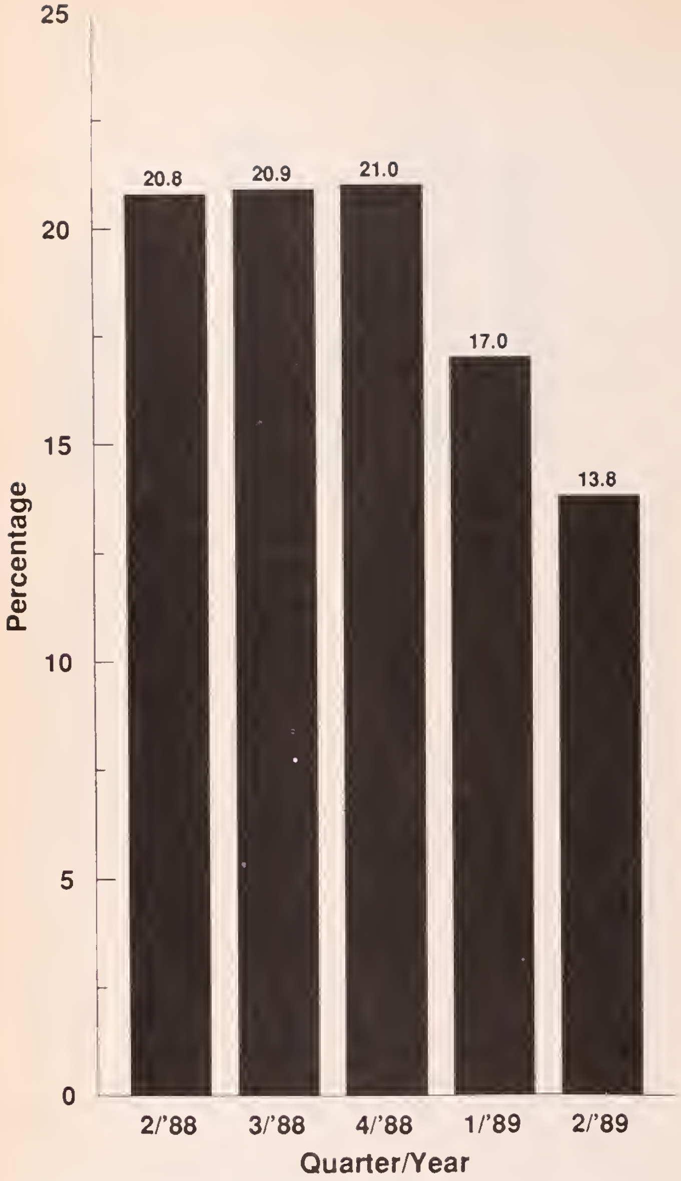

FIGURE 4. EPIC Program participants with benzodiazepine claims.

to 55%, in benzodiazepine prescriptions from state and private insurance and social welfare programs.

Data from the first six months of the program show the most sizable drop in prescribing within the Medicaid program where diversion of drugs for profiteering and drug abuse has long been suspected. Benzodiazepine prescribing for other populations, where abuse and diversion are considered to be less of a problem, also declined. Goals of adding the benzodiazepines to the triplicate program included not only cutting diversion and abuse, but also educating physicians and patients about the risks associated with these drugs, as a means of reducing unnecessary and inappropriate use. The decrease in prescribing found in the three groups studied may reflect decisions by patient and physician alike that other modes of therapy may be preferable. Medicaid data on high-volume users of pharmacy services show the clear impact of the triplicate program on eliminating diversion.

While EPIC data on the elderly's use of benzodiazepines are limited, they do offer an encouraging picture of what may be happening as a result of the new regulation among this particularly vulnerable population. Recent articles have indicated that as a group, seniors face special risks from these drugs and may often receive them inappropriately or for excessive lengths of time, increasing the likelihood of dependence.[2-4]

As has been the experience with other drugs on the program, there has not simply been an across-the-board decrease for all products. Some medications, particularly those known to be diverted or abused, such as Valium, have been more dramatically impacted than others such as clonazepam.

While anecdotal reports of physicians refusing to write benzodiazepine prescriptions have been received by the department, the data suggest that, as a group, physicians have responded appropriately to the new regulation. Prior to the addition of benzodiazepines to the triplicate system, only Schedule II drugs, with limited, specialized medical uses, required a triplicate form and only 58% of private practitioners used triplicates. The benzodiazepines, Schedule IV drugs, have a much broader medical use and the data indicate 86% of private practitioners are now using triplicate prescriptions.

Early data on the impact of the addition of benzodiazepines to the program show the regulation has reduced diversion and abuse, and positively affected prescribing practices. As a public health initiative, the impact of those changed prescribing practices on reducing abuse, diversion, unnecessary utilization, and iatrogenic injury must continue to be followed closely in the months and years ahead.

The January 1990 issue of *Epidemiology Notes* will contain information on the effects of the change in benzodiazepine prescribing practices. Data will be presented from a survey of New York State pharmacies and from an analysis of drug abuse-related emergency room visits. Patterns of substitute drug prescriptions pre- and post-regulation will also be included.

For further information regarding the Triplicate Prescription Program, contact the Division of Public Health Protection, (518) 474-3276.

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M.D.: ONE DOCTOR'S ADVENTURES AMONG THE FAMOUS AND INFAMOUS FROM THE JUNGLES OF PANAMA TO A PARK AVENUE PRACTICE

By B. H. Kean, MD, with Tracy Dahlby, 403 pp. New York, Ballantine Books, 1990. $19.95 (hardcover)

B. H. Kean is the doyen of clinical tropical medicine in America. That alone should alert readers that his autobiography stands well apart from others of the medical autobiography genre. However, it is not only the specialty of tropical medicine that sets Dr Kean apart, but more importantly the man and physician he is. He would be a distinguished medical figure against the canvas of any age because of his intellectual curiosity, insights, dedication, scientific acumen, and sense of caring. He is the type of physician many young medical students dream of becoming, the doctor most of us would like to have at our bedside in a time of serious illness. That his specialty brings him into contact with unusual problems and famous people adds to the lure of his tale. But it is the persona of Dr Kean, the man and doctor, which makes the book rise into a special class occupied by Victor Heiser's *An American Doctor's Odyssey* and Milton Slocum's *Manhattan Country Doctor.*

Dr Kean was trained in both pathology and tropical medicine at the Gorgas Hospital in the Canal Zone after graduating from Columbia University's College of Physicians and Surgeons. He eventually returned to New York City, began private practice, and became affiliated with Cornell University Medical Center-New York Hospital where he is currently Clinical Professor Emeritus of Tropical Medicine and Public Health. Over the years he not only built up a practice but also distinguished himself in his specialty as a clinical scientist and medical educator.

Tropical medicine is often viewed as irrelevant and boring to some medical students and confusing to many because it is easy to become muddled in parasitic life cycles. That it became one of the most popular courses for students at Cornell reflects on Dr Kean's skills as a teacher. His lectures are legendary to those who have heard them. Thousands of physicians have been taught by him and have carried away vital knowledge of unusual infections which they might otherwise overlook.

The scientist and teacher do not figure in this autobiography. Yet they prominently define Dr Kean to his colleagues and students. What is highlighted are Dr Kean's experiences as a clinical practitioner managing unusual medical problems and equally unusual people. Readers are treated to exciting, page-turner accounts of a number of his patient encounters. Among his patients were both the famous and the infamous as stated in the subtitle of the book. The celebrities include Oscar Hammerstein, Gertrude Lawrence, Edna Ferber, and Salvador Dali, who expectorated with panache on the waiting room carpet. Four chapters are devoted to the Shah of Iran who was Dr Kean's patient during his final illnesses.

Dr Kean is a wonderful raconteur who leavens his tales with humor, seasoned insight, and a scientist's bent for fascinating detail. His stories are sometimes humorous, at times sad, and always spellbinding. Woven through them all as subtle nuances are the author's humanity, dedication, and dogged determination to find both cause and cure. There are enough experiences in this book to sustain several autobiographies. Yet readers like this reviewer will hope that Dr Kean will provide more.

PASCAL JAMES IMPERATO, MD
Editor

ILLUSTRATED GUIDE TO THE CENTRAL NERVOUS SYSTEM

By Kazuaki Sugiura, MD, Grant A. Robinson, PhD, and Douglas G. Stuart, PhD. 160 pp, illustrated. St Louis, Mo, Ishiyaku EuroAmerica, Inc, 1989. $24.50 (softcover)

This book by Sugiura, Robinson, and Stuart was originally designed by the senior author as a refresher and primer for medical students of the brain's structural and functional relationships. Overall it is a concise, general text with easily interpretable figures. The illustrations in the initial chapters are simple and effective. Particularly useful are figures which correlate osseus and vascular anatomy with radiologic images, such as Figures 11–18 and 32–41. Chapter 6, "Cranial Nerves and Their Functions," provides a concise introduction to the cranial nerves, although Figure 95 would benefit from a figure legend. Figures 98A and B are praiseworthy attempts to understand the rostro-caudal relationships of various cranial nerve nuclei.

Unfortunately, an inordinately long (pp 52–65) discourse on pneumoencephalography is included. It is fair to say that this information belongs in the archives of the history of neurology and not in an up-to-date compendium of clinical neuroanatomy. Conspicuously absent in the section on the spinal cord is any mention of myelography. Although computerized axial tomography is nicely correlated with brain topography and representative pathology, magnetic resonance images are absent. It is unfortunate that the translating and editorial efforts that went into this publication were eclipsed by the fast paced technological changes in diagnostic neurology. Systems neuroanatomy is treated superficially, with only three diagrams (Figures 121, 124, and 125) of major descending and ascending pathways.

The only true error I found in the text related to the description of Hoffman's sign (p 128) as being a "pathologic sign." This is a sign of hyperreflexia, but not pathologic hyperreflexia. Section II, "Pathologic Physiology," is scant, minimally discussing intracranial pressure and the assessment of level of consciousness and coma. This section provides no rationale or motivation for laboring through the topographic and systems organization of the brain and spinal cord.

New students of neuroanatomy will easily grasp the major concepts of brain organization, but will be shortchanged in the understanding of systems neuroanatomy and pathways. Experienced students and neurology house officers will be frustrated by the numerous gaps in detailed neuroanatomy required for accurate neurologic diagnoses.

This book should be compared with Manter and Gatz's *Essentials of Clinical Neuroanatomy and Neurophysiology* and Alpers and Mancall's *Essentials of the Neurologic Examination.* The former provides clinically useful neuroanatomy and physiology and correlates pathology. The latter teaches the reader to conduct a neurologic examination and correlates signs of dysfunction with neuroanatomy. My money would go toward the purchase of either or both of these books to replace my aging editions.

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DRUGS AND THE ATHLETE

By Gary I. Wadler, MD, and Brian Hainline, MD. 353 pp, illustrated. Philadelphia, Pa, F. A. Davis Company, 1989. $45.00 (hardcover); $29.95 (softcover)

This book is a largely successful attempt to provide physicians with a comprehensive but readily digestible text on drug abuse in athletics. Both of the physician-authors are affiliated with Cornell University Medical College, and Dr Wadler is Tournament Physician for the US Open Tennis Championships and a Fellow of the American College of Sports Medicine. The style of writing is concise and straightforward, and the numerous case studies that are included give the text a distinctly "clinical" taste that will appeal to most physicians.

The book is divided into three main sections plus an extensive appendix (83 pages) that reviews the drug policies and position statements of a wide variety of sports organizations. The initial three chapters (52 pages) of the book are devoted to a general discussion of the history of drug abuse by athletes and the societal, psychological, physiological, and performance-related factors that can lead to drug problems. This is the weakest section of the book, because it is the most superficially treated. The next 13 chapters (125 pages) of the text provide succinct reviews of specific drugs abused by athletes, and the final four chapters (66 pages) provide a unique and helpful overview of how clinicians can recognize, detect, and manage drug abuse by athletes in their care while minimizing potential litigation.

The main strength of this text is that it is written for the busy physician who wants a comprehensive clinical approach to the topic. It provides few details of original papers on drug use and relies quite heavily on references from medical newsletters, newspapers, and news magazines. These characteristics will be seen as shortcomings for those who seek a more exhaustive and careful scientific analysis of the original research. As one example of what might be viewed as superficial treatment of a topic, less than one full page of text is devoted to the use of vitamins and minerals by athletes. To their credit, the authors have included in their reference lists many citations as recent as 1988. They have also chosen an effective organizational structure for each of the review chapters on individual drugs or drug classes. These chapters begin with historical comments and proceed through drug chemistry, physiology, and clinical pharmacology, to use of the drug in sports and its adverse effects.

This book will be especially valuable to those physicians who have had little exposure to sports medicine and need a concise and well-written overview of drug abuse in athletics to better address the needs of their patients.

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EXERCISE AND SPORT SCIENCES REVIEWS

Volume 17. Edited by Kent B. Pandolf, PHD. 569 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1989. $46.95 (hardcover)

This book is an annual publication of the American College of Sports Medicine which reviews current research in the fields of behavior, biomechanics, clinical issues, physiology, and rehabilitation as they pertain to exercise science. The editorial board is made up of 12 well-known authorities, each of whom is responsible for a topic. The objective of the college is to provide reviews of the latest investigations in broad areas of interest to clinicians, educators, exercise scientists, and students.

The editor of the book, Kent B. Pandolf, PHD, has impeccable credentials, as do the members of the editorial board. They are all distinguished, well-published scientists who have made many contributions to their fields.

This book is a recommended addition to the medical literature for several reasons. First, it is written for the clinician and the research scientist alike. The topics are chosen and presented in a format that has wide variety and appeal, and can be applied in private practice or serve as a catalyst for further investigation. It also allows the busy clinician the opportunity to expand his knowledge in exercise science.

The book opens with a chapter on the application of molecular biology in exercise physiology. The first section provides some background and defines some technical terms used in molecular biology. The second section gives the results of research employing molecular biological technique questions in exercise physiology. The third section discusses potential future applications of molecular biology within exercise physiology. The second chapter, "Metabolic and Physiological Factors in Fatigue," examines and discusses many studies reported in the literature on factors contributing to human skeletal muscle fatigue. Chapter 3, "Skeletal Muscle Disorders and Associated Factors That Limit Exercise Performance," primarily deals with the general metabolic and physiologic factors which set an upper limit to performance in patients with a variety of specific disorders affecting the metabolism or structure of skeletal muscle. Chapters 4, 5, and 6 address three key areas of muscle physiology: lactate uptake, eccentric action of muscle, and determining muscle's force and action in multiarticular movement.

In chapters 7 and 8, two aspects of hemodynamics and exercise are presented. They deal with impedance cardiology: non-invasive assessment of human central hemodynamics at rest and during exercise, and acute polycythemia and human performance during exercise and exposure to extreme environments. The next chapter, "Physiological Interactions Between Pregnancy and Aerobic Exercise," is a popular subject, and it is increasingly important for clinicians to have the latest up-to-date information. Many of today's young women and soon-to-be mothers have been brought up with exercise and wish to continue it through their pregnancy. Physicians must have a good understanding of the effects of exercise on these women, and this chapter does a good job of presenting some of the key issues within this topic. Chapter 10 addresses growth hormone, its therapeutic use, and the potential for abuse. Chapter 11 reviews exercise-induced stress fractures and stress reactions of bone. It discusses epidemiology, etiology, and classifications.

Chapter 12 discusses physical activity, exercise, and epidemiology; giving epidemiology as part of exercise science. Chapter 13 is a review of motor skill acquisition and performance. The book concludes with a chapter titled "The Tradition of the 'Six Things Non-Natural': Exercise and Medicine from Hippocrates through Ante-Bellum America."

The quality of the book is excellent. It is highly accurate, with information being drawn from the latest literature. In addition, it is very well organized and presented in a clear and concise manner. As a part of the American College of Sports Medicine series, this book's style and technical features meet very high standards.

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MODERN PERSPECTIVES IN THE PSYCHIATRY OF THE NEUROSES

Edited by John G. Howells, MD. 384 pp. New York, Brunner/Mazel Publishers, 1989. $50.00 (hardcover)

This book, according to its editor, John G. Howells, MD, former director of the Institute of Family Psychiatry and Professor of Psychiatry at Cambridge University, "is planned to bring the facts from the growing points of psychiatry...to the attention of the clinician." In his attempt to do this Dr Howells has assembled a prestigious group of MDs and PhDs who have, for the most part, done a very worthy job in summarizing recent information on the neuroses.

The book contains 19 chapters with very creditable bibliographies that discuss various aspects of neurotic illness, including eating disorders, panic attacks, sleep disorders, post-traumatic stress disorder, and various treatment strategies in addition to other topics. A number of well-respected authors were assembled to present specific topics. Dr John Oldham, Associate Chairman, Department of Psychiatry at Columbia University College of Physicians and Surgeons, presents a chapter on the "Diagnosis of the Neuroses," in an exceedingly clear and precise manner. His expertise in this area of psychiatry and his very effective writing skills help the reader to gain an appreciable knowledge of this subject area.

Dr Peter Buckley, Professor of Psychiatry at Albert Einstein College of Medicine, has written a chapter on an often neglected area of psychiatry, "Supportive Psychotherapy." The chapter, although short, presents a great deal of pertinent information that the clinician should find helpful in his therapeutic endeavors.

Dr Walter V. Flegenheimer, of the Beth Israel Medical Center in New York, has written a very worthwhile chapter on "Brief Psychotherapy." He very aptly summarizes present thinking in this treatment area.

Other prominent figures who have written excellent chapters include D. P. T. Brown, Director of the Marylebone Consultancy Unit, London, who has co-authored "Psychological and Organic Factors in Sexual Dysfunction," and James C. Ballenger, MD, Chairman of the Department of Psychiatry at the Medical College of South Carolina, who co-authored the chapter on "Psychopharmacology of the Neuroses." Both of these scientists, in addition to the other authors, have done an admirable job in clearly summarizing the cutting edge issues concerning neuroses for the clinician.

Clinicians who wish to update themselves on the new therapeutic strategies concerning the neuroses, and to obtain a clear and basic understanding of them, would very much benefit by having a book of this caliber.

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A HANDBOOK OF PSYCHIATRIC NURSING

Edited by Lloyd A. Wells, MD. 423 pp. St Louis, Mo, Warren H. Green, Inc, 1989. $47.50 (hardcover)

This book is an unmemorable addition to the medical literature in general, and to the psychiatric nursing literature in particular. Doctors interested in understanding a nurse's perspective as well as nursing students and practicing nurses will be much better served by other competent books available. *Essential Psychopathology* by Jerrold Maxmen (Norton Press, 1986); *Principles and Practice of Psychiatric Nursing* by Gail Stuart and Sandra Sundeen (Mosby, 1987); and *Psychiatric Nursing* by Holly Wilson and Carol Kneisl (Addison-Wesley, 1988) are all good bets.

A Handbook of Psychiatric Nursing has many problems. The first is the self-declared mission of the book. Although Dr Wells' introduction states that the book is not a text, the structure of the book's 32 chapters suggests otherwise. Some of these chapters, including two entitled "Neuroses" and "Nurses and Psychoses," are reminiscent of textbooks from an earlier era. Others dealing with, for example, psychopharmacology, electroconvulsive therapy, schizophrenia, psychoanalytic theory, and emergencies are standard textbook fare. Although the book purports to be interdisciplinary, 90% of the authors are either physicians or nurses. Dr Wells wrote 17 of the chapters and his wife was the author of three.

The book falls short in treating topics it has chosen to address and much of the material is dated. Dr Leonard Stein's classic article "The Nurse-Doctor Game" was first published in 1967 and is reprinted with permission from the *Archives of General Psychiatry*. It is still fun to read, but 23 years later the acute nursing shortage and the women's movement have changed some elements of the "game." An update would have been welcome and more than justified. Instead, the chapter leaves the reader without any new insights into the problem.

Dr Wells also clearly disagrees with the new classification of schizophrenia that came with the 1980 edition of DSM III and prefers Bleuler's "four A's." While Dr Wells is not alone in his position on DSM III or DSM IIIR, he does not present any good rationale for his disagreement.

Lack of clarity is another failing. For example, the essential elements of suicidal assessment are not clearly delineated. And frequently when the basics of some aspect of practice are presented, the book's treatment remains far too simplistic. Although the principles of beginning safe practice are critical, especially for nursing students and recent nursing graduates, the book fails to provide enough detail to be helpful.

Equally or perhaps more glaring among the book's deficiencies are its omissions. With substance abuse in the United States at epidemic levels, the book devotes a mere three and a half pages to chemical dependency, constituting for the most part a few kind words about Alcoholics Anonymous. Also, other current issues are nowhere to be found. For example, no material appears on AIDS dementia, eating disorders, the homeless mentally ill, dual diagnosis, cognitive therapy, or multiple family therapy. Missing from an otherwise well-written chapter on the role of the nurse in community mental health is any mention of deinstitutionalization's impact on the chronically mentally ill.

In summary, the book has few redeeming characteristics and therefore readers are advised to look elsewhere.

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BLACK FAMILIES IN CRISIS: THE MIDDLE CLASS

Edited by Alice F. Coner-Edwards, DSW, and Jeanne Spurlock, MD. 320 pp. New York, Brunner/Mazel Publishers, 1988. $30.00 (hardcover)

The goal of this book is to point out particular issues that negatively affect the mental health of the black middle class family. The book grew out of a discussion group of black psychiatrists who were involved in a symposium at Howard University in 1985 entitled "The Black Families in Crisis." The authors include physicians, social workers, and a medical student, who contribute experiences from their professional lives.

The book focuses on some of the characteristic experiences and problems of the black family. It also points out how the change to so-called "black middle class" has its own sets of problems and how stress and racial prejudice are perceived from a black perspective.

I found this book to be very informative and believe it should be included in the libraries of physicians who care for black patients. For physicians with different racial and cultural backgrounds, this book offers an insight into the black family. The book covers many issues, including problems encountered in the workplace, male/female relationships, parenting, care of the elderly parent, educational and health issues, and treatment. The book is very well written. However, I found that it was too superficial in its coverage. The book was not written with the purpose of giving in-depth insight into the topics discussed, although there is an extensive bibliography at the end of each chapter. However, each chapter provides enough information so that any physician working with black families would be made cognizant of issues that impact upon a black person.

The book is well organized, concise, and gives statistical data to support the issues discussed. It quotes many nationally known authors and reports. Each chapter opens with an overview on the objectives to be met or points to be explored. It is straightforward reading, easily understood by professionals and lay persons.

I am not aware of any other publication that presents the black middle class of the 1980s in the same context as this book does. I believe the book's authors have achieved their objective of showing that economic/financial stability is not without emotional problems. The authors also emphasize the need for families to utilize therapeutic intervention when problems are recognized.

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LEGAL ASPECTS OF MEDICINE

Edited by James R. Vevaina, MD, Roger C. Bone, MD, and Edwin Kassoff. 340 pp. New York, Springer-Verlag, Inc, 1989. $73.00 (hardcover)

Drs Vevaina and Bone have impressive credentials in the fields of pulmonary and critical care medicine. While this book is intended to be of interest to all who work in the fields of law and medicine, particular emphasis is given to the fields of cardiology, pulmonary medicine, and critical care medicine. The third coeditor, Edwin Kassoff, is a presiding justice of the appellate term, 2nd and 11th Judicial Districts, of the Supreme Court of the State of New York; and is an experienced justice, writer, and lecturer. Dr Vevaina sparked the creation of the book because almost all physicians appear to be "deeply troubled by legal problems, professionally, economically, and most important of all, psychologically." His objective is to make every doctor better equipped to deal with medicolegal problems by being exposed to a "blend" of great minds in law and medicine.

The text is not a comprehensive or encyclopedic approach to law and medicine and by design was not intended to be. For the most part, each chapter is a succinct discussion designed to help physicians become more comfortable with the interface between law and medicine. The book contains 35 chapters written by various experts in both law and medicine, and covers a multitude of subjects ranging from the evolution of medical malpractice law, discussed by Melvin M. Belli, to the legal aspects of intensive care, airway disorders, lasers, HIV testing, respiratory therapy, lung disease, cancer, arterial catheterization, heart and lung transplantation, withholding and withdrawing life support, and finally the psychological effects of medical malpractice.

While the overall quality of the writing is good, in my opinion, certain chapters are noteworthy; eg, "Regulatory Law as It Affects Physicians" (S. A. Robert), "Risk Management for the Physician (M. S. Kaminski, et al), "Ethics, Medicine and the Law" (G. A. Kanoti), "Mysteries Unraveled by Postmortem Examination" (C. H. Wecht), and "Legal Aspects of Human Immunodeficiency Virus (HIV)" (H. M. Ginzburg, M. J. Rosen). In conclusion, even though this book is heavily weighted toward the legal aspects of cardiopulmonary and critical care medicine, physicians who read it will be better informed about overall legal aspects of medicine, and ultimately will recognize that good medicine is good law.

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Report of a specific and sensitive test for Lyme disease: Detection of a single organism is possible

Lyme disease is a multisystemic, potentially debilitating illness caused by the spirochete *Borrelia burgdorferi* and transmitted by infected ticks of the species *Ixodes ricinus*. In some persons, the initial sign of a bite by an infected tick is a skin rash, erythema migrans. However, in many cases the bite goes undetected, and subsequent diagnosis of Lyme disease is difficult, as its symptoms are common to a broad spectrum of ailments. Identification of Borrelia spirochetes has been an obstacle in rapid diagnosis because infection can occur with the presence of only a few organisms.

Rosa and Schwan have reported their success in using the polymerase chain reaction (PCR) to amplify and detect a small amount of *B burgdorferi* DNA in human tissue (*J Infect Dis* 1989; 160:1018–1028). Theoretically, this technique could detect a single organism. In PCR, DNA-specific enzymes copy a target DNA sequence, achieving amplification of more than one million times within several hours.

The initial goal of Rosa and Schwan was "to identify a sequence of DNA present in all strains of *B. burgdorferi* but not conserved in the most closely related member of the genus, *Borrelia hermsii*." They then amplified this DNA sequence to detectable levels. Further testing demonstrated that a small amount of *B burgdorferi* DNA was identifiable even "in the presence of a large excess of human DNA."

Although the initial studies were performed with purified DNA, the authors showed that the PCR assay worked effectively with crude lysates of *B burgdorferi*, thereby eliminating the need for labor and time intensive sample preparation. Rosa and Schwan do emphasize the importance of using appropriate negative controls when performing this highly sensitive assay.

Use of multivitamin/folic acid supplements during first weeks of pregnancy can help prevent neural tube defects

A startling conclusion: "We believe that the combined data from this and other studies provide good evidence that folic acid-containing MVs [multivitamins] taken during the first 6 weeks of pregnancy will prevent, by more than 50%, the occurrence of NTDs [neural tube defects]" (*JAMA* 1989; 262:2847–2852). Milunsky et al based this statement on data accumulated from previous studies and their own study, which involved a prospective evaluation of the relationship between maternal multivitamin usage and, specifically, folic acid intake from multivitamin and dietary sources, and the risk of neural tube defects among the children of 22,776 women.

All of the women who participated in the study underwent amniocentesis or serum alpha-fetoprotein determination at about the 16th week of pregnancy. All participants also completed an interview that produced information on "family, medical, and genetic history with special emphasis on diet, medication, and illness during the first trimester of pregnancy. Questions also covered environmental and occupational exposures prior to conception." The women reported on their use of vitamins and other nutritional supplements. Pregnancy outcome information was obtained from either the delivering physician or the woman herself.

For the purposes of this study, neural tube defects included spina bifida, anencephaly, or encephalocele. A deficiency of folic acid has been suspected as a contributing factor in the development of neural tube defects for many years. Previous research has demonstrated reduced levels of folic acid at term in the blood of women who had babies with NTDs. Furthermore, aminopterin, which is an antagonist of folic acid, is known to be teratogenic in humans and has been shown to cause NTDs in animals.

Forty-nine of the 22,776 women in this study had babies with NTDs. The authors found a substantially reduced risk of NTDs among the women who took multivitamin/folic acid supplements, at standard prescribed doses, and this protective effect appeared to be limited to use of the supplements during the first six weeks of pregnancy.

Milunsky et al support their belief that NTDs result from a genetic predisposition in combination with a deficiency of an essential nutrient, quite possibly folic acid, with the following finding: "the strikingly higher prevalence of NTDs in women with a positive family history who did not take supplements (13.0 per 1000) compared with those with a family history who did (3.5 per 1000)." The authors point out that the results of this study do not eliminate a role in NTDs for vitamins A, C, D, or E, either alone or in combination with folic acid, because most prenatal multivitamin preparations provide all these nutrients.

Computers aid in record keeping and can lower insurance costs

In Louisville, Kentucky, one malpractice underwriter rewards emergency room physicians who use computers for documenting patient information with a 5–40% reduction in their malpractice insurance premiums (*Wall Street Journal*, December 8, 1989, p B1). According to the company, lack of complete and accurate patient records in support of a physician's actions are an important problem when taking a malpractice case to court. One emergency physician has estimated that "about 35% of malpractice costs result from people whose heart attacks weren't diagnosed by emergency room doctors."

When used properly, computers can assure adequate record keeping. Products now available include computer software that rechecks the records of emergency room patients to determine whether, based on the presenting symptoms, the doctor asked each patient all the necessary and relevant questions. Another data management system evaluates all charts before patients are discharged.

Furthermore, computers can save physicians valuable time spent on record keeping and chart preparation. With one system, which incorporates speech-recognition computers, "a doctor dictates information that appears on the computer screen and prompts the machine to ask ques-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

tions about a patient's symptoms. The computer then makes a chart from the doctor's responses."

Health problems of the homeless

The Baltimore Homeless Study evaluated the health and mental health care needs of the homeless population in Baltimore, Md, during 1986 and 1987. This study differed from much of the previous literature in that the sampling method yielded a population of homeless persons from various settings. The more than 20 sampling sites, from which the subjects were randomly selected, included missions, shelters, and jails—"all the facilities serving homeless adults that were in operation in Baltimore during the period of the study." According to the authors, ". . . in the main, investigations of the physical health of the homeless have been based on treated populations, mostly users of clinics and emergency services, and thus may not describe the homeless in general."

The three-stage design of the Baltimore Homeless Study consisted of a baseline survey, a clinical evaluation, including physical and psychiatric evaluations of half of the baseline survey group, and a six-month follow-up of one third of the participants who underwent clinical evaluations. In a recent issue of *JAMA* (1989; 262:1352–1357), the authors reported their findings from the first two stages of this study.

The baseline survey, given to 528 homeless adults, collected information on demographic variables, personal, health and occupational history, substance abuse, patterns of health services utilization, and barriers to care. The results of the survey indicated that one third of men and less than half of the women had graduated from high school. Most were unemployed at the time of the survey, and about two fifths of the men and half of the women were receiving some form of public support. Arrest histories were present for more than two fifths of the women and four fifths of the men.

Mouth and teeth problems accounted for the greatest number of problems among participants. For women, gynecologic problems were common—found in two thirds of the homeless women examined—with 42% having menstrual or menopausal symptoms, and 34% with genital infections. More than half of the men and women had various skin conditions. The fourth most prevalent health problem affected the cardiovascular system, with approximately one fifth of men and one eighth of women exhibiting evidence of hypertension, and 15% having cardiac arrhythmias. One fourth of the homeless population examined displayed peripheral vascular problems, including varicose veins, venous stasis, ulceration, and inflammation of the lower extremities. Thirty-two percent of women and 26% of men suffered some type of arthritis. Anemia was present in 18% of men and 35% of women.

Both men and women reported heavy smoking, and substance abuse, primarily of alcohol, was a prominent finding in the medical histories of 85% of men and 67% of women. The Short Michigan Alcoholism Screening Test identified 69% of men and 38% of women as probable or definite alcoholics.

With regard to the psychiatric evaluations, 91% of the men and 80% of the women were found to suffer some form of mental illness or substance use disorder. The authors emphasized, however, that, "Contrary to stereotype, the majority do not have illnesses such as schizophrenia or bipolar disorder." Twelve percent of men and 17% of women were diagnosed with schizophrenia, while 19% of men and 24% of women were found to have affective disorders. Less severe mental disorders, such as phobias, panic or anxiety disorders, and personality disorders prevailed.

When asked to name their usual source of health care, only 47% of men and 30% of women could comply. Similarly, there was no third-party payer for health care for 52% of the men and 30% of the women.

The authors concluded that ". . . most of the general health problems of the homeless could be addressed through satisfying basic public health needs: clean and safe living conditions, adequate nutrition, primary care for common conditions, and dental hygiene and routine dental care." Substance abuse, and primarily that of alcohol, is the basis for the majority of disorders among the homeless. The availability of substance abuse treatment services does not begin to meet the growing need. Furthermore, ". . . improving the accessibility and availability of community mental health services for the homeless is more appropriate than advocating reinstitutionalization."

For every suture, a silver lining

Men and women undergoing surgery in the next decade may find themselves bejeweled with silver sutures. Researchers at Cornell University have developed a suture of metallic silver chemically bonded to nylon thread, which demonstrates bactericidal activity against seven types of cultured bacteria that frequently cause wound infections (*Science News* 1989: 136:223).

Passage of a small current, of a few millionths of an amp, through the silver coating enhances the sutures' antibacterial effect. Patients could receive the comparable effect of a continual release antibiotic through a small direct-current generator worn near the body like a portable radio. The next step in product development will be experimentation in mice, with clinical trials in humans anticipated within about five years.

Recall of about ten million generic estrogen tablets

Duramed Pharmaceuticals (Cincinnati, Oh) has recalled a large quantity of its generic conjugated estrogen tablets, citing inconsistencies in tablet formulations (*Wall Street Journal*, December 13, 1989). Duramed, one of the largest manufacturers of generic estrogens in the US, is trying to determine the cause of the inconsistencies and has broached the possibility of an additional recall. It is unlikely that the drug's therapeutic activity or safety was compromised, according to the company.

In 1988, Duramed applied to the FDA for a bioequivalence rating for its product with Premarin™ (Wyeth-Ayerst Laboratories), the brand-name conjugated estrogen with an annual market of approximately $400 million. Pursuit of a bioequivalence rating, which implies that the generic estrogen is absorbed into the body at about the same rate as Premarin, will now be put on hold.

OBITUARIES

Eric G. Altschul, MD, Woodside. Died December 27, 1989; age 78. Dr Altschul was a 1938 graduate of Middlesex University School of Medicine, Waltham, Massachusetts. He was a Fellow of the American Academy of Family Practice. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Robert Augenfeld, MD, Palm Beach, Florida. Died December 13, 1989; age 80. He was a 1933 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a Diplomate of the American Board of Family Practice. His memberships included the American Academy of Family Practice, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Giovanni Autuori, MD, Ridgefield, Connecticut. Died October 29, 1989; age 93. Dr Autuori was a 1920 graduate of Facolta di Medicina e Chirurgia dell' Universita di Palermo, Palermo, Italy. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Delmer E. Batcheller, MD, Phelps. Died November 15, 1989; age 62. Dr Batcheller was a 1951 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the Ontario County Medical Society, and the Medical Society of the State of New York.

Arthur Biezunski, MD, Rego Park. Died December 12, 1989; age 78. Dr Biezunski was a 1939 graduate of the Universite de Paris VI, Paris, France. He was a Diplomate of the American Board of Anesthesiology and a Fellow of the American College of Anesthesiologists. His memberships included the New York Academy of Medicine, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Angelo Robert Bologna, MD, Cranston, Rhode Island. Died November 19, 1989; age 84. Dr Bologna was a 1933 graduate of the Washington University School of Medicine, St Louis, Missouri. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Gerald Brill, MD, New York. Died November 21, 1989; age 62. Dr Brill was a 1949 graduate of Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Internal Medicine. His memberships included the Clinical Society of the New York Diabetes Association, the New York County Medical Society, and the Medical Society of the State of New York.

Leonard Burness, MD, New York. Died November 29, 1989; age 79. Dr Burness was a 1936 graduate of the Georgetown University School of Medicine, Washington, DC. He was a Fellow of the American College of Angiology. His memberships included the New York State Society of Internal Medicine, the American Public Health Association, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Seymour Harvey Cagan, MD, Mineola. Died November 25, 1989; age 61. Dr Cagan was a 1965 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Radiology. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Joseph T. Chiaramonte, MD, Rockville Centre. Died December 19, 1989; age 67. Dr Chiaramonte was a 1945 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Dermatology, Inc. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

David N. Chwatsky, MD, Oceanside. Died December 25, 1989; age 36. Dr Chwatsky was a 1980 graduate of the Facultad de Medicina de la Universidad Autonoma de Guadalajara, Guadalajara, Jalisco, Mexico. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Nicholas Robert Codik, MD, Bayside. Died December 17, 1989; age 82. Dr Codik was a 1934 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Modena, Modena, Italy. He was a Fellow of the American Academy of Family Practice. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Jeff Joseph Coletti, MD, Old Bethpage. Died December 9, 1989; age 79. Dr Coletti was a 1939 graduate of the University of Cincinnati College of Medicine, Cincinnati, Ohio. His memberships included the New York Academy of Medicine, the American Academy of Family Practice, the Nassau County Medical Society, and the Medical Society of the State of New York.

Francis Place Coombs, MD, Old Westbury. Died January 21, 1990; age 75. Dr Coombs was a 1940 graduate of the Cornell University Medical College, New York. He was a Diplomate of the American Board of Dermatology, Inc. His memberships included the New York State Dermatological Society, the American Academy of Dermatology, the Pan American Medical Association, the Nassau County Medical Society, and the Medical Society of the State of New York.

William McIntosh Coon, MD, New York. Died January 25, 1990; age 69. Dr Coon was a 1945 graduate of the University of Illinois at Chicago Health Sciences Center, Chicago, Illinois. He was a Diplomate of the American Board of Dermatology, Inc. His memberships included the New York State Dermatological Society, the American Academy of Dermatology, the Society for Investigative Dermatology, the Pan American Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Emanuel de Lalla, MD, Utica. Died November 13, 1989; age 85. Dr de Lalla was a 1932 graduate of the McGill University Faculty of Medicine, Montreal, Canada. He was a Diplomate of the American Board of Radiology. His memberships included the New York Academy of Medicine, the Radiological Society of New York State, the Radiological Society of North America, Inc, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Eugenio de Marchis, MD, New York. Died October 17, 1989; age 43. Dr de Marchis was a 1971 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Roma, Rome, Italy. His memberships included the American Society of Anesthesiologists, Inc, the New York County Medical Society, and the Medical Society of the State of New York.

Skottowe W. de Pass, MD, Ridgewood. Died January 8, 1990; age 62. Dr de Pass was a 1954 graduate of the Duke University School of Medicine, Durham, North Carolina. He was a Fellow of the American College of Radiology and a Diplomate of the American Board of Radiology. His memberships included the Radiological Society of North America, Inc, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Selden Jerome Dickinson, MD, New York. Died January 24, 1990; age 63. Dr Dickinson was a 1953 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American Academy of Pediatrics, a Fellow of the American College of Surgeons, and a Diplomate of the American Board of Surgery. His memberships included the New York Pediatric Society, the New York County Medical Society, and the Medical Society of the State of New York.

Joel Dlugash, MD, Long Beach. Died December 2, 1989; age 51. Dr Dlugash was a 1967 graduate of the Chicago Medical School, Chicago, Illinois. His memberships included the New York Cardiological Society, the Nassau County Medical Society, and the Medical Society of the State of New York.

Diana Olga Duszynski, MD, Buffalo. Died December 5, 1989; age 72. Dr Duszynski was a 1942 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. She was a Diplomate of the American Board of Radiology. Her memberships included the Society of Nuclear Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Joseph E. Dygert, MD, Pittsford. Died November 17, 1989; age 69. Dr Dygert was a 1958 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. He was a Fellow of the American College of Angiology. His memberships included the New York Academy of Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Walter F. Ehrlich, MD, Baldwin. Died December 25, 1989; age 96. Dr Ehrlich was a 1921 graduate of the Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Berlin, Germany. His memberships included the American Academy of Family Practice, the Nassau County Medical Society, and the Medical Society of the State of New York.

Matthew Elmer Fairbank, MD, Rochester. Died November 25, 1989; age 83. Dr Fairbank was a 1935 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. His memberships included the New York Academy of Medicine, the American Academy of Occupational Medicine, the American Occupational Medical Association, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

James Howard Finkelstein, MD, New York. Died July 22, 1989; age 59. Dr Finkelstein was a 1955 graduate of the New York University School of Medicine, New York. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Charles D. Foster, MD, Binghamton. Died December 2, 1989; age 65. Dr Foster was a 1949 graduate of the Tufts University School of Medicine, Boston. His memberships included the New York Academy of Medicine, the Broome County Medical Society, and the Medical Society of the State of New York.

Marcel Friedman, MD, New Port Richey, Florida. Died September 11, 1989; age 88. Dr Friedman was a 1931 graduate of the Institutul de Medicina si Farmacie, Bucuresti, Romania. He was a member of the Dutchess County Medical Society and the Medical Society of the State of New York.

Iago Galdston, MD, Brooklyn. Died December 19, 1989; age 94. Dr Galdston was a 1921 graduate of the Fordham University School of Medicine, New York. His memberships included the New York Academy of Medicine, the American Psychiatric Association, the Academy of Psychoanalysis, the American Public Health Association, the New York County Medical Society, and the Medical Society of the State of New York.

William John Garvin, MD, Shoreham. Died December 9, 1989; age 72. Dr Garvin was a 1943 graduate of the Temple University School of Medicine, Philadelphia. He was a Diplomate of the American Board of Ophthalmology and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the New York State Surgical Society, the American Academy of Ophthalmology and Otolaryngology, the Pan American Association of Ophthalmology, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Sidney S. Goldensohn, MD, New York. Died December 21, 1989; age 69. Dr Goldensohn was a 1944 graduate of the George Washington University School of Medicine, Washington, DC. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the Academy of Psychoanalysis, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Carl Goldmark, MD, New York. Died January 19, 1990; age 81. Dr Goldmark was a 1933 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Surgeons, a Fellow of the American College of Obstetricians and Gynecologists, and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the New York Academy of Medicine, the Pan American Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Arthur Gordon, MD, Flushing. Died January 17, 1990; age 81. Dr Gordon was a 1932 graduate of the New York University School of Medicine, New York. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

John Francis Grady, MD, Greenwich, Connecticut. Died November 28, 1989; age 87. Dr Grady was a 1929 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Surgeons. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Milton J. H. Grand, MD, Bronx. Died December 29, 1989; age 85. Dr Grand was a 1928 graduate of the New York University School of Medicine, New York. He was a Fellow of the American Academy of Pediatrics, a Fellow of the American College of Physicians, and a Diplomate of the American Board of Pediatrics. His memberships included the New York Academy of Medicine, the Bronx County Medical Society, and the Medical Society of the State of New York.

Esther Greenbaum, MD, Forest Hills. Died December 6, 1989; age 80. Dr Greenbaum was a 1935 graduate of the Universite de Paris VI, Paris, France. Her memberships included the American Psychiatric Association, the Academy of Psychoanalysis, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Paul Hecht, MD, Wyndmoor, Pennsylvania. Died May 1989; age 80. Dr Hecht was a 1934 graduate of the New York Medical College, New York. He was a Fellow of the American Psychiatric Association. His memberships included the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Walter T. Heldmann, MD, Staten Island. Died January 1, 1990; age 86. Dr Heldmann was a 1928 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology, the Richmond County Medical Society, and the Medical Society of the State of New York.

Lewis Jacob Herold, MD, Neponsit. Died December 4, 1989; age 87. Dr Herold was a 1928 graduate of the University of Maryland School of Medicine, Baltimore. His memberships included the American Academy of Family Practice, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Anthony J. Izzo, MD, Rochester. Died December 3, 1989; age 70. Dr Izzo was a 1945 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. He was a Diplomate of the American Board of Internal Medicine. His memberships included the Endocrine Society, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

A. Wilmot Jacobsen, MD, Buffalo. Died December 17, 1989; age 91. Dr Jacobsen was a 1923 graduate of the Johns Hopkins University School of Medicine, Baltimore, Maryland. He was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. His memberships included the New York Academy of Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Abraham Kahane, MD, Bronx. Died December 20, 1989; age 78. Dr Kahane was a 1936 graduate of the New York University School of Medicine, New York. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the New York Academy of Medicine, the Bronx County Medical Society, and the Medical Society of the State of New York.

Alexander A. Katz, MD, Cutchogue. Died December 8, 1989; age 81. Dr Katz was a 1933 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Obstetrics and Gynecology and a Fellow of the American College of Surgeons. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Joseph F. Kenzie, MD, Zephyrhills, Florida. Died November 4, 1989; age 84. Dr Kenzie was a 1929 graduate of the University of Western Ontario Faculty of Medicine, London, Ontario, Canada. He was a member of the Steuben County Medical Society and the Medical Society of the State of New York.

Mansho Tolaram Khilnani, MD, Scarsdale. Died January 4, 1990; age 71. Dr Khilnani was a 1949 graduate of the Calcutta Medical College, Calcutta University, Calcutta, West Bengal, India. He was a Diplomate of the American Board of Radiology. His memberships included the New York Roentgen Society, the Radiological Society of New York State, the New York County Medical Society, and the Medical Society of the State of New York.

Rolf Kroll, MD, Bronxville. Died December 22, 1989; age 76. Dr Kroll was a 1940 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Anthony E. Maniscalco, MD, Greenwich, Connecticut. Died December 25, 1989; age 78. Dr Maniscalco was a 1940 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna, Italy. He was a Fellow of the American Psychiatric Association and a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the New York Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Philip Marshall, MD, Harrisburg, Pennsylvania. Died January 1, 1990; age 84. Dr Marshall was a 1934 graduate of the Hahnemann Medical College of Philadelphia, Philadelphia, Pennsylvania. He was a Qualified Fellow of the International College of Surgeons and a Diplomate of the American Board of Surgery. His memberships included the American Society of Abdominal Surgeons, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Philip Menter, MD, Syracuse. Died December 22, 1989; age 80. Dr Menter was a 1936 graduate of the Universitaet Innsbruck, Medizinische Fakultaet, Innsbruck, Austria. His memberships included the New York Academy of Medicine, the American Occupational Medical Association, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Israel Miller, MD, Brooklyn. Died August 27, 1989; age 83. Dr Miller was a 1929 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American Academy of Pediatrics and a Diplomate of the American Board of Pediatrics. His memberships included the American Public Health Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

John Paul Munteanu, MD, Huntington. Died January 28, 1990; age 41. Dr Munteanu was a 1973 graduate of the Institutul de Medicina si Farmacie, Cluj, Romania. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Joseph Norman Nathanson, MD, New York. Died December 18, 1989; age 94. Dr Nathanson was a 1919 graduate of the McGill University Faculty of Medicine, Montreal, Quebec, Canada. He was a Fellow of the American College of Surgeons and a Diplomate of the American Board of Obstetrics and Gynecology. His memberships included the New York Obstetrical Society, the New York County Medical Society, and the Medical Society of the State of New York.

Leonard Rack, MD, New York. Died January 15, 1990; age 62. Dr Rack was a 1950 graduate of the University of Illinois at Chicago Health Sciences Center, Chicago, Illinois. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Harold L. Rakov, MD, Kingston. Died December 24, 1989; age 88. Dr Rakov was a 1925 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Fellow of the American College of Cardiology, a Fellow of the American College of Physicians, and a Diplomate of the American Board of Internal Medicine. He was a member of the Medical Society of the County of Ulster and the Medical Society of the State of New York.

Sidney Rosenfeld, MD, New York. Died January 8, 1990; age 82. Dr Rosenfeld was a 1932 graduate of the New York Medical College, New York. He was a Fellow of the American College of Physicians, the American College of Cardiology and the American College of Chest Physicians. He was a Diplomate of the American Board of Internal Medicine. His memberships included the New York State Society of Internal Medicine, the American Geriatrics Society, the American Society of Internal Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Sidney Hugh Saffer, MD, Charlottesville, Virginia. Died December 29, 1989; age 78. Dr Saffer was a 1937 graduate of the Faculte de Medecine de l'Universite de Geneve, Geneva, Switzerland. His memberships included the New York State Urological Society, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Anthony Adolf Scimeca, MD, West Islip. Died January 24, 1990; age 90. Dr Scimeca was a 1924 graduate of the University of Maryland School of Medicine, Baltimore, Maryland. He was a Diplomate of the American Board of Ophthalmology. His memberships included the New York Academy of Medicine, the American Academy of Ophthalmology and Otolaryngology, the Pan American Association of Ophthalmology, the Pan American Medical Association, the Nassau County Medical Society, and the Medical Society of the State of New York.

Vladimir L. Shusterov, MD, Forest Hills. Died January 25, 1990; age 39. Dr Shusterov was a 1977 graduate of the Jawanatak Nehru Medical College, Karnatak University, Belgaum, Mysore, India. His memberships included the American Academy of Otolaryngology, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Shirley R. Snow, MD, Rochester. Died October 4, 1989; age 92. Dr Snow was a 1923 graduate of Columbia University College of Physicians and Surgeons. He was a Diplomate of the American Board of Obstetrics and Gynecology and a Fellow of the American College of Surgeons. He was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Simon Stein, MD, Boca Raton, Florida. Died December 16, 1989; age 93. Dr Stein was a 1917 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Rudolf H. Steinharter, MD, Fairborn, Ohio. Died January 6, 1990; age 80. Dr Steinharter was a 1936 graduate of the Medizinische Fakultaet der Universitaet Bern, Bern, Switzerland. He was a Fellow of the American Academy of Family Practice. His memberships included the New York State Society of Occupational Medicine, the New York Academy of Medicine, the American Occupational Medical Association, the American Academy of Occupational Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Nathan Turner, MD, Plainview. Died January 7, 1990; age 56. Dr Turner was a 1960 graduate of the Faculte de Medecine de l'Universite de Geneve, Geneva, Switzerland. He was a Fellow of the American Academy of Family Practice and a Diplomate of the American Board of Family Practice. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Greta Herta Van Fenema, MD, New York. Died September 23, 1989; age 82. Dr Van Fenema was a 1937 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. Her memberships included the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Rodney Thorp Wood, MD, Southampton. Died December 7, 1989; age 61. Dr Wood was a 1953 graduate of the Oxford University Medical School, Oxford, England. He was a Diplomate of the American Board of Anesthesiology and a Fellow of the American College of Anesthesiologists. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Suffolk County Medical Society, and the Medical Society of the State of New York.

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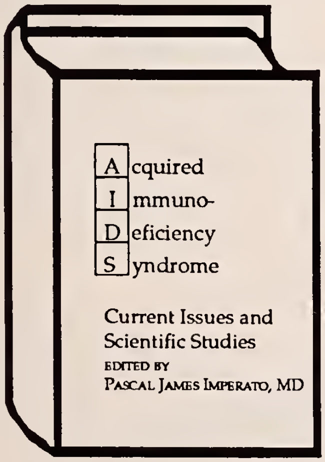
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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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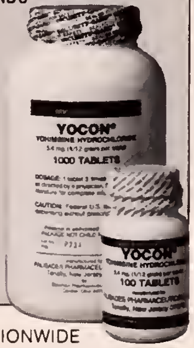

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May 20. **Department of Anesthesiology Centennial Program.** Beth Israel Medical Center. Contact: Administrator, Department of Anesthesiology, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003. Tel: (212) 420-2385.

May 24. **Cytokines and Cell Motility.** 7 Cat I Credits. New York Hilton. Contact: Ann J. Boehme, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

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May 9. **Psychotropic Drug Use in the Elderly: Current Concepts for Appropriate Management of Neuropsychiatric Disorders.** 7 Cat I Credits. The Parker Jewish Geriatric Institute. *May 18.* **Third Annual Sam Stein Memorial Lecture, Low Oxygen.** 5 Cat I Credits. Long Island Jewish Medical Center. *May 23.* **A Clinical Day in Ophthalmology, Ocular & Orbital Trauma.** 7 Cat I Credits. Garden City Hotel. Contact: Ann J. Boehme, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

SOUTHAMPTON

May 15. **Update: Asthma 1990.** Contact: Charlotte Henry-Sullivan, Medical Staff Office, Southampton Hospital, 240 Meeting House Lane, Southampton, NY 11968. Tel: (516) 283-2600, ext 519.

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May 30–June 3. **16th Annual Family Medicine Update.** 37½ Cat I Credits. State University of New York at Stony Brook. Contact: Dorothy S. Lane, MD, Associate Dean for CME, School of

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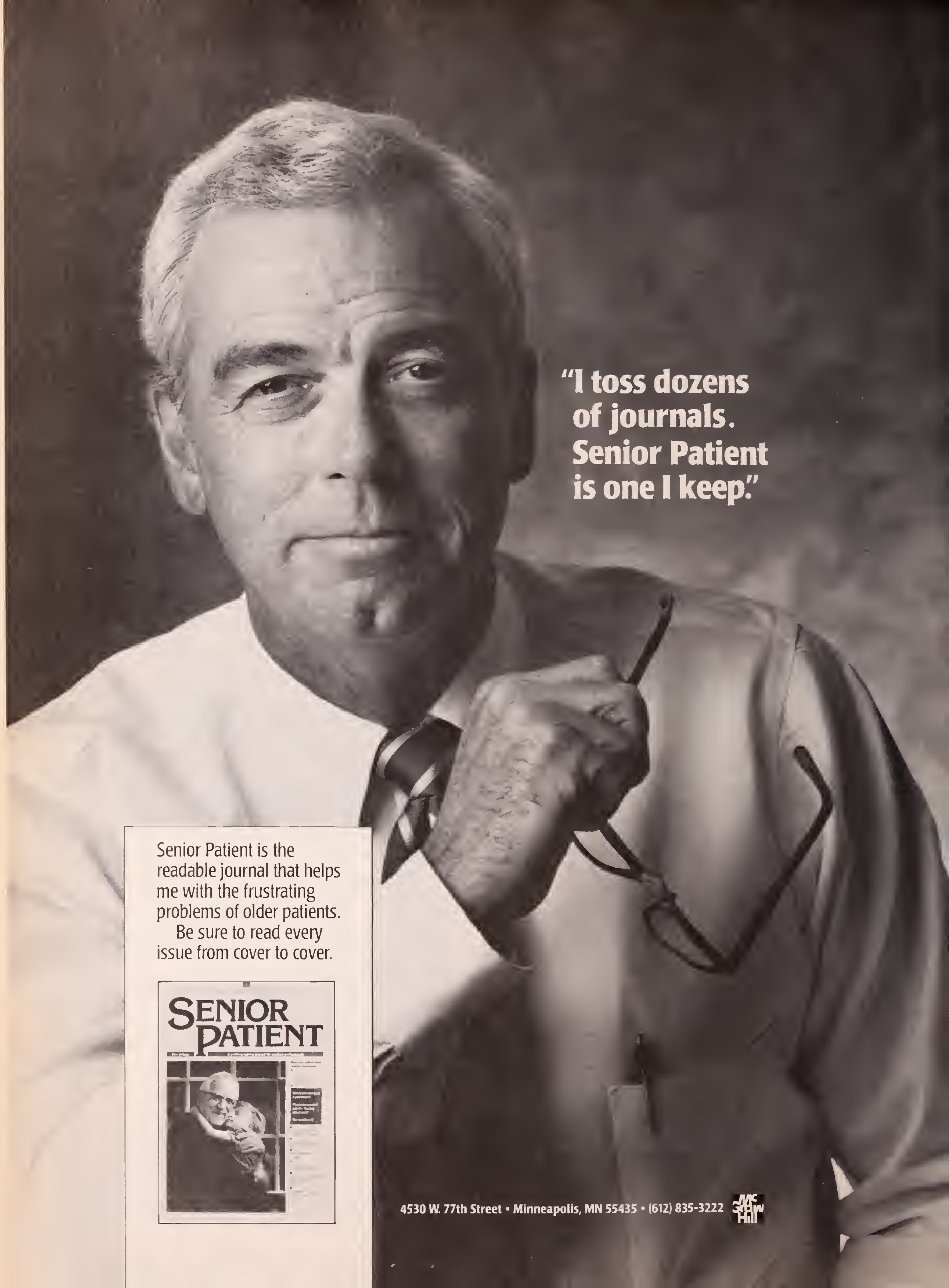
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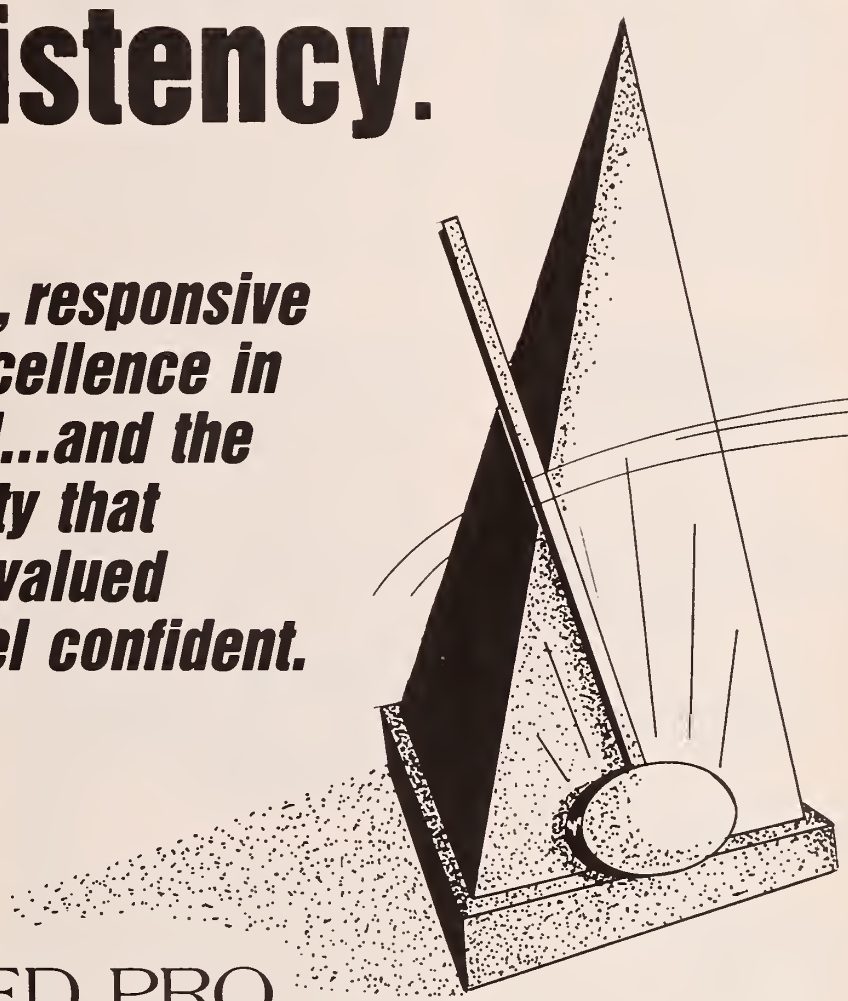

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(*continued from p 14A*)

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AROUND THE NATION

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May 17–19. **23rd Annual Advances & Controversies in Clinical Pediatrics.** 16 Cat I Credits. Fairmont Hotel, San Francisco. *May 24–25.* **Sixth Annual Current Issues in Anatomic Pathology: 1990.** 12 Cat I Credits. Union Square Holiday Inn, San Francisco. Contact: Extended Programs in Medical Education, Registration Office, Room C-124, University of California, San Francisco, CA 94132. Tel: (415) 476-5808.

May 22–25. **CT and MR in Body Imaging: A Comprehensive Course.** Monterey. Contact: Radiology Postgraduate Education, University of California, Room C324, San Franciso, CA 94142. Tel: (415) 476-5731.

May 29–June 1. **Perspectives: Clinical Trade-Offs in Critical Care.** 34 Cat I Credits. San Francisco Marriott. Contact: Lisa Parks, Director of Communications, Society of Critical Care Medicine, 251 E Imperial Highway, Suite 480, Fullerton, CA 92635. Tel: (714) 870-5243.

FLORIDA

May 16–18. **Pulmonary Emphysema: The Rationale for Therapeutic Intervention.** Grosvenor Resort, Orlando. Contact: Conference Director, The New York Academy of Sciences, 2 E 63rd St, New York, NY 10021. Tel: (212) 838-0230.

ILLINOIS

May 3–6. **Spring Symposium on Back Pain.** 18½ Cat I Credits. Fairmont Hotel, Chicago. Contact: American Back Society, 2647 E 14 St, Suite 401, Oakland, CA 94601. Tel: (415) 536-9929.

MARYLAND

May 2–4. **Hyperlipidemia in Childhood and the Development of Atherosclerosis.** Hyatt Regency, Bethesda. Contact: Conference Director, The New York Academy of Sciences, 2 E 63rd St, New York, NY 10021. Tel: (212) 838-0230.

SOUTH CAROLINA

May 3–5. **Emergency Medicine and Toxicology 1990.** 20 Cat I Credits. Sheraton Ashley Hotel, Charleston. *May 24–26.* **Annual Spoleto Course, Update in Geriatrics.** 24 Cat I Credits. Omni Hotel Charleston Place, Charleston. Contact: Gerri LaVia, Department of Family Medicine, Medical University of South Carolina, Charleston, SC 29425. Tel: (803) 792-2426.

AROUND THE WORLD

CANADA

May 4–5. **Toronto Neurology Update.** 15 Cat I Credits. Hotel Admiral, Toronto. Contact: Dr M. J. Gawel, University of Toronto. Tel: (416) 480-4959.

May 5. **Eighth Annual Can-Am Clinical Anaesthesia Conference.** Prince of Wales Hotel, Niagara-On-The-Lake, Ontario. Contact: Richard Ament, MD, Vice Chairman & Director of Educational Programs, Department of Anesthesiology, SUNY at Buffalo, 3 Gates Circle, Buffalo, NY 14209. Tel: (716) 887-5330.

ITALY

May 7–10. **Clinical Applications of Flow Cytometry—An International Course.** 23 Cat I Credits. Stresa. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773. □

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Eli Lilly and Company . . . 2nd Cover
Marine Midland Bank . . . 3A
McGraw Hill Publications . . . 6A, 16A
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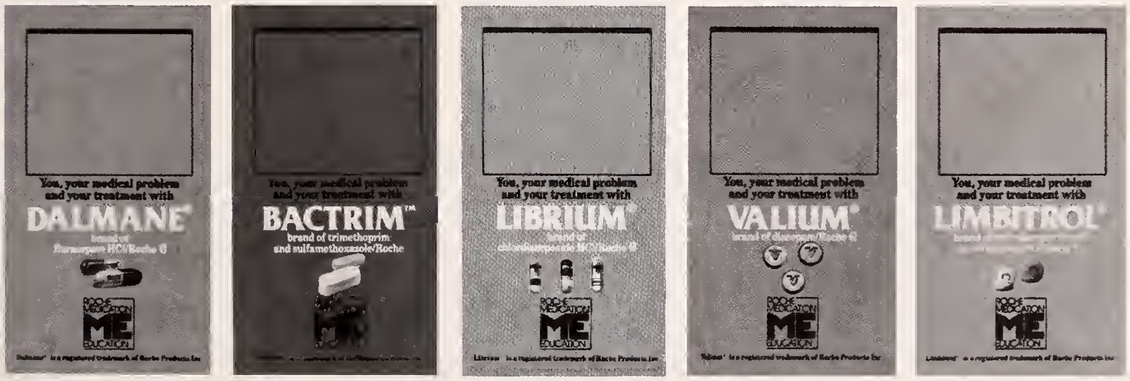

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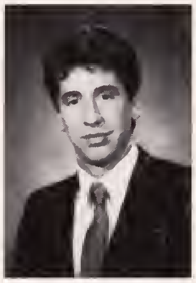
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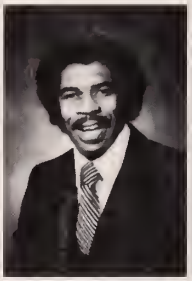
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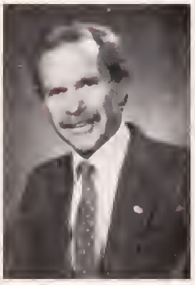
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Contents

COMMENTARIES

Circumcision of the newborn 231
TOM METCALF, MD

Hypersensitivity pneumonitis 232
RICHARD P. STANKUS, MD, PHD

RESEARCH PAPERS

Disseminated histoplasmosis in AIDS: Clinicopathologic features in seven patients from a non-endemic area 234
WILLIAM A. ANKOBIAH, MD; KAMINI VAIDYA, MD; STEVEN POWELL, MD; MANUEL CARRASCO, MD; AZMY ALLAM, MD; VIJAY CHECHANI, MD; STEPHAN L. KAMHOLZ, MD

HIV infected hospital patients in New York State: The development of longitudinal information from a hospital discharge data system 238
GERALD I. KAUFMAN, PHD, MCS; JOHN C. GRABAU, PHD, MPH; EUGENE M. SCHMIDT; YANGSOOK HAN, MS

Current trends in routine newborn male circumcision in New York State 243
MICHAEL S. WILKES, MD, MPH; STEVE BLUM, PHD

REVIEW ARTICLE

The current hospital crisis in New York City and policy options for resolving it 247
KENNETH E. THORPE, PHD

SPECIAL ARTICLE

He had a change of heart 253
IRVING A. BUNKIN, MD

HISTORY

Diphtheria and medical therapy in late 19th century New York City 256
VIRGINIA A. METAXAS QUIROGA, PHD

CASE REPORTS

Hypersensitivity pneumonitis associated with the use of ultrasonic humidifiers 263
SHYI-TANG SHIUE, MD; HERBERT H. SCHERZER, MD; ARTHUR C. DEGRAFF, JR, MD; SOLON R. COLE, MD

Salmonella arthritis 266
LAURA L. FISHER, MD, R. GORDON DOUGLAS, JR, MD

Manual labor metacarpophalangeal arthropathy in a baker 268
ELIZABETH MCDONALD, MD, CATHERINE MARINO, MD

LETTERS TO THE EDITOR

Intestinal perforation/fistulization complicating temporal arteritis 270
HENRY SCHNEIDERMAN, MD

Cervical tuberculosis 270
MUHAMMAD A. HENA, MD

The use of a Swan-Ganz catheter and streptokinase in the management of massive hemoptysis 270
MICHAEL A. NAKAO, MD

The PRO contract 271
MARTIN MARKOWITZ, MD

The influence of malpractice fears on the practice of medicine 271
BERNARD J. KATZ, MD

Fetal heart rate changes during general anesthesia 272
DANIEL FAUSTIN, MD; LOUIS GUTIERREZ, MD; M. RASHID CHAUDHRY, MD; MOKHTAR Y. ESTEFAN, MD

LEADS FROM EPIDEMIOLOGY NOTES 273
BOOK REVIEWS 276
NEWS BRIEFS 280
GUIDELINES FOR AUTHORS 282
MEDICAL MEETINGS AND LECTURES 3A

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References

1. *USP DI Update, September/October 1988, p 120.*
2. *Br J Clin Pharmacol 1985;20:710-713.*
3. *Data on file, Lilly Research Laboratories.*
4. *Scand J Gastroenterol 1987;22(suppl 136):61-70.*
5. *Am J Gastroenterol 1989;84:769-774.*

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Carcinogenesis, Mutagenesis, Impairment of Fertility—A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

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In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of varying durations included almost 5,000 patients. Among the more common adverse events in domestic placebo-controlled trials of over 1,900 nizatidine patients and over 1,300 on placebo, sweating (1% vs 0.2%), urticaria (0.5% vs <0.01%), and somnolence (2.4% vs 1.3%) were significantly more common with nizatidine. It was not possible to determine whether a variety of less common events was due to the drug.

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Hepatic—Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to three times the upper limit of normal, however, did not significantly differ from that in placebo patients. Hepatitis and jaundice have been reported. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS—Rare cases of reversible mental confusion have been reported.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with equal frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic—Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental—Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity—As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Because cross-sensitivity among this class has been observed, H_2-receptor antagonists should not be administered to those with a history of hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance by approximately 84%.

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MEETINGS AND LECTURES

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JUNE 1990

AROUND THE STATE

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June 8–9. **Radiation Oncology 1990.** Desmond Americana. Contact: Diane Martin, Conference Coordinator, Office of Continuing Medical Education, Albany Medical College, 47 New Scotland Ave, Administration Bldg, Room 217, Albany, NY 12208. Tel: (518) 445-5828.

COOPERSTOWN

June 13. **Common ENT Problems.** 3

(*continued on p 7A*)

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Hydrocodone Signs and Symptoms: Serious overdose with hydrocodone is characterized by respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, (cyanosis), extreme somnolence progressing to stupor or coma, skeletal muscle flaccidity, cold and clammy skin, and sometimes bradycardia and hypotension. In severe overdosage, apnea, circulatory collapse, cardiac arrest and death may occur.

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(*continued from p 3A*)

Cat I Credits. Bassett Hall Conference Center. Contact: Charlotte L. Hoag, Medical Education Coordinator, Office of Medical Education, The Mary Imogene Bassett Hospital, One Atwell Rd, Cooperstown, NY 13326. Tel: (607) 547-3926.

MANHATTAN

June 2. **Issues in Pediatric Otology.** 7 Cat I Credits. NYU Medical Center. *June 2–3.* **Hand and Wrist Injuries Among Professional Athletes.** 11½ Cat I Credits. NYU Medical Center. *June 8–9.* **Vascular Lesions of the Head and Neck.** 11 Cat I Credits. NYU Medical Center. *June 18–21.* **Annual Review: Selected Topics in Pediatrics.** 20 Cat I Credits. NYU Medical Center. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

June 8–9. **Masters of Cataract Surgery.** Waldorf Astoria Hotel. Contact: Francine Leinhardt, Course Coordinator, Manhattan Eye, Ear & Throat Hospital, 210 E 64th St, New York, NY 10021. Tel: (212) 838-9200, ext 2776.

June 11–15. **Update Your Medicine 1990.** 36 ½ Cat I Credits. New York Hospital-Cornell University Medical College. Contact: Office of Continuing Medical Education, 1330 York Ave, D-115, New York, NY 10021. Tel: (212) 746-4752.

AROUND THE NATION

CALIFORNIA

June 8–9. **The Threat to the Cosmic Order: Psychological, Social, and Health Implications of Richard Wagner's Ring of the Nibelung.** University of California School of Medicine, San Francisco. *June 8–9.* **Advances in Skin Cancer Management.** 10 Cat I Credits. Portman Hotel, San Francisco. Contact: Extended Programs in Medical Education, Room C-124, University of California School of Medicine, San Francisco, CA 94143. Tel: (415) 476-4251.

FLORIDA

June 28–July 1. **The Amelia Island Plantation Pediatric Conference.** Amelia Island Plantation, Amelia Island. Contact: Rayna Saville, Coordinator, Continuing Medical Education, 219 Bryant St, Buffalo, NY 14222-2099. Tel: (716) 877-7965.

ILLINOIS

June 14–16. **Contraception, Sterilization, and Sexually Transmitted Diseases.** Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024. Tel: (202) 638-5577.

MASSACHUSETTS

June 24–27. **17th Annual Harvard Medical School Course on Intensive Care Medicine.** Omni Parker House Hotel, Boston. Contact: Bart Chernow, MD, Dept of Anaesthesia, Harvard Medical School/Massachusetts General Hospital, 32 Fruit St, Boston, MA 02114. Tel: (617) 726-2858.

(*continued on p 10A*)

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NEW YORK STATE
JOURNAL OF MEDICINE

May 1990 • Volume 90, Number 5

COMMENTARIES

Circumcision of the newborn

Circumcision continues to be an enigma in American medicine. Nonreligious circumcision (along with clitoridectomy of the female) was first promoted in the United States in the second half of the 19th century as a method to prevent masturbation, then a much-feared precursor of insanity, retardation, and other mental ailments. It was later adopted for the same reasons in Europe.[1–3] It became entrenched in the social fabric of America and other English speaking countries during the late 19th and early 20th centuries. Holt wrote in his 1897 text of pediatrics,[4] "Circumcision should be done if phimosis exists, and even where it is not, the moral effect of the operation is sometimes of very great benefit." With the acceptance of Freud's psychoanalytic theories of mental illness, the medical profession in the United States promoted circumcision less and less over the next 50 years. The 1940 edition of Holt did not advocate circumcision either as a cure for masturbation or for medical reasons, asserting that masturbation produced no physical problems, that the only harm came from worry and guilt over the act.

In 1949, Gairdner[5] first wrote disputing the value of routine circumcision in Great Britain, and raised awareness concerning the lack of need for it to such a degree that the British National Health Service ultimately stopped payment for the procedure. Oster,[6] in Denmark, added prospective longitudinal data against the need for circumcision, asserting in 1968 that "The prepuce had not been the object of interest or of manipulation by doctors or parents, because in Denmark such a tradition has never existed." While numbers of newborns circumcised in the United States continued to increase, other English speaking countries ceased to circumcise, such that 80% of the males in the world are today uncircumcised, including those in Europe.[7] The United States stands alone in its high rate, estimated at 80–92%.[8–10]

Various studies have been published on the demographic characteristics of circumcision.[10–13] Wilkes and Blum,[14] in their article in this issue of the *Journal*, bring us a step closer to understanding which newborns in New York City and New York State are circumcised, and to some extent why. The authors document well the trends from 1980 to 1986 in a retrospective study of newborn records, showing decreasing overall rates in public hospitals in New York City, from 38% to 31%, but increasing rates in private hospitals, from 48% to 50%. Rates for circumcision in private hospitals within New York City are noted to have been statistically significantly higher than in public hospitals for each of the seven years studied, regardless of payment category, race, or type of hospital. They hypothesize that salaried physicians (rather than those reimbursed per circumcision) and the incredibly low reimbursement of Medicaid for the procedure ($175 billed versus $12 paid by Medicaid) may be the reason for this fact. By comparison, Utah physicians typically charge a $65 fee, and Medicaid reimburses $30.97 (personal communication, Utah State Medicaid Office, and Willow Creek Pediatrics accountant, November 20, 1989).

They further show that middle and upper class blacks and whites are likely to have their newborn males circumcised, while Asians and Hispanics are less likely to have theirs circumcised, perhaps reflecting that middle class families are less likely to follow traditional ethnic preferences.

Finally, Wilkes and Blum[14] describe physicians' advice as one of the influences in increasing or decreasing circumcision rates. This reflects the work of Warner[15] and Patel,[16] although Patel noted only 11 of 100 cases where the doctor's input was responsible for the decision whether or not to circumcise the babies. Brown et al[17] reflect similar data: "In both our survey and a more recent one by Stein et al,[18] parents infrequently perceived the physician or clinic as influential in the circumcision decision." In a study conducted by the author and his colleagues, only 3% of 231 parents gave doctor's advice as their reason for having a baby circumcised.[10]

Wilkes and Blum's article is important in illustrating the demographics of infant circumcision during the period of time between the two statements on circumcision by the American Academy of Pediatrics, in 1975 and 1989. This predated the data of Wiswell et al[19] and others[20] on the protective effects of circumcision against urinary tract infections, and the work of Simonsen et al[21] and others[22] in showing the intact foreskin as possibly increasing susceptibility to HIV. Finally, the striking difference between amounts billed to Medicaid and amounts paid in New York State make one wonder what may be the effect of third party reimbursement, or lack of it, on this socially and culturally motivated procedure. A study similar to that of Wilkes and Blum in five years will provide valuable follow-up data regarding the effect of these latest developments on the demography of circumcision.

Advocates of circumcision are currently seeing valid reasons to support their long held position. Opponents counter by saying that the foreskin is not a mistake of nature and that one should be suspicious of a urinary tract infection in *any* child less than a year of age, circumcised or not. Most physicians realize that the decision whether or not to circumcise a male infant will continue to be made by the parents, with input other than that of physicians being the chief determinant of their choice. When physicians are asked for their opinion about whether a male infant should be circumcised, their input to parents should be objective, based on the best data available at the time, with realization of how nonreligious circumcision has evolved over the past century.

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Hypersensitivity pneumonitis

Hypersensitivity pneumonitis (HP) and extrinsic allergic alveolitis are descriptive terms that characterize a growing spectrum of granulomatous, interstitial, and alveolar-filling pulmonary disorders. Alluded to as early as 1713 by Ramazzini, this group of diseases continues to expand and include pulmonary responses to both organic dust antigens and simple inorganic chemicals as well. In the current issue of the *Journal*, Shiue et al[1] describe a form of HP associated with the use of ultrasonic humidifiers. Their report and previous studies document the occurrence of "humidifier lung" associated with exposure to microorganisms contaminating humidification systems.[2,3] Offending organisms have included *Thermoactinomyces vulgaris*, species of *Cephalosporium* and *Penicillium* in addition to *Bacillus sereus*, and several amoebae including *Naegleria gruberi* and *Acanthamoeba polyphagi*.[4] It is important to emphasize at the outset that HP is not induced by infection but rather by antigenic sensitization. Indeed, overt disease develops in less than 10% of individuals exposed to inciting antigen(s).[5]

Clinically, HP may present in two major forms: acute and chronic. Acute disease is exemplified in the case reports described by Shiue et al.[1] In the acute form, symptoms begin after heavy exposure to antigen. In case report 2, the patient "leaned over into the rising mist hoping that the humidity might relieve his dry cough." Coughing is often the patient's initial complaint, and it clears after the patient leaves the area of contact with antigen. Several hours (4 to 12) after exposure, the patient may have dyspnea, generalized malaise, and myalgia. These symptoms may be accompanied by fever and chills. Though the symptoms are transient, initial complaints are again repeated upon the next exposure with increasing dyspnea each time. Of importance in categorizing a disease as a form of HP, repeated exacerbations occur with each return to the source of exposure. Tolerance in a clinical setting has not been described in classic HP.

Interestingly, in the report by Shiue et al,[1] one patient was treated with corticosteroids while the second was not. Avoidance of repeated antigen exposure is the only established treatment for HP, and this treatment usually results in complete remission of symptoms and in return to normal pulmonary function. While corticosteroids inhibit the lesions of HP in animal models, their efficacy in humans is difficult to document because of the usual spontaneous resolution of disease following antigen avoidance.

Nevertheless, corticosteroids are often used for exacerbations following acute exposure and also to inhibit or delay pulmonary fibrosis in chronic disease. The usual treatment regimen for acute disease calls for administering prednisone, 40 to 60 mg daily in divided doses for five to seven days, and tapering the dose over the subsequent two-week period.

Antihistamines and bronchodilators are not useful unless there is significant airway obstruction caused by either HP or another concomitant respiratory disease. Hyposensitization is not indicated and can in fact potentially produce immune complex-mediated disease.

While the pathogenesis of HP remains as yet undefined, clues to important immune mechanisms have been derived from animal model and bronchoalveolar lavage studies. Recent observations favor the importance of "local" or bronchopulmonary cellular immunologic involvement. Although humoral alterations in HP have been well documented, serum precipitins to offending antigens may be present in up to 50% of exposed but asymptomatic individuals and may not be present in all patients with overt disease.[6] Moreover, passive transfer of lymph node cells and not serum from sensitized animals followed by challenge of recipients with inhaled antigen has produced pulmonary lesions typical of HP.[7]

Bronchoalveolar lavage is perhaps as informative as tissue biopsy in defining the lesions and immunopathogenesis of HP. Lavage fluid from patients with chronic HP contains large numbers of lymphocytes (generally greater than 50% by differential). The observation of lung lavage lymphocytosis in HP, although similar to findings in sarcoidosis, is more pronounced and grossly increased in comparison to values of less than 10% lavage lymphocytes obtained from "normal" controls. Several studies have confirmed the large proportion of lymphocytes in bronchoalveolar lavage fluid obtained from subjects with HP.[8,9] However, in at least two studies, asymptomatic, antigen-exposed subjects (pigeon breeders) also had high proportions of lymphocytes in bronchoalveolar lavage fluids.[9,10] Monoclonal antibodies were used to determine surface phenotypes of bronchoalveolar lavage and peripheral blood lymphocytes in both groups. The predominant T cell subset was T8+ (suppressor-cytotoxic subset). Peripheral blood T cell subsets were normal in both groups. In contrast to subjects with HP, subjects with sarcoidosis have a predominance of T4+ or helper T cells in lung lavage fluid.

The role of suppressor T cells in the clinical expression of HP may lie in their loss of functional suppression of disease activity (ie, functional immunoregulatory imbalance). Keller et al[9] observed such a loss of function in cells obtained from symptomatic subjects. In an animal model of HP, in vitro studies revealed T cells functioning as suppressors in asymptomatic, antigen-exposed animals which were nonfunctional in symptomatic animals.[11] Conceivably, suppression of disease activity may be mediated by suppressor T cells, which either become "nonfunctional" or the effects of which are overcome by separate disease promoting cellular and/or humoral factors.

The current studies by Shiue et al[1] emphasize the need for all physicians to obtain an adequate occupational and environmental history in patients with chronic respiratory complaints. Although classic forms of HP including farmer's lung and pigeon breeder's disease are relatively easy to diagnose, the diagnosis of HP associated with contaminated heating/air conditioning and humidification systems is often overlooked. Hopefully, these current studies will promote a continued awareness of common environmental and occupational exposures that produce recurrent respiratory symptoms which may progress to pulmonary fibrosis if undiagnosed and not appropriately treated.

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Disseminated histoplasmosis in AIDS

Clinicopathologic features in seven patients from a non-endemic area

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ABSTRACT. Although the clinical and epidemiologic features of progressive disseminated histoplasmosis (PDH) in the acquired immunodeficiency syndrome (AIDS) have been well described, the pathologic and pulmonary aspects remain to be fully defined. A retrospective review of three patients and a prospective study of four more with PDH and AIDS recently admitted to an inner city hospital in a non-endemic area were used to elucidate these features more fully. All patients were men aged 23 to 46 years, born in endemic areas, who had immigrated to the US seven to 15 years before the onset of their illnesses. Five had been exposed to human immunodeficiency virus (HIV) through intravenous drug use (one was also a homosexual), and two through heterosexual contacts.

Respiratory symptoms were evident in five of the seven patients, fever in seven, weight loss in seven, hepatomegaly in four, splenomegaly in three, peripheral adenopathy in three, and gastrointestinal symptoms in three. PDH was the initial or only opportunistic infection in five patients. Bilateral nodular infiltrates (4/7), bilateral interstitial infiltrates (2/7), and mediastinal adenopathy associated with pleural effusion (1/7) were the chest roentgenographic findings.

Histoplasma capsulatum was isolated from five of five bronchoalveolar lavages, four of four transbronchial biopsies, one of one endobronchial biopsy, one of one brushing, one of one pleural biopsy, three of three lymph node biopsies, two of two bone marrow biopsies, one of one liver biopsy, and three of four peripheral blood smears. Granuloma formation was seen in only three of 12 biopsies. There were ten or more fungi per monocyte in almost all tissues, some with extracellular forms. Budding was common, but the hyphal forms were not seen. Serologic studies were positive in three of six cases. Despite early initiation of therapy with amphotericin B, three patients died on their initial admission and two on the second admission. Four deaths were associated with acute respiratory failure.

PDH in AIDS in non-endemic areas occurs mainly as secondary dissemination with frequent involvement of the respiratory system on clinical and pathologic examination. Granuloma formation is rare. Diff-Quik stain is helpful in making early diagnosis. Extensive tissue involvement and high mortality reflects severe immune suppression by both HIV and *H capsulatum*, with the PDH poorly responsive to therapy.

(NY State J Med 1990; 90:234-238)

Progressive disseminated histoplasmosis (PDH) is a rare manifestation of infection due to *Histoplasma capsulatum*.[1] It is usually observed in immunocompromised hosts[2] and as severe primary infection in children.[3,4] An association between PDH and the acquired immunodeficiency syndrome (AIDS) was first proposed by workers during a histoplasmosis epidemic in 1981.[5] However, the first case report appeared in 1982.[6] Subsequent reports have come from the endemic areas of the United States[7-12] where, in some series, PDH was found to be the most common opportunistic infection in patients with AIDS.[9,11] Other cases have been reported from non-endemic areas in the United States[13-16] and from other parts of the world.[17-21]

More than 100 cases have been reported in the literature, and PDH is included in the case definition of AIDS by the Centers for Disease Control (CDC), highlighting it as a major opportunistic infection in the AIDS epidemic.[22] Despite this recent emphasis, PDH in AIDS is probably underdiagnosed.[12] Optimal approaches to early diagnosis and treatment remain to be defined, particularly in non-endemic areas where prior clinical experience with histoplasmosis may be limited.

Seven patients with AIDS complicated by PDH were diagnosed and treated during a recent 15-month period at this institution and form the basis of this report. The clinical and pathologic features of these patients are compared to those of previously reported immunocompromised hosts with PDH, in an attempt to identify clinical features that may aid in early diagnosis. A specific cytologic screening method is suggested to improve early diagnosis of histoplasmosis infection.

Patients and Methods

We reviewed the records of the seven patients with PDH complicating AIDS admitted to Woodhull Medical and Mental Health Center in Brooklyn, NY (three retrospectively and four prospectively). All the patients were admitted between January 1987 and May 1988. Histoplasmosis had not been diagnosed at

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this hospital prior to January 1987. All patients met the CDC criteria for AIDS,[22] and six had positive anti-HIV serology (New York City Department of Health Laboratory). One refused to be tested.

The diagnosis of PDH was made by finding intracytoplasmic yeast morphologically consistent with *H capsulatum* in reticuloendothelial cells (histiocytes or macrophages), and/or by isolating *H capsulatum* by culture from at least one organ outside the lung or in the peripheral blood smear alone. However, it is recognized that recovery of *H capsulatum* from the lung in AIDS patients is presumptive evidence of dissemination. The presence of granulomata was not considered essential in making a histologic diagnosis. The diagnosis of PDH was further supported by a positive serology for *H capsulatum* in some patients. A positive serology was defined as a positive M arc on immunodiffusion test, and/or a positive complement fixation titer to mycelial or yeast antigen of greater than 1:16. All tissue specimens were reviewed by one of the authors to confirm the extent of intracytoplasmic yeast, the presence of granuloma, and/or any other microbial agents before the preparation of this report.

To facilitate early diagnosis, we adopted a systematic pathologic approach. All unfixed pathologic specimens from which smears or touch preparations could be made were immediately stained with Diff-Quick, a modified Wright's stain (American Scientific Products, McGraw Park, Ill), which yielded morphologically positive stains for *H capsulatum* in all specimens in which it was used. The fungi typically appear as clusters of oval yeast, either free or within macrophages, often enveloped by a clear zone, giving the appearance of a capsule. The cell wall stains purple to blue, and the interior is clear except for a crescent-shaped pink to red polar body at the wide end (Fig 1). Frequently, budding could be seen.

Confirmatory stains were Gomori's silver methenamine and/or periodic acid-Schiff stain. Appropriate cultures for viruses, bacteria, mycobacteria, and fungi were performed on all specimens.

RESULTS

Epidemiologic Characteristics. As summarized in Table I, all seven patients were males born outside of the United States who had lived in New York City for seven to 15 years prior to the onset of their clinical illness. None were known to have visited an endemic area for histoplasmosis in this country or to have worked in an occupation known to lead to exposure. More than 60% of all patients in this hospital are Hispanics from the Caribbean and Central and South America (areas classified as endemic for histoplasmosis); this population also accounts for 50% of the AIDS patients. No known cases of histoplasmosis have been diagnosed in this locality in non-AIDS patients to suggest an epidemic.

Clinical Features. Several major organ systems were clinically involved at presentation. Four of the patients presented to the pulmonary service for evaluation of possible *Pneumocystis carinii* pneumonia, only to have *H capsulatum* found in bronchoscopic specimens. Gastrointestinal symptoms were nonspecific but abdominal pain was common (3/7). In one patient, acute cholecystitis was suspected from clinical and radiologic findings, although the gallbladder was normal at laparotomy and *H capsulatum* was isolated from the para-aortic nodes.

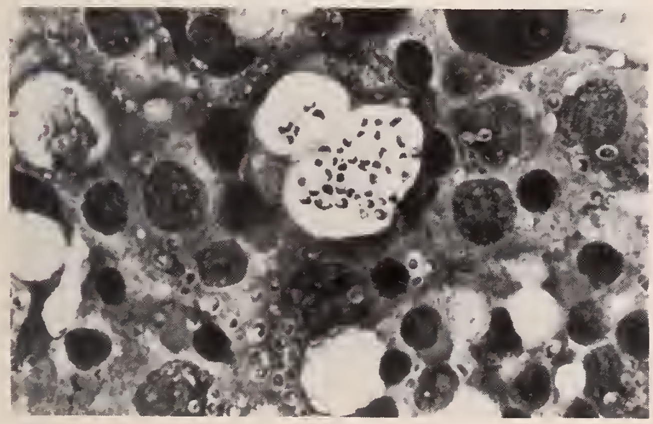
FIGURE 1. Touch smear of cervical lymph node shows intrahistiocytic aggregates of *Histoplasma capsulatum* (Diff-Quik stain; original magnification × 400).

None of the patients were initially suspected of having histoplasmosis. Mediastinal adenopathy compressing the esophagus was found in one patient, and another had extensive retroperitoneal adenopathy on computerized axial tomography (CAT) scan. One patient had persistent lower gastrointestinal bleeding caused by large intestinal ulcerations from histoplasmosis, complicated by disseminated intravascular coagulation (DIC) and liver failure due to prior alcohol abuse. He required a right hemicolectomy to control the bleeding. Five of the patients received amphotericin B for suspected histoplasmosis within 72 hours of their admission to the hospital. Three of the five died within three weeks of their initial admission despite early initiation of therapy. Two of the patients died within the first 48 hours of the second admission, six to eight months after initial presentation. The patients (or their relatives) had indicated noncompliance with oral ketoconazole prophylaxis and were admitted with acute respiratory failure, septic shock, and DIC. (Debate exists as to the prophylactic efficacy of ketoconazole versus amphotericin B in this setting.) Autopsy data are limited in these patients, but antemortem specimens obtained have not revealed any other infectious agents.

Pathologic and Laboratory Findings. The diagnostic procedures and the pathologic and microbiologic outcomes are summarized in Table II. Thrombocytopenia (4/7) was more common than usually observed in our other AIDS patients. DIC occurred in three of seven patients within 72 hours of death.

The fungi were found in the yeast form, few with budding, in histiocytes or macrophages in all the specimens reviewed and in monocytes in the peripheral blood smears (Fig 2). The hyphal form was not identified in any tissue. The yeasts were often packed in groups of ten or more in the cytoplasm or extracellular material. Granuloma formation was found in only three tissue specimens (liver, lung, and mesenteric lymph nodes) and in only one of these (endobronchial mass biopsy) was the yeast form found in the multinucleated giant cells (Fig 3). Intravascular yeast was found in only one tissue (intestinal mucosa), although the sinusoids of a liver biopsy were filled with free yeast. Tissue necrosis was found in only three biopsy specimens.

Although the bone marrow has previously been found useful in confirming the diagnosis of histoplasmosis infection,[1,4,5,12,23] we utilized it in only two patients. The lung was the predominant organ from which the organism was isolated in five of the patients, four of whom presented with suspected *P carinii* pneumonia. In one patient, biopsy of a 2-cm endobronchial mass in the bronchus intermedius was morphologically positive for *H capsulatum*. Pleural biopsy in the fifth patient in whom pleural effusion developed ten days after amphotericin therapy was started had *H capsulatum* demonstrable in pleural tissue and culture.

Roentgenography. All patients had abnormal findings on their chest roentgenograms in the course of their clinical illnesses. Three had normal chest roentgenograms initially—two later displayed reticulonodular infiltrates, and one had pleural effusion. Bilateral reticulonodular infiltrates (two patients) and bilateral interstitial infiltrates (two patients) were found in the other patients at presentation. There was improvement in the chest roentgenogram in four patients while on therapy.

DISCUSSION

The epidemiologic and demographic aspects of these patients are typical of those previously reported—predominantly male, Hispanic, intravenous drug users in New York City.[13-16] PDH in an immunocompromised host may be an acute primary infection, reinfection, or reactivation. There is no evidence that these patients had a

TABLE I. Epidemiologic and Clinical Features of Seven Patients with Disseminated Histoplasmosis and the Acquired Immunodeficiency Syndrome

Patients' Age (yr)/Sex/ Risk Factors	Country of Origin/Year Entry to US	Clinical and Laboratory Presentation	Chest Roentgenogram	Associated Infection	Outcome
47/male/heterosexual (HIV positive)	Ecuador/ 1976	Fever, weight loss (20 lb), dyspnea (3 mo), hepatosplenomegaly, abnormal liver function tests, thrombocytopenia	Bilateral interstitial infiltrates	None	Diagnosis three weeks after admission. Died five days after therapy started. Autopsy: *Histoplasma* in liver, spleen, adrenal, skin, lymph nodes. *Toxoplasma* in lungs and central nervous system.
31/male/homosexual (HIV positive)	Puerto Rico/ 1975	Fever, weight loss (30 lb), dyspnea (2 mo), abdominal pain (1 wk), cervical adenopathy, hepatosplenomegaly, abnormal liver function tests, thrombocytopenia	Bilateral reticulonodular infiltrates	Previous *Pneumocystis carinii*	Improved clinically and radiologically after 440 mg amphotericin (May 1987). Signed out. Readmitted in Sept 1987 in respiratory failure and died 48 hr later. No autopsy.
39/male/unknown (HIV not done)	Colombia/ 1973	Fever, weight loss (25 lb), abdominal pain (3 days), dysphagia (3 wk), cervical adenopathy, thrombocytopenia	Mediastinal adenopathy, bilateral pleural effusion	Esophageal candidiasis	Improved clinically and radiologically on 700 mg amphotericin and signed out against medical advice. Lost to follow-up.
26/male/homosexual/intravenous drug user (HIV positive)	Puerto Rico/ 1972	Fever, weight loss (20 lb), cough, shortness of breath (6 wk), hypoxia	Bilateral reticulonodular infiltrates	Oropharyngeal candidiasis	Improved after 2 g amphotericin. Discharged (December 1987) on ketoconazole, 400 mg/day. Lost to follow-up. Readmitted with disseminated intravascular coagulopathy and died within 24 hr (May 1988).
36/male/intravenous drug user (HIV positive)	Puerto Rico/ 1973	Fever, dry cough, dyspnea, weight loss (30 lb in 3 mos), hypoxia	Bilateral nodular infiltrates	Subsequent pulmonary tuberculosis	Improved after 2.4 g amphotericin. Discharged on ketoconazole, 400 mg/day. Readmitted with fever three mos later and found to have miliary tuberculosis. Alive after seven mos of follow-up.
36/male/intravenous drug user (HIV positive)	Puerto Rico/ 1968	Fever, productive cough, weight loss (24 lb), pleuritic chest pain (1 mo), cervical adenopathy, thrombocytopenia, intestinal bleeding	Bilateral interstitial infiltrates	Previous *Pneumocystis carinii*	Started amphotericin three days after admission. Had right hemicolectomy for intestinal bleeding but continued to bleed from DIC and died after two wk of amphotericin B therapy.
35/male/intravenous drug user (HIV positive)	Puerto Rico/ 1973	Fever, dry cough, weight loss (15 lb), abdominal pain, diarrhea, dysphagia (1 mo), hepatomegaly, cervical adenopathy	Diffuse bilateral miliary infiltrates	Concurrent pulmonary and central nervous system cryptococcus	Improved clinically and radiologically on amphotericin, 350 mg. Died suddenly (June 1988).

primary infection or reinfection from a point source in New York City. Secondary dissemination may be the predominant form of histoplasmosis in both endemic and non-endemic areas.[12] Since all of these patients came from known endemic areas, we suspected the dissemination to be secondary. We believe this to be true for the following reasons: absence of cases in locally born children; absence of cases in non-AIDS patients; previous residence in an endemic area; the rarity of case reports prior to the AIDS epidemic; the long interval since immigration from the endemic area before the onset of histoplasmosis; and onset of illness after immunosuppression by HIV infection. The presence of granuloma in some of the tissues may imply an old infection in a previously immunocompetent host.[24]

The clinical presentation of PDH in these patients was similar to reports of other AIDS patients with PDH.[12,15,16] When compared to other immunocompromised hosts with PDH[2,25] and the rare host with no immunosuppression in

TABLE II. Diagnostic Methods and Results for Histoplasmosis

Organ	Diagnostic Procedure	Histopathology Fungi*	Histopathology Granuloma*	Culture*
Lung	Bronchoalveolar lavage	5/5	—	2/5
	Transbronchial biopsy	3/3	1/3	0/3
	Endobronchial biopsy	1/1	1/1	0/1
	Pleural biopsy	1/1	0/1	0/1
Bone marrow smear and biopsy		2/2	1/2	1/2
Peripheral blood smear		3/4	—	2/7
Liver	Biopsy	2/2	1/2	1/2
Abdomen (laparotomy)	Mesenteric nodes	2/2	1/2	0/2
	Intestinal biopsy	1/1	0/1	0/1
Total		20/21 (97%)	5/12 (43%)	6/24 (25%)

* Number positive/number done.

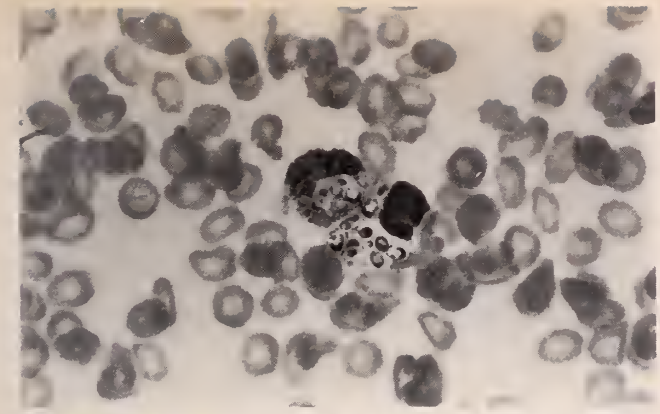

FIGURE 2. Peripheral blood smear reveals a monocyte containing aggregates of *Histoplasma capsulatum* (Diff-Quik stain; original magnification × 1,000).

whom PDH develops,[24] these patients exhibited no real differences except for the high mortality. Other immunocompromised hosts with PDH are, however, found mainly in endemic areas where the suspicion of histoplasmosis is high. In non-endemic areas, a low index of suspicion and overlap with the clinical features of other HIV-associated infections has often resulted in a low rate of antemortem diagnosis.[10,12]

Thrombocytopenia should heighten the suspicion of PDH in AIDS. Although immune thrombocytopenia has been well described in AIDS patients,[26] it is relatively rare and is also a known feature of PDH in patients who do not have AIDS.[27] It is reasonable to postulate that the combination of HIV/PDH may lead to a higher incidence of thrombocytopenia as noted in these patients. The onset of DIC was predictive of death within a few days in these patients, as previously noted by Wheat et al.[8] DIC has been reported in other series in patients who later died,[15] although recovery after development of DIC can occur.[16]

Radiologic features were nonspecific in these patients and have not been previously reported in detail to allow a general review. Initial chest roentgenographic findings may be normal, but when abnormal, nodular infiltrates appear to be the most common finding, occurring in 60% of the patients reported here. This finding is not different from that of PDH in hosts with no immunosuppression[24] or in other immunocompromised hosts.[25] Two patients had hilar/mediastinal adenopathy, which is an uncommon feature in the AIDS/ARC patient as part of the generalized lymphadenopathy syndrome.[28] This prompted us to search for an opportunistic infection. Pleural effusion associated with *H capsulatum* is extremely rare in both AIDS and non-AIDS patients.[24,25]

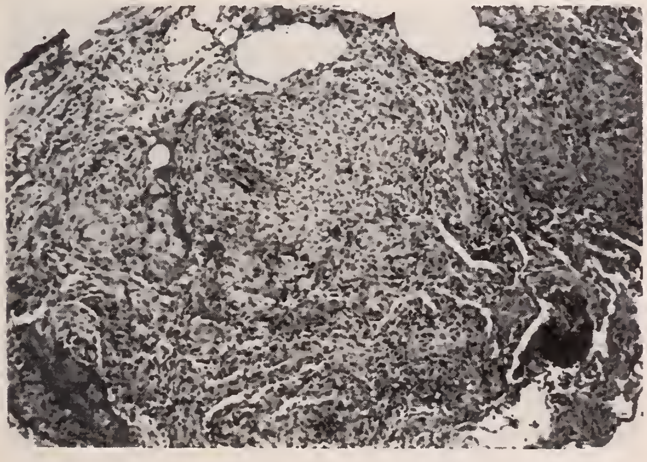

FIGURE 3. Biopsy of endobronchial mass shows submucosal non-necrotizing granuloma (hematoxylin-eosin stain; original magnification × 160).

H capsulatum was detected in specimens from bronchial brushing, bronchoalveolar lavage (BAL), and trans- and/or endobronchial biopsies obtained during bronchoscopy. The fungi were found in all five patients in whom BAL was done. The use of BAL in the diagnosis of pulmonary histoplasmosis has not been stressed in previous reports, and reliance has been placed on biopsies. In one series, BAL was not useful in making the diagnosis.[12] In patients with AIDS, the extent of dissemination (macrophages often have 10 to 12 fungi per cell and free forms often occur in tissue) probably leads to alveolar colonization. More cases are needed to confirm the high yield we observed in BAL, before obviating biopsy in the pulmonary evaluation of PDH. In BAL, *H capsulatum* can be confused with *P carinii* on silver stain. The use of Diff-Quik as an initial screening stain often helped us avoid this confusion. *Cryptococcus* is less likely to be confused because of its larger size, thicker capsule, and frequent budding. Where available, immunoperoxidase stain is also a promising means of early diagnosis,[29] and was used to identify specific fungi in a lymph node biopsy of one patient in the absence of fresh specimen. Lysis-centrifugation offers hope in improving the yield from blood culture when there is a question of dissemination.[30]

The poor prognosis of PDH in AIDS may be due to immunologic interaction between HIV and *H capsulatum*. Both cause diminished cellular immunity with reversal of the CD4/CD8 cell ratio.[31] Synergistic immunosuppression may account for PDH being the first or only opportunistic infection in five of seven of the patients described here, and in 60% of the patients in one previously reported series.[12] Although appropriate therapy for hosts with PDH and no immunosuppression may result in return of the CD4/CD8 ratio to normal,[32] this may not occur in AIDS patients because of the HIV infection. Alternatively, PDH, like tuberculosis, may appear before *P carinii* because less intense immunosuppression (ie, higher CD4 counts) is needed for these intracellular pathogens to reactivate than for *P carinii* to flourish.[33]

In conclusion, these findings suggest that PDH occurs in patients in New York as secondary dissemination, primarily in patients who previously resided in endemic areas (the Caribbean, and Central and South America). The occurrence of granulomata in tissue is rare. The use of Diff-Quik staining for screening tissue specimens facilitates early diagnosis and decreases under-diagnosis, especially in non-endemic areas where prior experience is limited. An increased awareness of the global distribution, the spectrum of clinical presentations, and the likelihood that reactivation is more frequent than primary infection are all essential for improved recognition of this disease in non-endemic areas. A classification of patients infected with both HIV and *Histoplasma* and primary chemoprophylaxis for those cases most likely to progress to dissemination is also required, in an effort to help decrease the high mortality rate witnessed in patients with PDH and AIDS.

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HIV infected hospital patients in New York State

The development of longitudinal information from a hospital discharge data system

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ABSTRACT. Abstracted records of all patients discharged from New York State acute care hospitals from January 1, 1983, through October 1, 1987, containing a diagnosis of HIV infection (N = 36,664) were linked into a longitudinal file of 20,005 patient-specific case histories. A validation study utilizing Medicaid patient-specific discharge information for calendar year 1985 showed that, on the average, each case history of the longitudinal file contained 85% (±5%) of the expected discharges. The number of patients present in the longitudinal file was 10% (±4%)—too large a percentage, due to a failure to link all the discharges to the appropriate case histories. The number of patients with a diagnosis of *Pneumocystis carinii* pneumonia (PCP) found in the longitudinal file was found to be consistent with the number reported to the New York State AIDS Registry. The longitudinal file appears to be sufficiently accurate and complete to use in evaluating HIV-related acute care in hospitals.

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New York State has been severely affected by the human immunodeficiency virus (HIV) epidemic. In the early 1980s, cases of acquired immunodeficiency syndrome (AIDS) originating in New York State accounted for more than half the cases nationwide. As of January 31, 1989, the state has reported 20,917 cases, which is approximately 25% of the cumulative national total.[1] There is significant mortality associated with AIDS: 11,730 patients, representing 56% of the total cases reported in New York State thus far, have died.

The epidemiologic characteristics and clinical management of AIDS-infected individuals is changing in New York State. Historically, the homosexual/bisexual risk

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behavior group accounted for the greatest number of AIDS cases. However, in 1988, this pattern shifted, and new cases of AIDS among intravenous drug users exceeded the number of new cases among the homosexual/bisexual risk group.[2] New treatment methods for HIV infection are leading to longer survival times and thereby changing hospital utilization patterns.[3] In order for the New York State public health authorities to identify accurately the necessary acute care facilities for the present and future, the following information about acute care resource consumption by the HIV-infected population is required:

- Number of patients entering the acute care system for the first time due to HIV infection, per selected time period;
- Demographic characteristics;
- Changes in diagnoses over time;
- Changes in reimbursement source (eg, private to public payors subsequent to the first HIV-related hospitalization);
- Lifetime costs and cost per time period;
- Lifetime admissions, length of stay, and intervals between stays;
- Hospital migration patterns.

A review of the literature indicates that little is known about how HIV-infected individuals utilize acute care facilities over time. Much of the case study analysis has been performed outside New York State[4–6] and has been limited to AIDS. A study of hospital experience with AIDS from four hospitals in New York City,[7] with an emphasis on cost, has been performed. These studies show considerable variations in utilization patterns and costs.

Hospital utilization measures have been reported based on data collected from a number of states' discharge data systems.[8,9] The systems do not link readmissions of patients, so analysis was limited to describing hospitalization utilization on a per admission basis, with no information regarding individual patient utilization patterns over time or the trends in the number of new patients seen.

A number of hospital surveys of HIV patients were conducted to obtain utilization rates of acute care facilities in New York State during 1987.[10,11] Each survey counted the number of patients hospitalized on a particular day, but the readmission patterns of patients could not be studied due to the absence of patient identifiers.

With changes occurring in the New York State HIV-infected hospital population, we saw the need for a method to monitor patient-specific utilization characteristics in acute care facilities. A New York State-specific information system which would capture data describing each hospital stay for each HIV-infected individual in a timely and cost efficient manner would be extremely useful.

Prior to initiating development of such a data collection system, it is prudent to determine if such information is available from existing data files. As Roos and his colleagues[12] have stated, "If a well-organized data base can be accessed, health care researchers should seriously consider its use before committing themselves to primary data collection." The purpose of this report is to describe the development of a new source of HIV longitudinal information which addresses the effect of the HIV epidemic on acute care facilities by using an existing statewide reporting system for acute care hospitals.

METHODS

Creation. The longitudinal information was assembled by linking all the separate HIV-related hospital discharge reports for each patient found in the Statewide Planning and Research Cooperative System (SPARCS) into a patient-specific case history (case). SPARCS is maintained by the New York State Department of Health and collects a discharge data abstract (DDA) and uniform billing form (UBF) for every acute care hospital stay in New York State, with the exception of those from psychiatric and federal hospitals. The DDA and UBF were joined into a single record, when possible, to create a "matched file."

The SPARCS system was created with strong confidentiality protections, and no explicit patient identifiers are present. Data are readily available on a discharge by discharge basis, but these discharges cannot be directly linked to form an individual's case. However, variables are available to establish linkages connecting records of the same patient. The useful variables include the hospital identifier; patient medical record number and gender from the DDA; and the insurance plan numbers, recipient numbers, patient address, and date of birth from the UBF. To establish the HIV longitudinal file (HIVLF), matched file records from the period 1983 through 1986 were selected if the DDA portion explicitly indicated HIV infection. A matched file was not available for the 1987 records, but the DDA records were included to maximize information for cases detected in earlier years. A DDA with an HIV infection was defined as one in which any of the diagnoses listed in Table I was coded as the principal or one of the four secondary diagnoses. These records were then linked into cases by processing them through a series of three phases, as shown in Figure 1.

Phase 1. The majority of hospitals assign a unique medical record number to an individual, which is reused for that patient on subsequent readmission. This numbering system allows the longitudinal tracking of patients

TABLE I. HIV Diagnoses ICD-9-CM Codes

ICD-9-CM Code	Description	Initiated
042	Includes acquired immunodeficiency syndrome (AIDS)	Fall 1986
043	Includes AIDS-related complex (ARC)	Fall 1986
044	Includes other HIV infection	Fall 1986
795.8	Positive serological or viral culture for HIV	Fall 1986
279.19	Other deficiency of cell-mediated immunity as a principal or secondary diagnosis	Fall 1982
279.10	Immunodeficiency with predominant T-cell defect as a principal or secondary diagnosis	Fall 1982
279.3	Unspecified immunity deficiency as a principal diagnosis with or without 279.19 or 279.10 as secondary diagnosis	Fall 1982
136.3	Pneumocystosis as a principal diagnosis with or without 279.10 or 279.19 as secondary diagnosis	Fall 1982

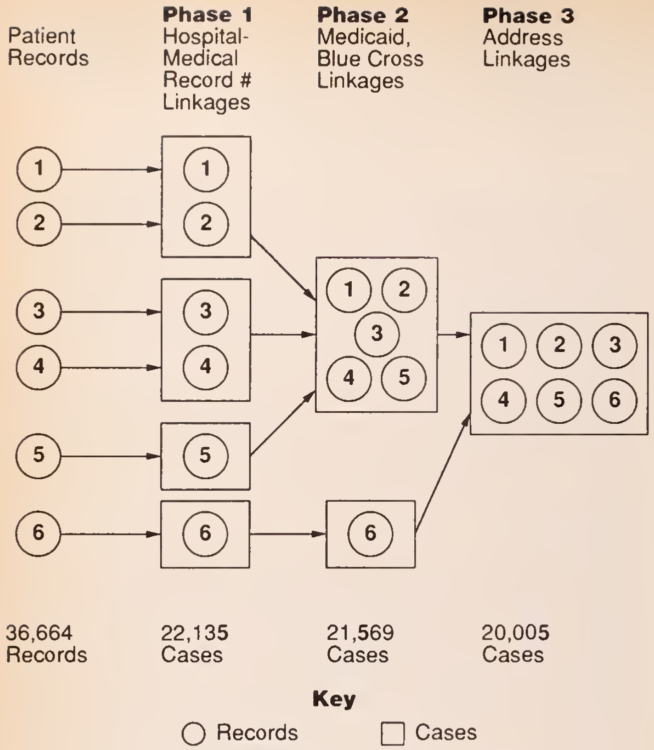

FIGURE 1. Linkage phase results (eg, a six-record case).

within the same hospital. Records with the same medical record number in the same hospital were linked. At the end of phase 1, each matched file record was assigned a case number, case gender, and case date of birth. The consistency of the date of birth and gender among the records of the newly linked cases was reviewed. At the end of this phase and each subsequent phase, the case date of birth and case gender were reassigned based on the principle of selecting that value which appeared most frequently. If a tie existed, the field was left undetermined.

Phase 2. Phase 1 cases were next linked by Medicaid recipient number and Blue Cross recipient number. The information from the remaining payors was not utilized for linkage in this phase, but all discharges independent of payor were included in the HIVLF.

Phase 3. Phase 2 cases were next linked by date of birth and address. Manual review of records sorted by address was performed, and linkage was based on matched addresses. Addresses were considered to be matched if no more than one character of the first five characters disagreed or if alternate spellings were present, eg, first and 1st.

Evaluation. To evaluate the completeness and accuracy of the HIVLF, an independent source of complete HIV case data was needed. Analysis of the HIVLF revealed that the highest proportion of first discharges (42%) was paid by Medicaid. The Medicaid system was selected as an independent source of patient-specific hospitalization histories in order to assess the completeness and linkage accuracy of the HIVLF. The New York State Department of Social Services (DSS) maintains a computer file of all discharges paid by Medicaid funds, which includes patient name, address, date of birth, Medicaid recipient number (MRN), admission date, discharge date, hospital record number, and hospital identifier. The MRN is an identification number typically assigned to only one person. However, more than one MRN can be assigned to the same individual when that person changes programs within DSS. The DSS file contains all Medicaid funded discharges. Therefore, it was necessary to eliminate all non-HIV related stays. For comparative purposes, it was decided that when a DSS record was found without any HIV diagnoses, and the associated SPARCS record did not contain any HIV diagnoses, that stay would be excluded from consideration. As a result, when comparisons between the DSS and HIVLF files were performed, records meeting these conditions were not considered missing from the HIVLF file. This approach was used to estimate the degree of completeness of the HIVLF.

To evaluate the completeness and accuracy of the HIVLF, the following questions were raised: How accurately did the linkage phases connect records of the same person? How complete were the HIVLF cases? How many extra cases were present in the HIVLF, ie, how frequently were patients assigned more than one case number?

Calendar year 1985 was selected for the validation study, since it was the most recent calendar year DSS considered complete. The DSS provided information for all hospital discharges during 1985 paid by Medicaid. A simple random sample of 213 cases was selected from the 1,976 HIVLF cases with 1985 discharges paid for by Medicaid. This sample was of sufficient size to determine the completeness of the HIVLF Medicaid records with 95% confidence and 80% power.[13] MRNs were obtained from the sample cases, and all discharges with those MRNs were extracted from the 1985 DSS file. Eleven cases had MRNs that were not found in the DSS file and are considered to be extra cases created due to MRN transcription errors in SPARCS. Thus 202 MRN unique patient-specific cases were available from DSS to evaluate the accuracy and completeness of the HIVLF. This reduced the power by 3%.

A binomial distribution normal approximation was used to estimate the 95% confidence limits standard error of the sample proportion (P) being investigated.[13] The value of P will be presented by $P \pm 1.96\ P(1 - P)/\sqrt{N}$.

RESULTS

Creation. Using the HIV diagnostic criteria, 36,664 discharge records were obtained: 2,106 discharges occurred in 1983; 4,177 occurred in 1984; 8,078 occurred in 1985; 13,114 occurred in 1986; 9,189 occurred in 1987 prior to October 1. The number of cases at the end of each phase was as follows: 22,135 for phase 1; 21,569 for phase 2; 20,005 for phase 3. The initial phase linked intra-hospital records, with the remaining phases primarily linking inter-hospital movements (not all hospitals assign a unique medical record number to a patient).

Among the 20,005 cases, 82 had gender inconsistencies, of which 59 were assigned a gender, and 23 were left undetermined. In 598 cases, the dates of birth were found to be inconsistent among the records. After review, 343 dates of birth were determined and 255 were left undetermined. There were 13,367 (67%) cases with one discharge, 3,168 (16%) with two discharges, and 1,553 (8%) with three discharges. Major expected payors at the time of the first discharge were Medicaid and Blue Cross, with frequencies of 42% and 27%, respectively.

Evaluation. All the results in this section relate to the study of the 202 Medicaid case sample, except where noted.

Linkage Accuracy. In 11 of 202 HIVLF cases, multiple (two different) MRNs were present. By searching the DSS file for the patient name by MRN, it was found that in all 11 cases the patient's records were correctly linked. This indicates that the medical record number and address linkage phases accurately linked records from the same patient.

Case Completeness/Discharge Records. The 202 HIVLF cases contained 76% of all the DSS records. This proportion was adjusted to eliminate from consideration those DSS records that were not associated with HIV infection. Elimination of such records from the DSS file resulted in the HIVLF cases containing 88% (±4%) of the DSS HIV records. This is a conservative estimate, as it assumes that all non-HIV-related DSS records not found in SPARCS are HIV related.

HIVLF case completeness, measured by number of discharges per case, was performed using the DSS HIV records for comparison. The average completeness of HIVLF cases with more than one record was as follows: 81% for the 49 cases with two records; 81% for the 23 cases with three records; 83% for the 12 cases with four records; 64% for the five cases with five records; 92% for the eight cases with six or more records. Of the 202 HIVLF cases, 162 (80%) were 100% complete, 169 (84%) were at least 75% complete, and 195 (97%) were at least 50% complete.

Cases paid by Medicaid and Blue Cross were more complete than the remainder of the cases, due to the matching phases which linked records by the patients' insurance company information. To assess the effect of the specific payor linkage phases, the Medicaid recipient number phase was eliminated for the 202 cases. The percentage completeness was reduced by 3%, to 85% (±5%). Assuming homogeneity among all payors, 85% (±5%) is considered to be the completeness estimate for the 1985 portion of the HIVLF cases.

Extra Cases in the HIVLF. Of the cases from the Medicaid HIVLF sample, 11 were extra cases which arose from unlinked records. This suggests that the proportion of extra Medicaid cases in 1985 is approximately 5%. If the other patients have admission patterns similar to the Medicaid population, the estimate of extra 1985 cases is 5% (±3%), plus the percentage of extra cases due to the absence of a linkage phase for the non-Blue Cross and non-Medicaid cases. Approximately one third of the cases were not reimbursed by either of these two payors. To estimate the number of extra cases present for other payor type cases, the effectiveness of phase 2 in linking Medicaid cases was examined. The Medicaid linkage in phase 2 produced 16% fewer Medicaid cases. If the remaining payor type linkage phases produced results similar to Medicaid, a 10% (±4%) estimate of extra cases in the 1985 HIVLF is obtained, ie, 5% + (⅓ × 16%). The effect of the Blue Cross linkage phase was examined and found to decrease the number of Blue Cross cases by 22%. This is consistent with the Medicaid estimate.

Accuracy of HIV Coding in SPARCS. The larger question of the completeness of SPARCS in identifying HIV discharges was partially addressed by comparing the number of *Pneumocystis carinii* pneumonia (PCP) cases identified in the HIVLF to the diagnostic classification of cases to the AIDS case registry for the same time periods. For the period 1983 through 1986, the HIVLF contained 9% more PCP reports than did the AIDS Registry (Bureau of Communicable Disease Control, NYS Department of Health, unpublished data). However, the HIVLF includes a 2% background level of non-AIDS PCP based on 1980–1981 SPARCS PCP discharges, with the excess per year ranging between 1% and 14%. This is consistent with the 10% (±4%) overestimate of cases presented previously.

Examples of Uses of a Longitudinal File. In order to demonstrate the capabilities of the HIVLF, provided below are examples of its ability to generate the necessary information identified earlier.

New Patients Entering the Acute Care System. Table II presents the number of HIV patients aged 13 or over hospitalized for the first time, on a semi-annual basis. The data presented are limited to the period 1984–1986, during which the cases were identified with a particular time period based on the admission date of their first HIV-related stay. Information from 1983 is not displayed, as a patient's first hospital stay might be erroneously identified due to the absence of 1982 information. The results from 1987 are not displayed, as they represent cases that are not well linked due to the absence of bills for the 1987 stays. Therefore, the results are shown for those individuals who, for at least one year's experience, were not previously seen with an HIV-specific diagnosis. The number of new patients has increased 4-fold between the first half of 1984 and the last half of 1986. Given the limitations of the linkage process, the absolute number of new patients is better estimated by reducing the estimates by 10% (±4%) to reflect the inability to link all records from the same patient.

Demographics of HIV-Infected Patients. Also included in Table II are the residence, gender, and age attributes of the individuals aged 13 or over first hospitalized with HIV-related conditions from 1984 through 1986. An increase in the female proportion of cases is visible over this three-year time period.

Changes in Diagnosis Over Time. Another valuable use of the longitudinal file is to identify the illnesses experienced by HIV-infected patients during their hospital stays. For example, the development of PCP and Kaposi's sarcoma (KS) following the first HIV-related hospital stay was obtained from the HIVLF by following patients from their first hospital stay through their last available stay. Data were limited to patients first diagnosed in either 1984 or 1985, and thus include at least one year subsequent to the first HIV-

TABLE II. Age, Gender, and Residence Characteristics of Individuals, Aged 13 and Older, When First Hospitalized with an HIV-Related Condition

	Date of First HIV-Related Admission					
	1984		1985		1986	
	Jan–June	July–Dec	Jan–June	July–Dec	Jan–June	July–Dec
Number of cases	977	1,342	1,791	2,349	2,925	3,574
Gender (%)						
Male	88.0	87.9	85.1	85.4	84.6	83.3
Female	11.8	12.1	14.5	14.5	15.3	16.6
Unknown	0.2	—	0.4	—	0.1	0.1
Residence (%)						
New York City	80.9	82.2	81.1	81.1	79.8	79.0
Rest of state	15.3	15.4	15.3	15.9	16.4	16.9
Out of state	3.9	2.5	3.6	3.0	3.8	4.1
Age (%)						
13 to 19	0.9	0.8	0.6	0.5	0.5	0.7
20 to 29	21.2	21.1	19.7	20.5	19.4	19.4
30 to 39	45.9	44.9	48.6	47.4	49.7	50.3
40 to 49	21.1	21.3	21.5	22.3	20.9	19.8
50+	11.0	11.9	9.7	9.3	9.5	9.9

identified hospital stay. The patients were divided into four groups based on the diagnoses identified during their first HIV-identified hospital stays: PCP and KS simultaneously; PCP without KS; KS without PCP; and other HIV-related diseases. Among those initially identified with only PCP (N = 1,904), 18% eventually developed KS; among those presenting with KS alone (N = 397), 47% went on to develop PCP; and for those with other diseases (N = 1,743), 14% developed only PCP, 4% developed only KS, and 16% developed both PCP and KS (not necessarily simultaneously). The percent increase in PCP was 22%, and KS increased by 70%, while the combination of both PCP and KS increased by 118% over the initial 200 cases. Given that some patients are still alive and may yet require hospitalization for PCP or KS, and due to technical limitations associated with record linkage, the changes are in fact larger.

Shift in Reimbursement Source. Patients with more than one discharge during the 1983-1987 period were reviewed for change in reimbursement source. Of the 2,855 patients who indicated private sources in their initial hospital stay, it was found that 17% shifted to government reimbursement in their last discharge.

DISCUSSION

Comparison of 1985 data from the HIVLF to the Medicaid file revealed, that, on average, 88% (±4%) of the Medicaid discharges per case were identified by the HIVLF. An estimate made for the 1985 portion of cases indicated that the average number of records per case is 85% (±5%) of the true number. Since a large number of cases are single record cases, this small reduction in completeness is not surprising. The result is consistent with the estimated number of extra cases.

Based on the sample studied, the HIVLF is consistent with the AIDS Registry in identifying the number of PCP cases reported from 1983 to 1986. However, we have not calculated the extent of underreporting of HIV discharges to SPARCS. In the earlier years of the HIVLF, identification of non-AIDS HIV-infected individuals was unreliable, and the degree of underreporting is unknown. This will lead to an underestimate of HIV-infected individuals and discharges in the HIVLF.

There is an overestimate of cases due to the inability to link all discharges of all cases. This is particularly true for cases with payors other than Blue Cross and Medicaid, since only Blue Cross and Medicaid had specific linkage phases. The proportion of extra cases was estimated to be 10% (±4%) among the cases with 1985 discharges. The proportion of extra cases from 1987 discharges will be higher, since matched bills were not available for that year.

Care must be taken in using this file for analysis. Discharges of patients who visited different hospitals and did not have matched bills would not be linked using the current methodology due to the absence of payor or address information. This may lead to underestimation of multihospital use on the part of HIV-infected patients.

Based on our experience in developing and evaluating this longitudinal file, there are some additional steps we will perform when a new version of the file is prepared. The new version will include several improvements: the addition of other linkage phases for payors such as Medicare and commercial insurers; additional evaluations of the cases, such as for the presence of any hospitalization overlaps among stays from the same case; addition of other case-related variables such as race or total cost from all stays; and inclusion of all stays for a case independent of the presence of HIV infection.

The HIVLF is a useful tool to analyze acute care utilization patterns of individuals identified as infected with the human immunodeficiency virus. Similar files can also be constructed to identify the characteristics of individuals associated with other diseases. There are distinct advantages in assessing the health care system using this type of file: continuity—data have been collected in the same manner for several years; timeliness—data are gathered and submitted as an ongoing process, information is available at predictable points in time and, based on historical patterns, the approximate portion of data that will be complete at successive monthly intervals is known; cost—creation of the longitudinal file is a cost efficient way to collect and analyze data, ie, the data are already available. Once the computer programs are prepared, rerunning the generation and analysis programs is a straightforward task. As the need for timely information increases and the technology is available to access large databases, it is imperative that the health care analyst be creative in the use of existing information to answer public health questions. States with similar discharge data systems should consider developing longitudinal files as an alternative to developing new primary data collection systems.

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Current trends in routine newborn male circumcision in New York State

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ABSTRACT. Despite the fact that circumcision of the newborn male has been practiced for at least 6,000 years, the procedure remains controversial. Circumcision rates in the United States remain among the highest of any nation in the industrialized world, yet there is little descriptive data on the demographic characteristics of newborns who are circumcised. New York State's hospital discharge data were used to examine demographic patterns for the procedure for the period 1980–1986. Circumcision was less common in New York City than in other areas of New York State, and was more common in private hospitals than in public hospitals. Whites had the highest rate of circumcision (70%), while Hispanics had the lowest (25%). Newborns under Medicaid coverage were more likely to be circumcised in voluntary hospitals than in public hospitals (45% vs 33%). We conclude that circumcision rates vary depending on a variety of factors, some of which may be attributable to the physician and others to family and society.

(NY State J Med 1990; 90:243–246)

Male circumcision has been practiced in various rituals for at least 6,000 years.[1] For decades there has been an ongoing debate concerning the merits and utility of routine newborn circumcision. Critics of circumcision, citing statistics from other countries,[1-3] complication rates,[4-6] "human suffering,"[7,8] and economic costs,[2,9] claim that the procedure serves no beneficial purpose. Others, citing medical studies, claim that the surgical procedure decreases the incidence of sexually transmitted diseases,[10,11] decreases the rate of neonatal urinary tract infections,[12-14] decreases the incidence of certain types of cancer,[15-17] and decreases the transmission of AIDS.[15,18]

In 1975, the Ad Hoc Task Force on Circumcision of the American Academy of Pediatrics[19] declared the absence of any absolute medical indications for routine circumcision of the newborn male. But this did not end the controversy. Apparently, there was little change in the practice of circumcision after the issuance of the task force's findings.[3] The circumcision rate in the United States remains among the highest of any nation in the industrialized world.[1,20] The controversy continues regarding the medical benefits of circumcision, with the American Academy of Pediatrics agreeing, in February 1988, to reconsider the question of medical indications. This paper describes current trends in routine neonatal circumcision of males in New York City and New York State.

METHODS

Data for 1980 through 1986 were abstracted from New York State's Statewide Planning and Research Cooperative System's (SPARCS) hospital discharge information data. This system was initiated in 1980. For this study, data have been utilized for all the years for which the records are complete. All discharge diagnoses are coded using the DRGs/ICD-9-CM system. Overall, in New York State, there were approximately 71,000 circumcisions per year performed during the study period.

The study population consisted of the 880,475 male newborns in New York State that were born between 1980 and 1986, of which 394,523 births occurred in New York City. Data on male circumcision within New York City were analyzed by hospital type (public vs private), race, payment category (Medicaid vs other), and calendar year. Proprietary and voluntary hospitals were combined to form the "private hospital" category, and municipal hospitals, which are mandated to care for all patients regardless of ability to pay, were categorized as "public hospitals." Since there is no state equivalent to city public hospitals, the New York State analyses were not as extensive. Race was categorized into four groups: Hispanic, non-Hispanic white, non-Hispanic black, and Asian. Information regarding religion was not available from SPARCS or from birth certificates.

Type of payment was categorized into two groups: Medicaid and "other." Within private hospitals, most people in the "other" category were covered by some type of insurance, while in the public hospital the "other" group consisted of a great many uninsured patients.[21] We were unable to separate out the uninsured patients.

For analytic purposes, we used the Statistical Analysis System (SAS) and performed chi-squared tests for bivariate comparisons and analysis of variance, with an arcsine transformation of proportions for multiple comparisons. Differences were considered to be statistically significant if they would have occurred by chance less than one time in 20 ($p < 0.05$, two-tailed test).

RESULTS

For New York City and New York State, the proportions of newborn males who were circumcised in the hospital during the seven-year study period are shown in Table I. The overall hospital circumcision rate in New York City has remained relatively stable at about 45%. During the same period, there was more variability in New York State rates (exclusive of New York City), which were substantially higher in every year studied.

Of the total 394,523 male births in New York City between 1980 and 1986, 36% of the babies were white, 30% were black, 23% were Hispanic, 4% were Asian, and the remaining 7% were either of un-

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TABLE I. Hospital Circumcision Rates (%) by Year, 1980–1986*

Year	New York State	New York City	New York City Public Hospital			New York City Private Hospital		
			Total	Medicaid	Other	Total	Medicaid	Other
1980	67.0	44.9	37.9	37.2	38.7	47.9	43.9††	48.9
1981	62.5	45.2	38.9	37.0	41.2	47.9	45.7††	48.5
1982	60.7	44.3	34.8	32.8	37.9	48.5	43.3††	50.0
1983	70.6	46.5	33.9	33.2	35.0	52.2	45.6††	54.2
1984	71.3	47.8	32.9	32.0	34.1	54.8	46.6††	57.3
1985	69.6	45.5	32.2	30.5	34.8	51.6	45.0††	53.5
1986	70.2	43.5	30.6	29.3	32.9	49.9	44.5††	51.3
Total	67.5	45.4	34.1	32.8	36.3	50.5	45.0††	52.0

* Source: New York State Department of Health (SPARCS).
† New York State excluding New York City.
†† Differences between public and private hospitals among Medicaid patients are statistically significant.

known race or were in a very small category; this latter group was excluded from this analysis.

In New York City, during the seven-year study period, 31% of births occurred in public hospitals, 61% occurred in private facilities, and the remainder occurred in other locations. The overall circumcision rate in public hospitals decreased during this period from 38% to 31% ($p < 0.001$), while the rate in private hospitals increased from 48% to 50% ($p < 0.001$). The circumcision rates in private hospitals within New York City were significantly higher than the rates in public hospitals for each of the years studied ($p < 0.001$). These p values are based on a test of the proportion circumcised in each of the hospital types.

As can be seen from Table II, there are sizable differences in circumcision rates between each of the racial groups in each of the years studied in both the state and the city. For every racial group, other than Asians, the state rate exceeded the city rate. In both the state and the city, Hispanics consistently were the least likely to be circumcised, and blacks and whites were consistently most likely, while Asians had intermediate rates. This pattern was true in each of the seven years.

When circumcision rates within specific racial groups are examined separately for births in public and private hospitals, the rates tend to follow the general yearly trends within these facility types (Table I), although the relative differences between the races remains the same. That is, the circumcision rates in public hospitals generally decreased over time, and the rates within each ethnic group born in these facilities also tended to decrease. Within private facilities, the overall rate increased slightly during the seven-year period, and circumcision rates within each of the ethnic groups at these facilities followed the same general trend. For each race group, circumcision rates were higher in private hospitals than in public hospitals.

Circumcision rates in New York City varied by hospital type and payment category: 34% of males born in public hospitals were circumcised, compared with 51% born in the private voluntary hospitals ($p < 0.001$). Newborns covered by Medicaid comprised a larger share of births in public hospitals than in voluntary hospitals (61% vs 22%, $p < 0.001$), but were more likely to be circumcised in the latter (33% vs 45%, $p < 0.001$).

In private hospitals, newborns in the "other" category had a significantly higher circumcision rate (52%) than the Medicaid group (45%). In public hospitals, the effect of payment category was less pronounced.

Since race is a major predictor for circumcision, the question arose as to whether there is a social class difference within the races. To examine this, we compared rates within the races according to payment type. This was done in the private setting only, in which the Medicaid and "other" categories could be used as proxies for social class. That is, a comparison of circumcision rates within any race group using the two payment categories would help determine if there was a social class difference.

Among blacks and whites in the private setting, rates did not differ by payment category, and presumably by social class. For example, within private hospitals, 69% of blacks in both payment groups were circumcised, and among whites the rates in the two payment groups were also equivalent.

On the other hand, among Hispanics and Asians in private hospitals, payment category made a large difference in circumcision rates. In 1981, 26% of Hispanics in the Medicaid group were circumcised, compared with 40% in the "other" category; for Asians the rates were also different—61% and 52% respectively. For public hospitals, in which payment category does not differentiate the social classes well, rates within the races did not differ by payment group.

DISCUSSION

Circumcision is the most commonly performed surgical

TABLE II. New York City (NYC) and New York State (NYS) Hospital Circumcision Rates (%) by Race, 1980–1986*

	White†				Black				Hispanic				Asian			
	New York City		New York State		New York City		New York State		New York City		New York State		New York City		New York State	
Year	%	(No.)	%	(No.)	%	(No.)	%	(No.)	%	(No.)	%	(No.)	%	(No.)	%	(No.)
1980	63	(10,188)	77	(39,421)	62	(9,484)	65	(3,047)	28	(2,719)	36	(302)	60	(916)	50	(208)
1981	64	(9,582)	71	(38,263)	63	(10,102)	57	(2,909)	28	(3,332)	37	(350)	60	(1,083)	50	(255)
1982	67	(9,380)	68	(38,090)	61	(10,404)	56	(3,142)	26	(3,376)	38	(451)	56	(1,190)	44	(232)
1983	73	(9,950)	79	(44,016)	61	(10,409)	70	(3,992)	28	(3,888)	41	(549)	58	(1,316)	48	(274)
1984	79	(9,975)	80	(44,875)	61	(10,602)	70	(4,134)	29	(3,842)	42	(601)	55	(1,389)	51	(295)
1985	74	(9,753)	78	(44,758)	60	(10,679)	69	(4,248)	28	(3,654)	43	(627)	54	(1,418)	50	(319)
1986	74	(8,833)	79	(44,556)	59	(10,644)	69	(4,290)	23	(3,518)	43	(585)	49	(1,327)	48	(313)
Total	70	(67,661)	76	(293,979)	61	(72,324)	65	(25,762)	24	(21,330)	41	(3,465)	55	(8,639)	49	(1,896)

* Source: New York State Department of Health (SPARCS).
† White exclusive of Jewish births (see text).

procedure in New York State. The medical and economic utility as well as the cultural and ritualistic nature of circumcision have been amply addressed in the literature.[22,23] By far the greatest number of routine male circumcisions are performed in the hospital by a physician shortly after birth. In addition, it has been suggested that approximately 10% of males not circumcised at birth will eventually require circumcision.[2]

In routine newborn circumcision, unlike most other surgical procedures, the newborn patient is obviously never able to consent to or be part of the decision making process. As such, parental cultural preferences and prejudices, presumed health benefits, and aesthetic choices of family, physicians, and society play a large role in determining whether the procedure is done. Studies suggest that the single most important determinant of whether a newborn male will be circumcised is the physician's attitude.[2,6]

Notwithstanding anecdotal reports that physicians are increasingly reluctant to recommend or perform circumcisions, there has been no significant decrease in circumcision rates in New York during the seven years studied. However, payment category, race, and type of hospital are predictors of circumcision rates in New York City.

Across all groupings, the circumcision rates in public hospitals are lower than those in private hospitals. Possible explanations include staff and equipment limitations and a high demand for space at public hospitals, as well as the fact that physicians working there are salaried and are not reimbursed on the basis of the number of procedures performed. Similarly, the low rate of reimbursement for Medicaid newborn circumcision ($12 vs an average physician fee of $175 for the procedure) may explain the difference by insurance type in the voluntary hospitals.

Race and ethnicity clearly play a major role in predicting circumcision. Many Jews, who currently represent 12% of the population in New York City and 7% of the population in the remainder of New York State (personal communication, P. Ritterband, City University of New York, 1988), traditionally have been circumcised after hospital discharge in religious ceremonies. Because data on religion are not available from SPARCS, the number of Jewish births and the number of ritual circumcisions performed shortly after hospital discharge are missing from this analysis. To include these babies in the denominator (live male births) of white rates, but to be unable to count them in the numerator (number of circumcisions), would result in an underestimation of the white circumcision rate. By making an assumption that Jewish births account for approximately 12% of all births (47,378 male births for the six-year period), we are able to make a rough estimation of the non-Jewish white circumcision rate in New York City (70%) by decreasing the denominator by 12%. The same was done for New York State (excluding the City) using the 7% estimate. However, because there are a number of Jews who are circumcised in hospitals, this calculation over-estimates the white non-Jewish circumcision rate by including some circumcised Jewish male newborns in the numerator but not in the denominator. Given the assumption that Jews account for 12% of all births in New York City, it appears that the white, non-Jewish circumcision rate is roughly equivalent to the black rate.

The rate of circumcision in New York State is higher than that in New York City, possibly because there are fewer Medicaid patients, and a smaller percentage of Hispanic patients born outside New York City. Hispanics in both types of hospitals, in both pay categories, in both the city and the state, and in every year examined, had a lower circumcision rate than any other race. If physician attitude is indeed the most important determinant of circumcision, we would expect that all patients in a public hospital, where there is little, if any, opportunity to select a personal physician, would have similar circumcision rates regardless of payment category or race. But this is not the case; in the public setting, there are marked differences within ethnic groups according to payment category.

Possible explanations for the low circumcision rate among Hispanics may be the existence of language barriers to a full and complete discussion of the risks and benefits of the procedure (with patients opting not to have their child undergo a procedure they do not understand). Another explanation may be that if the father was not born on the US mainland, as is often the case, he is less likely to be circumcised, and parents may prefer to have their sons physically resemble their fathers. However, why this is not true for Asians and blacks, among whom circumcision rates have also been traditionally low, is not clear.[24] Furthermore, it is not clear why such high rates occur among blacks. Regardless of hospital type and type of payment, blacks continue to have a high rate of newborn circumcision.

Type of payment was also an important predictor of circumcision. Due to limitations in the data collected, we could only identify two payment groups, "Medicaid" and "other." However, "other" in public hospitals represents primarily those with no insurance, while "other" in private hospitals is a more economically diverse group composed predominantly of those with private insurance, but it also includes some with no coverage.[25]

Among blacks and whites in the private setting, rates do not differ by payment category, indicating that social class is not a factor in these groups. However, for Asians and Hispanics within private hospitals, the circumcision rate for Medicaid patients is lower than for the "other" group in every year studied. This indicates that for these groups, the middle and upper classes are more likely to have their babies circumcised. There is no ready explanation for this disparity in the effects of social class on a family's decision regarding circumcision. It appears that those in the middle and upper classes are less likely to be influenced by traditional ethnic preferences.

In conclusion, circumcision rates vary greatly depending on a variety of factors, some of which may be attributable to the physician and others to the family and society. Although this study provides no answers to the important question of whether healthy male newborns should receive a routine circumcision, it does examine the current and recent practice of this surgical procedure in New York State and City. Whether these trends are unique to New York is a topic for further study.

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GENITAL CANCER IN JEWS

History of circumcision. In discussing aspects of Jewish sex life in biblical Egypt, Lewinsohn suggests that circumcision, which for two thousand years was counted as a reproach, was originally and is again now regarded as a sign of progress.

The origin of the practice is lost in the mists of prehistoric time. Some hold that the operation is a survival of the days of human sacrifice and symbolic in the purging of an infant of the sins of his forefathers. In primitive cultures it is a test of courage, a ceremonial of admission to manhood, of elevation to full membership in the tribe, and other variants of ritualistic custom. Frazer, in *The Golden Bough*, speaks of the religious type of tribal ritual practiced in this regard in New Guinea; Australian Bush; Maori, New Zealand; and other primitive areas. I have seen the ceremonial huts and some of the paraphernalia used in Micronesian villages of the South Pacific islands where the barbaric custom of infibulation and circumcision of male and sometimes female children was practiced on adolescents between the ages of nine and thirteen years.

Certainly the thought that Jews introduced circumcision to distinguish themselves from other peoples is untenable, for most of the peoples with whom they came in contact were themselves circumcised, and during their stay in Egypt they found their uncircumcised condition a disadvantage and an embarrassment. The Egyptian doctor-priests adopted the custom which was current throughout most of Africa, as a hygienic ritual. Moses, who was a protégé of the court and had entree to educated circles, seemingly never thought much of the fact that a long foreskin stamped one as a barbarian. He was never circumcised himself, although his wife, Zipporah, daughter of a priest of Midian, circumcised his son.

It was only after Moses' death that Joshua issued an edict for all Jews to be circumcised, referring for support of the idea to the fact that Abraham, of Ur, near Babylon where circumcision was not generally practiced, and all the males of his household had undergone the operation after his visit to Egypt. For long it was considered a requirement of cleanliness and therefore of ritual, for what was unclean was an abomination of the Lord.

This was still the point of view in Joshua's day when he revived the custom. Jews were being circumcised and despised the Philistines for being uncircumcised as they themselves had been despised by the Egyptians for the same reason!

Gradually, however, many Eastern peoples, beginning with the Phoenicians, gave up the practice. But the Jews retained it even in the Diaspora. Thus a measure which began as a sign of assimilation with adaptation to more civilized peoples, became a characteristic derided by the uncircumcised as a barbaric atavism!

LIONEL SANDLER AUSTER, MD
(*NY State J Med* 1965; 65:266-280)

REVIEW ARTICLE

The current hospital crisis in New York City and policy options for resolving it

KENNETH E. THORPE, PHD

Health care in New York City is widely perceived as suffering from three major problems. First, a capacity problem is evidenced by overcrowded emergency rooms and long queues for inpatient services. Second, many hospitals have shortages of staff ranging from supervising physicians to registered nurses to technicians. These staff shortages prevent expansion of services to meet demand and sometimes jeopardize quality. Finally, most private hospitals are experiencing operating deficits that threaten the continuation of some services and deplete capital assets. Together, these shortages of money, staff, and capacity create the image of an industry in crisis.

The first part of this article analyzes the magnitude of these problems and identifies their underlying causes. The latter part presents policy options to alleviate the problems and assesses their costs and benefits. Because legislation and regulations of the State of New York are important determinants of health policy in New York City, the options relate to state as well as municipal priorities.

THE PROBLEMS AND THEIR CAUSES

Overcrowding. Crowded emergency rooms and long waits for inpatient admission have become commonplace among New York City hospitals. The two phenomena are closely related. Emergency rooms become crowded because no beds are available for arriving patients who require hospitalization. The bed shortage also requires patients seeking elective care, such as cosmetic surgery or postponable operations, to wait long periods.

That the emergency room problem is basically caused by a shortage of beds instead of increased demand for emergency rooms is evident from the data presented in Table I. Between 1980 and 1988, emergency room visits decreased 3%; at the same time, hospital admissions from the emergency room increased 28%.

The underlying source of overcrowding is a convergence of increased demand for, and decreased supply of, inpatient care. Hospital occupancy rates are extraordinarily high. While New York City usually has occupancy rates above the national average, the discrepancy has grown in recent years (Table II). Until 1982, occupancy rates in New York City were approximately ten percentage points higher than the comparable national figure. Subsequently, national rates decreased sharply, falling from 75.6% to 65.5%. The city's municipal hospitals experienced the most rapid rise, from 82% to 92.5%. By 1988, New York City occupancy was nearly 30 percentage points above the national average.

TABLE I. Visits to and Admissions from Emergency Rooms, New York City Hospitals, 1980–1988

	Visits			Admissions		
Year	Total	Municipal	Other	Total	Municipal	Other
1980	3,349,131	1,356,026	1,993,105	—	—	—
1981	3,266,707	1,328,649	1,938,058	412,899	157,317	255,582
1982	3,269,251	1,290,538	1,978,713	433,440	166,308	267,132
1983	3,318,065	1,318,095	1,999,970	438,123	169,226	268,897
1984	3,333,567	1,304,557	2,029,010	469,915	174,470	295,445
1985	3,301,543	1,315,646	1,985,897	492,613	175,471	317,142
1986	3,262,431	1,239,756	2,022,675	515,418	175,149	340,269
1987	3,173,786	1,200,548	1,973,238	529,590	178,959	350,631
1988*	3,235,185	1,265,909	1,969,276	529,855	174,455	355,400

Source: United Hospital Fund of New York, Division of Research, Analysis and Planning.

* Based on actual data for 74 hospitals and estimated data based on 1987 actuals for hospitals.

The high occupancy rates in New York City can be attributed to both decreases in bed supply and increases in demand. The decline in bed supply is traced in Table III.

TABLE II. General Care Hospital Occupancy Rates, New York City and United States, 1980–1988

	United States, All Community Hospitals	New York City		
Year		Total*	Municipal	Voluntary
1980	75.6%	86.6%	78.9%	83.3%
1981	76.0	86.5	81.0	87.5
1982	75.3	86.9	82.0	88.0
1983	73.5	87.6	83.0	88.7
1984	69.0	86.9	84.0	87.9
1985	64.8	83.6	80.8	84.4
1986	64.3	82.4	81.0	83.4
1987	64.9	86.6	91.7	86.4
1988†	65.5	92.2	92.5	NA

Sources: National data from American Hospital Association, Hospital Statistics, 1986 and 1988 editions; New York City data from United Hospital Fund of New York, Division of Research, Analysis and Planning.

* Includes proprietary and state general care hospitals not shown separately.

† New York City total for 1988 is based on actual data for 74 hospitals and estimated data based on 1987 actuals for four hospitals.

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TABLE III. General Care Hospital Beds In New York City, 1980–1989

	Total					Municipal Hospitals				
	General Care					General Care				
Year	Total*	Medical/ Surgical	Obstetrics	Pediatrics	Psychiatric Care	Total*	Medical/ Surgical	Obstetrics	Pediatrics	Psychiatric Care
1980	33,596	28,723	2,018	2,855	2,812	6,629	5,279	512	838	1,367
1981	32,778	27,945	1,986	2,847	3,006	6,072	4,733	499	820	1,353
1982	32,656	27,863	1,914	2,879	3,074	6,078	7,753	499	826	1,368
1983	32,708	28,086	1,882	2,740	3,110	5,964	4,667	487	800	1,368
1984	32,051	27,522	1,882	2,707	3,179	5,770	4,515	494	761	1,401
1985	32,016	27,446	1,804	2,766	3,243	5,856	4,569	500	787	1,436
1986	31,251	26,776	1,805	2,670	3,305	5,871	4,578	500	793	1,565
1987	30,263	25,807	1,797	2,659	3,563	5,632	4,339	500	793	1,580
1988	29,427	24,980	1,797	2,650	3,691	5,241	3,948	500	793	1,635
1989	29,610	25,070	1,886	2,654	3,411	5,243	3,916	500	827	1,672

Source: See Table I. Figures for 1980 and 1981 are as of January 1; figures for 1982 to 1989 are as of December 31 of the preceding year.

* General care total excludes psychiatric beds.

Between 1980 and 1988, the number of general care hospital beds dropped 12%. At the municipal hospitals, one in five beds was closed.

This decrease was planned by the New York State Department of Health (DOH). The DOH has authority to regulate capital investments; hospitals cannot build or renovate facilities without its approval. To guide its investment decisions, the DOH developed and periodically revises a plan that includes demand estimates for inpatient care. It seeks to balance the number of hospital beds with its estimates of need.

When these planning efforts started in the 1960s, the consensus was that the city was "overbedded," and that reducing supply would both increase hospital efficiency and permit services to be concentrated in higher quality facilities. In 1985, the DOH projected a decrease of 21% in needed medical/surgical beds and 27% in obstetrics beds between 1985 and 1990.[1] The basis for this projected decline included expected reductions in average length of stay, fewer surgical admissions due to expanded ambulatory surgery facilities, and a relatively modest (in retrospect) increase in the average daily number of patients with the acquired immunodeficiency syndrome (AIDS) requiring hospitalization.

Through 1988, the number of beds was reduced largely in accord with the DOH plan, but the demand trends did not follow the plan (Table IV). After falling 11% during the 1980–1986 period, general care days rose 2.2% between 1986 and 1988. The post-1986 increase in demand has been felt most sharply by the municipal hospitals. The number of general care days provided by that system increased nearly 6%; the increase in psychiatric care was 10%.

The reasons for the unanticipated changes in demand trends are not yet fully understood, but several factors are clear. First, AIDS is necessitating more hospital care than was expected. Between 1987 and 1990, the number of AIDS patients in New York City hospitals increased from 1,071, or 3.5% of the average daily total, to 1,981, or 7%.[2]

A second factor is the unforeseen rise in demand for psychiatric care. This appears to be related to increased substance abuse, notably of the cocaine derivative known as "crack." As shown in Table IV, psychiatric care days

TABLE IV. General Care Hospital Days In New York City, 1980–1988

	Total					Municipal Hospitals				
	General Care					General Care				
Year	Total*	Medical/ Surgical	Obstetrics	Pediatrics	Psychiatric Care	Total*	Medical/ Surgical	Obstetrics	Pediatrics	Psychiatric Care
1980	10,468,894	9,121,979	NA	NA	955,271	1,833,364	1,483,256	NA	NA	NA
1981	10,353,004	9,014,578	796,128	542,298	1,019,572	1,797,650	1,450,705	208,951	137,994	449,396
1982	10,369,266	9,038,837	781,067	549,362	1,031,053	1,822,317	1,453,386	222,265	146,666	446,267
1983	10,319,272	8,993,049	777,695	548,528	1,029,586	1,795,955	1,418,930	222,126	154,899	449,079
1984	—	—	—	—	—	—	—	—	—	—
1985	—	—	—	—	—	—	—	—	—	—
1986	9,115,085	7,794,676	772,005	548,404	1,176,093	1,665,878	1,275,900	229,690	160,288	526,977
1987	9,298,755	7,958,444	770,390	569,921	1,248,060	1,754,942	1,360,632	230,149	164,161	556,836
1988†	9,312,632	7,969,711	761,683	581,238	1,166,312	1,760,036	1,364,057	233,711	163,068	579,314
Percentage change										
1980–86	−11.0%	−2.6%	—	—	23.1%	−4.0%	−14.0%	—	—	—
1986–88	2.2%	2.2%	−1.3%	16.0%	−0.8%	5.6%	6.9%	1.8%	1.7%	9.9%

Source: See Table I.

* General care total excludes psychiatric days.

† The totals for 1988 are based on actual data for 74 hospitals and estimated data based on 1987 actuals for four hospitals.

increased 13% between 1983 and 1988, including a 29% increase in the municipal system.

Third, the number of births has not fallen as anticipated. Obstetric care days rose 6% in the 1983–1988 period. There was also an increased need for intensive newborn care as the number of low birth weight infants and other infants with problems increased.[3]

Finally, length of stay has not declined as anticipated. The average stay among general care patients in New York City was 8.8 days in 1988, the same as in 1985. In contrast, the national average in 1988 was 7.2 days. The reasons include a shortage of nursing home beds that causes some patients to remain in hospitals while awaiting nursing home placement, but other factors also play a role.[4]

Staff Shortages. The city's hospitals are experiencing severe staff shortages, in particular, insufficient nursing and ancillary personnel. The nursing shortage is the most well documented of the health care industry's personnel problems. The nurse vacancy rate in large urban hospitals is estimated at 15%.[5] These unfilled positions translate into reduced services at the affected facilities, the use of temporary or "agency" nurses, or the use of nurses' aides in place of registered nurses.

The situation is similar within New York City. A 1989 survey by the Greater New York Hospital Association found a vacancy rate for registered nurses averaging 15%.[6] To fill the vacant positions would require an additional 5,400 registered nurses. With nurses' salaries averaging between $30,000 and $35,000, this suggests additional expenditures of between $162 million and $189 million annually.[7]

The vacancies are not spread evenly among the city's hospitals. Those with the greatest vacancies are in poorer neighborhoods, where many nurses do not want to work, and those hospitals with reputations for difficult working conditions, which includes many municipal hospitals. The shortage of nurses is particularly acute in the 11 municipal hospitals, where the number of full time registered nurses decreased 113% to 5,383 between 1986 and 1988. To partly offset this loss, the Health and Hospitals Corporation (HHC) increasingly relies on agency or per diem nurses. In June 1988, HHC employed 1,037 temporary nurses in its acute care hospitals, a 105% increase over the number in 1986.[8]

Nurses are not the only type of hospital personnel in short supply. Technicians who work on intravenous teams and phlebotomists, who draw blood samples, are examples of other skilled personnel in shortage. One estimate is that there are about 700 vacant positions in these categories.[9]

These shortages result from a combination of increased demand and reduced supply. The greater demand is due primarily to the more intensive care required by patients in hospitals. As the bed supply becomes tighter, the patients remaining in hospitals typically are more ill. At the same time advances in medical technology require more complex treatment methods. New types of diagnostic and surgical procedures require more sophisticated personnel. The combined effect is that more nursing and ancillary services are required for the average hospital patient.

The market would solve this problem by driving up wages, but as described more fully below, the state regulates hospital operating revenues. This makes it possible to raise nurses' salaries significantly only if the number of other personnel is reduced.

There is some evidence that hospital administrators recently have financed greater than average pay increases for nurses by cutting the number of other employees. In New York State, the average number of nonmedical, nonnurse hospital employees per 1,000 admissions declined from 7.6 to 6.9 between 1980 and 1987.[10,11] The HHC increased starting annual salaries for registered nurses from $25,000 to $28,750, or 15%, effective in the spring of 1988, and subsequent increments to maintain parity with private hospitals brought the figure to $29,235 in January 1989.[8] Nevertheless, shortages persist, which suggests that nonwage factors affect the local nursing shortage.

Financial Stress. According to commonly used measures of financial health, hospitals in New York City are among the sickest in the nation. In 1988, the city's voluntary hospitals had an average operating loss of 3.6%, with 38 of the 49 hospitals reporting operating deficits; in contrast, nationally, hospitals approximately broke even (Table V). This pattern of poor financial results has characterized New York City voluntary hospitals since at least the late 1970s.[12]

Like any enterprise, a hospital sustains an operating loss when its expenditures exceed its revenues. Do New York City hospitals spend too much or have insufficient revenues? As indicated in Table V, the problem is not rapidly escalating expenditures. Except for 1984, increases in average expenses per admission in the city have trailed the national average. Over the 1980–1987 period, they averaged 9.0% annually in New York City versus 11.3% nationally.

The latest turnaround in hospital financial condition can be related to four factors. First, Medicare became a less generous revenue source. Actions by Congress and the federal Department of Health and Human Services

TABLE V. Selected Characteristics of Hospitals in New York City and the United States, 1980–1988

	Operating Margin		Percent Increase In Expenditures Per Admission	
Year	United States*	New York City†	United States	New York City
1980	0.3%	NA	12.8%	11.4%
1981	0.2	−4.2%	17.3	12.4
1982	0.7	−3.9	15.2	6.8
1983	1.0	−2.3	11.5	10.8
1984	2.0	−2.2	7.4	11.8
1985	1.5	−3.0	8.3	5.9
1986	0.7	−0.7	8.9	6.0
1987	0.1	−1.8	9.0	6.6
1988	0.0	−3.6	NA	NA

Sources: Expenditure data from American Hospital Association, Hospital Statistics, 1981–1989 editions; national operating margins are American Hospital Association, National Hospital Public Survey data as reported in Prospective Payment Assessment Commission, Medicare Prospective Payment and the American Health Care System, Report to the Congress, June, 1989, p 99; New York City operating margins are unpublished data provided by the Hospital Association of New York State.

* United States data for all community hospitals, and represents the American Hospital Association's patient care margin.

† New York City data for voluntary hospitals only.

(DHHS) kept increases in payment rates below hospital cost inflation in each of the last three years, cut the payments for indirect teaching costs, reduced the payments for hospital capital costs, and established less generous payments for "outlier" cases. As a result, hospitals no longer make a substantial profit from Medicare patients.

Second, the state tightened its already strict payment rules for the revenue sources it controls. In 1988, the state shifted from a per diem to a per case system of payment. This proved financially difficult for hospitals with high average lengths of stay. The state also maintained a low base figure for establishing its basic rates and kept its annual adjustments to these basic rates relatively low. Specifically, the state continues to base its payments on hospital expenditures in 1981. The 1981 costs are increased by an inflation factor that is linked to overall national inflation rather than hospital specific costs, and that makes no allowance for changes in medical technology. While this strategy has limited hospital cost growth, the current per case payment rates are significantly below most hospitals' actual costs for treating the cases. Whether these additional expenditures improve the quality of care remains at issue.

Third, the state maintains controls on outpatient as well as inpatient payment rates for Medicaid. The 1989 maximum rate for a Medicaid clinic visit was $60, and emergency room visits receive at most $90—the same rates originally established in 1981 and 1987, respectively. In contrast, the average cost of an outpatient department visit among New York City hospitals is about $119.

Fouth, the revenues distributed by the state through the bad debt and charity care (BDCC) pool have not been sufficient to offset fully the hospitals' costs of treating uninsured patients. The decline in the proportion of bad debt and charity care financed through the BDCC pool is documented in Table VI. In 1985, more than 68% of all uncompensated care among New York City voluntary hospitals was financed through the pool. By 1987, however, only 27% of hospital uncompensated care was financed through the pool. The HHC facilities had only about 10% of their uncompensated care financed directly through the pool.

POLICY OPTIONS

This section reviews a series of options to address the problems confronting New York City's hospitals. It assesses their effectiveness in solving the three problems discussed above—financial stress, labor shortages, and overcrowding.

Changes in the Payment System. Several changes to New York's hospital reimbursement system are currently under discussion. These changes range from comprehensive reforms such as universal health insurance to incremental adjustments in the rate setting rules.

Universal health insurance. One proposal recently advanced by the New York State Department of Health would extend health insurance coverage to all New Yorkers.[13] The proposal, known as UNY*CARE, would use two means: a mandate that employers offer health insurance (or pay an assessment), and an expansion of Medicaid. The workplace approach would require that employers offer insurance for all full time workers and their dependents. Those not receiving workplace coverage and with incomes below 200% of poverty would become eligible for a subsidized health insurance policy. Under the proposal, all covered health care costs would be billed to a central agency, which would subsequently assign the costs to the proper insurance company.

TABLE VI. Uncompensated Care and Bad Debt and Charity Care Pool Payments to New York City Voluntary Hospitals, 1983–1987 (dollars in millions)

		Payments from		Percent of Care Financed by	
Year	Uncompensated Care*	Bad Debt Pools	All Pools	Bad Debt Pools	All Pools
1983	$230.3	$ 80.1	$101.2	34.8%	43.9%
1984	226.3	133.6	155.4	59.0	68.7
1985	266.1	181.8	198.1	68.3	74.4
1986	345.4	122.9	135.8	35.6	39.3
1987†	444.2	120.1	129.1	27.0	29.1

Source: Unpublished data from Office of Health Systems Management, New York State Department of Health. Totals include all payments from regional pools plus net payments to hospitals from the statewide pools.

* Uncompensated care represents inpatient bad debt and charity care plus adjusted (for government payors) outpatient department deficit.

† 1987 totals include $7.4 million contributed by New York City voluntary hospitals to statewide financially distressed pool. The distribution of these payments for 1987 to support capital projects is not currently available.

Costs of implementing UNY*CARE would depend on the scope of benefits provided to the uninsured and the payment rates negotiated with physicians. Estimates of the gross costs could exceed $6.3 billion, an 18% increase over current New York State health care spending. A major issue in the debate surrounding UNY*CARE is the "appropriate" distribution of these costs among the state, employers, and the currently uninsured. As currently planned, funds from the bad debt and charity care pool, as well as other state programs targeting the indigent, would be applied to reduce the added costs of the proposal.

Covering the uninsured would have a number of important impacts on the health care delivery system. First, extending coverage would result in additional demand for medical care services. National studies indicate that extending coverage to the uninsured could increase utilization by approximately 20%.[14] Extended coverage combined with higher Medicaid payments to physicians could, over time, shift a significant number of ambulatory care visits from hospitals to physicians' offices. This would reduce some overcrowding of emergency rooms. On the other hand, increased hospitalization among the newly insured would add to already long waits for inpatient admissions.

Universal coverage could significantly reduce hospital operating deficits. The extent of the reductions, however, depends on higher Medicaid payment rates for ambulatory care and a reasonably broad benefit package. Expanded Medicaid coverage also would substantially reduce the necessary level of New York City's tax levy support of HHC. In addition, potential reductions in hospital charges over time could generate significant savings to individuals covered under commercial health insurance plans. On the other hand, any significant expansion of Medicaid that leaves the existing ambulatory care payment structure in place and increases utilization would actually add to hospital deficits. Thus, the actual impact of the UNY*CARE proposal depends on the resolution of the key design issues of how to pay for ambulatory care and what benefits to cover.

Enhanced ambulatory care payments. It is possible to enhance payments for ambulatory care through Medicaid and the BDCC pool without simultaneously providing universal coverage. Such a measure could take the form of higher ceilings on Medicaid outpatient visit rates and higher Medicaid fees for physician office services.

The clearest consequence of such a policy would be to reduce hospital deficits. That portion of the deficit attributable to the gap between Medicaid payment costs and actual costs for Medicaid enrollees would be reduced or eliminated. In addition, if higher private physician fees reduced hospital outpatient department utilization by shifting some patients to other ambulatory care sites, this, too, would reduce deficits and possibly reduce aggregate expenditures.

The impact on overcrowding and staff shortages is less clear-cut. If, as some studies suggest, a more rational system of ambulatory care reduces need for inpatient care, then use of hospital beds should decline and overall waits for beds should be shorter.[15] A corresponding reduction in need for hospital staff also would occur. However, these anticipated consequences are largely speculative; it is possible that greater use of ambulatory care could have no impact on, or even reverse, inpatient utilization. Regardless of the impact on inpatient demand, however, an improved system of ambulatory care is likely to mean greater patient satisfaction and more frequent provision of primary care services.

Additional targeted funds. A more incremental approach is to continue the recent trend of providing targeted funds to hospitals to address specific needs. Two examples of this approach are the $225 million in funds recently provided hospitals to attract new ancillary personnel, to compensate for shorter hours of work by residents, and to finance supervision of residents during the evenings and nights. These additional revenues were distributed to hospitals based on state calculations of the funds required to comply with the state's new graduate medical education regulations. Another example is the additional resources provided individual hospitals, again based on estimates of need, to attract new nursing personnel.

This targeted approach is likely to achieve more modest results. Most directly, this approach represents a stop-gap solution designed to address immediate financing needs and state policy objectives. In addition, these revenues do not represent unrestricted support. Since these revenues must be spent for specific purposes, they will not have a perceptible impact on hospital operating deficits. While the hospital industry would prefer unrestricted support, the targeted approach allows the state to direct scarce revenues to needs high on its agenda. However, this approach, by itself, will do little to address the longer term financing issues facing hospitals in New York City.

Change the base year. Perhaps the most controversial aspect of New York State's rate setting program is the base year used to determine payment rates. Inpatient payment rates for calendar year 1990 still are linked to each hospital's "allowed" costs in calendar year 1981. With few exceptions, expenses exceeding the yearly trend factor are not recognized or reimbursed in subsequent years. While New York State's approach is truly prospective and has contained costs, there has been a substantial growth in expenses over allowed revenues. Certainly, a substantial portion of cost growth over the trend factor reflects management decisions. Remaining expenditure growth over the trend factor represents changes in practice patterns and new technologies not accounted for in the state's payment system.

Updating the base year would add a substantial amount of revenue to New York hospitals. Moving the base year to 1989 could provide approximately $700 million in additional revenues statewide. At issue from the state's perspective is whether expenditures over the trend factor represent managerial inefficiencies, deliberate decisions to run a deficit, or factors beyond the hospitals' control (such as more AIDS patients). Thus, all expenditures exceeding the trend factor are not likely to be recognized in future enhancements of the base year. As a result, the ultimate impact of updating the base year on hospital deficits will depend on the aggregate increase in revenues and their distribution across hospitals.

New hospital revenues provided by updating the base year would have little impact on overcrowding or staff shortages. The extra money is likely to be used by hospitals to reduce deficits from existing services rather than to expand bed capacity. Similarly, the revenue will not alter the ability to attract needed personnel.

In summary, some of the proposed payment changes could have a positive and substantial impact on New York City's hospital industry. The ability of proposals such as the state's proposed UNY*CARE program to address the access problems facing the uninsured and the financial issues facing hospitals largely depends on the scope of benefits provided and the ambulatory care payment rates established. None of the proposed changes in payments are likely to address overcrowding due to substantial growth in admissions through the emergency room. In fact, universal coverage proposals are likely to increase the demand for medical care, placing additional pressure on the delivery system. The recent trend toward targeted revenues may address specific shortcomings (such as persistent staff shortages), but they do little to improve hospital financial conditions.

Freeing More Beds. A major source of overcrowding is a shortage of acute care beds. Part of the solution may be to provide additional beds, but a higher priority should be to free acute care beds that are used for discretionary hospital admissions or for non-acute care functions. Several means to this end have been identified.

Expand outpatient care. Approximately 85,000 admissions and 365,000 days of inpatient care in New York City represent patients who are potentially treatable on an outpatient basis. Examples of such care include back and neck treatment, urinary tract infection treatment, and tonsillectomies. The relatively large number of discretionary hospital admissions indicates that the state and third party payors should improve their utilization review and case management efforts to achieve the shift. Moving these admissions outside the hospital would be equivalent to providing 1,000 new acute care beds.

Decrease lengths of stay. Despite the implementation of case payment for all third party payors, average length of stay in New York State is higher than the national average. A portion of these longer stays is traceable to a substantial number of patients awaiting placement for nursing home beds and home care.[16] Thus, one approach would be to expand the supply of nursing home beds and home care providers to reduce the queues, freeing up beds for patients waiting for acute care beds in the emergency room. These expansions would, of course, increase expenditures elsewhere in the system.

Upgrade the mental health care system. Much of the existing backlog in the emergency rooms involves patients waiting for acute care psychiatric beds. The recent surge in demand for psychiatric beds primarily involves drug and substance abuse problems. The rapid growth in the number of such cases requires additional outpatient treatment centers, as well as more comprehensive long term inpatient care in specialty hospitals designed to address the needs of this population. In the interim, the acute care hospitals, especially in HHC facilities, are forced to cope with the continued rise in psychiatric patients. Thus, an improved mental health care system would free hospital beds for active Medicaid care.

Reduce the treatment of nonmedical problems by hospitals. For many New Yorkers in need, the hospital represents a 24-hour assistance center. Thus, many patients in the emergency room do not require acute care treatment. Use of the emergency room and inpatient beds by homeless singles, boarder babies, and individuals with chronic health problems reflects broader failures of the city's social service agencies. Thus, in addition to improving its hospital and health care delivery system, the city also needs more foster care homes, better shelter and housing arrangements for low and moderate income families, residential drug treatment facilities, and more appropriate outpatient facilities to treat patients with AIDS. While providing additional hospital beds to address these needs may be politically feasible in the context of a perceived hospital "crisis," this approach may be less effective than directly confronting the range of social problems that are helping to create overcrowded emergency rooms and hospital bed shortages.

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He had a change of heart

IRVING A. BUNKIN, MD

> Our life consists of resurrections.
> —Pacifici, 1912

At one point in modern surgery, somewhere around the year 1960, many of us thought that we had reached the ultimate in technical skills. We had elegant support systems: good anesthesia, blood banks, antibiotics, and intensive care units. Courageous surgeons saw themselves in the vanguard of medical science fiction. Time was beginning to reduce the complex to the banal.

Along came the heart transplant era. In 1967, the first human transplant was performed in South Africa by Dr Christian Barnard. In 1986, more than 1,000 hearts were transplanted in the United States and in 1987 about 1,500. Each event was accompanied by an extravagantly structured script. Briefly co-starred in the story were the "brain-dead" donors whose hearts might be a gift to dying and potential recipients. Regional organ procurement centers in the background, with a computer network, located the "brain-dead." The data base contained waiting recipients to be matched. Cardiac surgical teams, each with emergency transport facilities and hospital stage, completed the production. A macabre event in the making! If one were to fantasize being present at a resurrection, vintage 1989, the heart transplantation scenario would be the most relevant. The drama would be entitled, "Life After Life."

In the lifetime of a physician concerned with the practice of internal medicine, it is unlikely that he or she will encounter a heart transplantee as a patient. This probably holds true even for the busy cardiologist.

Our subject, Chris (a fictitious name), nearing 50, the first and only transplantee I came to know, had had his first major myocardial infarction at the age of 40. Soon thereafter, however, he was able to return to productive living. The quality of his life in a highly complex and sophisticated workplace, a sedentary one, remained balanced and conventional for several years. Eventually there followed bouts of rapid heartbeat, which were disabling, and repeated hospitalizations. One of these paroxysms, during which there was loss of consciousness, was electrically controlled by a defibrillator brought to the scene. Chris referred to the incident thus: "I knew that I was dying. I was resuscitated by one of those 'jump-starts.' I used to have a chess player's brain, always two steps ahead and very methodical. Not any more. There must have been some brain damage. I'm adjusting. I don't worry."

There were recurrent attacks of fast and irregular heartbeat which did not respond to drugs . . . more episodes of constant dying.

Cardiac researchers discovered that the scarred area remaining in the heart after a blood vessel closure was the source of wildfire, electrical currents. Currents also arose in the muscle just beyond the scar. A new operative treatment involved removal of the scar and a significant area of adjacent inner heart muscle. Chris survived such a procedure and a regular heart rhythm reestablished itself. But the good result was only temporary. Stormy palpitations recurred. Uncontrollable heart failure ensued.

It was then that we met for the first time in a cardiac intensive care unit. It was a family support call with no more than an introduction and a handshake. To me the man appeared terminally ill. I returned several days later to find that he had left our unit for a transplant center nearby. Why the sudden move? He had risen to first place on their cardiac transplantation waiting list. Memorial Day weekend was coming up. There was bound to be a near fatality on the highways of the states surrounding the transplant center. Previous years' statistics encouraged this belief. The area's four-state computer network was always alert and prowling, looking for a potential brain-dead donor. Agonizing and contradictory thoughts for our patient. An eternity.

During the waiting period psychiatrists and social workers performed their piece. Their's was a tightrope balancing act over hope and grieving, dealing with hope for post-transplant survival and grief if a heart were not available in time to save Chris. This was preparation for both living and dying.

Finally, after about a week, at about 10:00 PM, the computer at the organ search center brought Chris and a brain-dead donor together. A good match. Correct blood type. No AIDS, syphilis, hepatitis, or other blood-transmissible diseases. A plasmapheresis was performed during the night. (The red and white blood cells were separated from his plasma and this latter fluid component of his blood was replaced with fresh bank plasma, ridding Chris of most of the "bad" antibodies.)

Meanwhile, two of the local heart transplant team of four were on their way by chartered plane to "harvest" the heart. Chris was in the home operating room being prepared by the other two surgeons for the arrival of the new heart. The patient would be transplant-ready.

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A mad night unto day. Other transplant teams in the computerized system had been alerted. It was their priority to remove the kidneys, liver, and corneas first while the heart was beating and maintaining blood supply to these organs. Always last, the heart team then skillfully removed the heart and placed it in hyperosmolar saline. In this solution the heart remained in a relaxed and noncontractile state. The transport container was a sterile plastic bag, enveloped in ice, carted home in an ordinary picnic box. The teams were in constant radio communication. (They alternated in "harvesting" and in the transplant procedure.) The surgically opened chest was almost ready back home. Air trip—500 miles as the crow flies. One hundred and ninety minutes from securing the heart to the moment of beginning the transplant. By now the transplantee heart had been without oxygen for about three hours. Transplant procedure about 45 minutes! As soon as the transplanted heart began to receive the recipient's own blood it began to beat on its own! (About 50% of the time these transplanted hearts require an electrical "jump start" to get them into a regular self-sustaining rhythm.) The "resurrection" had begun.

I had consulted with both of our patient's surgeons. An excerpt:

Me: "You are the last of the 'harvestors.' How does it feel to open the chest of a man who is still alive, but brain-dead, and to meticulously dissect out his heart?"

Surgeon: "Well, he's already minus his liver, kidneys, and corneas. At first I felt like a ghoul. Not any more. Because of the brain-dead donor's gift I envision unknown people walking around, somewhere, alive and in the mainstream of things. You once saw Chris terminal. Look at him now!"

Since the human body would recognize what was not its own it usually began a process of rejection. Cyclosporin and Imuran™ were therefore administered preoperatively and continued long after. So began on day one a lifelong vigil against rejection of the foreign heart. This required repeated heart biopsy. Shortly after transplant a first biopsy revealed beginning rejection. Five subsequent biopsies were negative. Our heart transplantee thus became a captive of the transplant surgeon.

Chris and his wife were my guests for lunch six months after the transplant. At such a confrontation how did one avoid pity or intrusive curiosity?

I met a tall, erect, apparently vigorous man, youthful and impeccably groomed. He grasped my hand firmly. At lunch he ordered a beer and a spicy New Mexican dish. His wife was quiet.

Affable and communicative, Chris said, "They use me to talk to the waiting transplant candidates because I look so good." Mostly small talk. But he was the aggressor. My strategy: listen. I listened as he spilled his story. When he and his wife departed for home about 60 miles away, I noted that he was at the steering wheel of the car.

There was a tape recorded interview one month later, seven months after the transplant. This time I was his luncheon guest. Two recent heart biopsies had again revealed rejection, necessitating more cortisone. A piece of his conversation: "I'm not as clear as I want to be. Maybe that's the cortisone. Today my blood pressure is somewhat elevated." (Impulsively, I took his pulse: 100 and regular.) He added, "Since the transplant I've been on a 'high'. Remember that my brain is now being supplied with much more oxygen than before the transplant. I'm usually on 'Cloud Nine'. Not today."

Looking for meaningful conversation, I said hesitatingly, "Did you pray during the night while waiting for the arrival of the heart?" He sobbed briefly and then said, "Yes, I did." There was a long pause and I followed with what I knew might be an imprudent, but needed question, "Do you look back with guilt, having waited hopefully for a brain-dead donor that week?" He answered, "No." Then he observed that the week of waiting and the preparatory night were a blur. He added, "Before leaving the hospital I wrote a letter of thanks to the family of the donor. Transplantees are advised to do that. The organ search center is the intermediary."

"What's on the agenda for your immediate future?"

"I'll be studying horticulture. On the first day home from the convalescent center I sat alone in a park looking at the grass. I wanted to sit under a tree and watch it grow."

My amateur psychoanalytic interpretation of this last remark was that the moments of solitude had been spiritual ones for meditation and celebration. Grass. Trees. Leaves on branches. All symbolic of life's renewal. Hadn't Chris been gazing at regeneration?

He continued, "You are not right about resurrection. I never died. I was told that my heart valves were to be used for transplant into a patient who needed them. So I was a living donor. Do you know that I now have two "P" waves in my electrocardiogram?" (In the technical procedure of transplant a rim of the patient's right auricle is left behind to facilitate connection to the donor's whole heart. The electrical impulses which drive the whole heart arise in this anatomical area. It is also responsible for the first blip in the electrocardiogram's wave complex. The donor's heart adds an identical, anatomical piece. Hence two impulses. Result: two "P" waves.)

From "P" waves we moved to a discussion of the reactions of his children to his heart transplant. (This initiative came from him.) The youngest, a ten-year-old son, was having difficulty in explaining his father's new heart to his fifth grade classmates. The older, a 17-year-old girl, was quiet and grim. And what about his wife? To avoid intrusiveness I did not pursue this avenue of inquiry.

Chris continued, "I know all of the statistics. I stand an 80% chance of being here one year from now. Rejection problems will be with me forever, at least with the present immunosuppressive drugs. We may have better ones in ten years. We transplantees are prone to hardening of the heart's arteries, especially after the fifth year. They'll be taking pictures of the arteries in my heart every year. Things may be brighter down the road."

I was learning.

"For the moment I am content with my life. I have a fixed routine. I am up at 5:30 AM, do my exercises, ride ten to 15 miles on my indoor bike, I do the house cleaning, shopping, and cooking. Mondays I do the laundry. My wife has had to go back to work."

I presumed that the program was the beginning of his return to the mainstream and eventually his former workplace.

"Doesn't the problem of rejection plague you?"

"No. Not much. When it occurs they'll treat it. I'm not depressed nor am I totally happy. There's no big deal in having a transplant. If the rejection is overwhelming, I could always have another transplant."

Had the heart transplantation infused Chris with a taint of capricious majesty? Maybe. Certainly he was a symbol of life-affirmation.

For days after our meeting I was overly aware of my own heart. Nights I was looking for the pulse in the ear that was against the pillow. One night I calculated that there already had been more than two and a half billion beats in my lifetime of 75 years. What a tireless bundle of muscle that heart is! And what a dependable electrical system in it!

I had been humbled and sobered. My professional eye had met man, one who would not let his new, unknown heart unsettle his soul. The birthday card Chris received from me on the anniversary of transplant day could not have produced a wrench in his heart because the new heart, obviously, was without nerve connections.

Talk with the heart transplantee brings a haunting confrontation. Feeling the pulse at his wrist locks one into the imaginative world of medical science fiction. One is silenced, unable to find the words to express a wonderment at a living reality.

FROM THE LIBRARY

TRANSPLANTATION AND THE LOCAL PHYSICIAN

Daily we hear about the fantastic achievements of our colleagues in the realm of transplantation. The most dramatic forays in this field undoubtedly involve replacement of the heart. I am sure the subject of the practitioner's role in facilitating this work is of considerable pertinence. He must perforce be part of the system, participating in it and making judgments which will bring the benefits of this realm of immunology, cardiology, and surgery expeditiously to the patient.

The role of the attending physician is vital. His judgment and participation in the clinical sphere, in decisions on which the patient and his family must agree, are of the utmost importance. An understanding of the complex factors that precede a decision for transplantation and a well-informed capacity for judgment must be considered our obligations in the future. . . .

. . . Are we in any way evolving these services to the detriment of prevention? Have we, in relationship to funds that are available, the right perspective on the problem? Have we, ethically and morally, given the correct stress to the over-all problem? The matter of transplantation, however dramatic, especially in relation to the heart, tends to get out of perspective. The drama of spectacularly curing something which should never have been allowed to happen does tend to overshadow, indeed completely obliterate, the urgency of the less dramatic causative agent.

Rheumatic fever still exists in Western New York; this disease, and congenital defects, which predetermine the problems for which transplantation (or indeed any cardiac surgery) is required should be tackled with the aggression they merit. If we are to look for financial support in the sphere of cardiac or any other form of transplantation, then we must do so as a composite attack on the congenital and acquired problems. Many of our patients would not demand transplantations had the root cause for their deterioration been tackled. . . .

It is unreasonable to underestimate the cost of anything these days, and if we are to make any judgment on the money required this should be done in relationship to previous experience which is considerable throughout the country, indeed throughout the world. The astronomical expense surely cannot be borne by the patient and the relatives. It will have to be decided that under no circumstances should the patient subjected to this procedure have to pay in part or in whole. If we solve the immunologic problem, which I think we can, and if we solve our team approach to the program, which is obviously possible, we must not fail to solve the problem of cost to the patient, who always comes first.

The rehabilitation services which embrace several disciplines, psychiatric and others, have to be given their part in the over-all plan. Lip service to their part in returning the patient to useful life is no longer acceptable. . . .

JOHN R. F. INGALL, MD
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Diphtheria and medical therapy in late 19th century New York City

VIRGINIA A. METAXAS QUIROGA, PHD

Diphtheria was a major epidemic disease in the last third of the 19th century, leading many New York City pediatricians to struggle to find effective treatments. During the 1890s, when the New York City Health Department established a diagnostic laboratory and produced antitoxin serum, it gained an international reputation for its innovative and practical applications of the new science of bacteriology.

Yet historians have often depicted American physicians as skeptical of the germ theory of disease and critical of the new laboratory science. Mainstream American physicians, so the argument goes, valued the early 19th century French tradition of clinical observation as well as an environmental theory of disease etiology. Only an elite few, who traveled to France or Germany for training, valued laboratory research. Most saw laboratory science as inapplicable at the bedside, and "specific" disease etiology as antithetical to sanitary science. If so many were resistant to the new bacteriology, how then do we explain the success of the anti-diphtheria programs of the New York City Board of Health?[1-13]

This article examines diphtheria treatments practiced by New York City physicians prior to the introduction of laboratory diagnosis and antitoxin treatment. A review of the medical literature of the time shows that physicians in New York City during the late 19th century were not so strongly resistant to laboratory science as was once thought. Rather, they prudently accepted change when it was applicable to and efficient in clinical practice. In this period of transformation in American medicine, physicians were eclectic in selecting their therapeutic choices. They continued to use clinical and environmental observation as their guides, without rejecting the helpful aids provided by the laboratory.

HISTORY OF DIPHTHERIA: CLINICAL OBSERVATION AND SANITARY SCIENCE

Diphtheria is a specific, infectious, and contagious disease which mainly struck young children, killing them quickly as a result of a toxin-induced myocarditis, or else slowly by suffocation, due to the build-up of pseudo-membrane in the throat. According to the medical literature, epidemics with symptoms similar to those associated with diphtheria in modern times have existed for many centuries. In the sixth century BCE, an Indian physician described the symptoms of an incurable disease that closed the throat. In the first century BCE, Aesclepiades of Greece is said to have performed laryngotomy. Some of the plagues of the Middle Ages may have been epidemics of diphtheria, such as one described by Saint Denis in 580 AD, or another by Baronius in 1004 AD. In the American colonies, epidemics of so-called croupous inflammations have been noted since the late 17th century. Such epidemics were described by New York City physician Samuel Bard in the late 18th century as well.[14-16]

According to American medical literature of the late 19th century, the French brought knowledge of the disease diphtheria into the modern era. It was in Paris in 1821 that the French clinician Bretonneau gave diphtheria its name, deriving it from the Greek compound word meaning "inflammation of the membrane." The naming of the disease was telling: It signified an emphasis on the specificity of disease. Rather than simply describing symptoms, Bretonneau, like others of the Paris school, promoted the notion that disease was localized, and could be anatomically detected in specific organs or tissues. Bretonneau had studied three epidemics that had occurred in France during the years 1818, 1825, and 1826. Based on 60 autopsies, he analyzed the pseudo-membrane and other surrounding tissues of the mouth, nose, and throat. He carefully described these tissues so as to clarify differences between diphtheria lesions and those of other diseases. He distinguished between localized and generalized manifestations of the disease.[14,15]

In spite of the fact that French and German scientists had begun to demonstrate in the 1870s and 1880s that a number of diseases were of microbiological origin, American physicians did not necessarily agree with such a view. Instead, they generally believed in the "miasmatic" or "zymotic" theory of disease etiology.[1] As late as 1887, at the College of Physicians and Surgeons in New York City, William Park recalled being taught that sewer gas caused diphtheria. He received a lecture there, based on three illustrations: the first pictured a child sleeping on its bed, with a diphtheria miasma passing through the wall from a broken sewer pipe to the child's throat. The second illustration showed a physician examining the child, and the third depicted the child's funeral, with weeping parents.[4(p 1441)] In an 1885 textbook titled *Diphtheria and Its Management*, Joseph E. Winters blamed epidemics of

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FIGURE 1. Joseph Winters, MD (courtesy of The New York Academy of Medicine Library).

diphtheria on "sudden vicissitudes of temperature and rainfall, in connection with, or soon after, prevailing east or northeast winds."[16(p 15)]

During the 1870s and 1880s, a number of diphtheria epidemics occurred in New York City, with a case fatality rate ranging from 42% to 49%. These epidemics certainly turned the attention of the city's physicians to the disease. Reflecting their concern to find useful therapies for the disease, for example, members of the New York Academy of Medicine heard papers such as "Diphtheria and its treatment," and "Is tracheotomy justifiable in diphtheria?" in 1883.[2(pp 154-155)]

Similarly, upstate New York communities suffered epidemic outbreaks. Both William C. Wey[17] of Elmira and N. B. Bayley[18] of Brewster, New York, published articles in 1881 describing their experience with the epidemic of 1877. Wey traced the cause of the outbreak to "a small, sluggish stream of impure water," while Bayley analyzed the "sanitary conditions" of the houses and surroundings of 49 sick patients.

There were also a number of treatises on diphtheria published by New York physicians in the 1880s, some of which expressed some distrust of the bacteriologic work of French and German scientists. Among them were Abraham Jacobi's *A Treatise on Diphtheria* (1880), Joseph E. Winter's *Diphtheria and Its Management* (1885), and C. E. Billington's *Diphtheria: Its Nature and Treatment* (1889).[14-16] The latter two were published after the diphtheria bacillus was isolated by Klebs and Loeffler in 1884.[1,5,6,19,20] Billington[14] mentioned this discovery, but was skeptical that Loeffler's work had positively proven that the Klebs-Loeffler bacillus was the true cause of diphtheria. He did say, however, that "the possibility of their being so [was] not to be excluded."[14(p 38)] Winters,[16] on the other hand, was much more negative toward a bacteriologic approach. He anticipated an attempted takeover of his world view by germ theorists when he said, "the mode of treatment in vogue [meaning germ theory] has for its object (according to its exponents) to attack the germ or poison of the disease, as an entity, and destroy it, leaving out of view *every other consideration.* Physicians in search for such a specific are distracted from rational treatment. These experiments have doubtless killed more patients than germs."[15(pp 21-22)]

It is more apparent that all three authors believed in selecting compatible ideas from both miasmatic theory and French clinical observation in their approaches to diphtheria. For example, all the authors agreed that although the disease had been observed in many diverse climates, epidemics usually occurred in periods of cold and humidity. They also concurred that the disease was contagious, although the authors did not agree on a single mode of transmission. Billington, for example, said that "direct contact or the deposition of diphtheritic matter on the mucous membrane" contaminated persons.[14(pp 18-23)] Jacobi, on the other hand, cited "moist soil assist[ing] in the spread of the disease," as well as "a connection between diphtheria and filth."[15(pp 33-35)] In spite of these differences of opinion, both Billington and Jacobi recommended isolating patients with diphtheria, and removing well children from a home with active diphtheria present. They also both agreed that the sick room should be kept as sanitary as possible, and that bedding and clothing used by the ill be disinfected, but this should not be seen as a concession to germ theory. This was standard procedure, according to miasmatic theory of contagion—decaying matter on such objects spread the disease.

In these books of the 1880s, the authors' recommended therapies for the disease were strongly based on clinical observation. To them, appropriate treatment depended on a number of factors that had to be determined by the physician. Observation, not theory, was necessary in making diagnoses. The age of the patient, for example, was a primary guide in determining whether diphtheria was present, as well as if there were other cases appearing simultaneously, such as in an epidemic. These factors, along with an appearance of the pseudo-membrane and fever, usually convinced the physician to pronounce a diagnosis of diphtheria. Then a number of other decisions had to be made. Was the diphtheria classified as pharyngeal, nasal, laryngeal, ocular, or cutaneous? Was the intensity of the case mild, severe, or malignant? Was the degree of toxic absorption benign or septic?

Underscoring the value in and necessity of the clinician's observational powers, Billington declared that although each case could be guided by some general principles, no routine course of treatment was recommended. He devoted more than two thirds of his textbook to discussing various treatments and their possible applications, as did Jacobi and Winters. Billington went on to say that beyond a "careful adaptation of measures to each individual case," the physician needed a "patient persistency in a few well-selected ones." He went on to warn his readers that "impatience, indecision, and changes of method *without definite reason*" . . . would "invite defeat."[14(p 15)]

These therapies were meant mainly to treat the local symptoms and to support the strength of the patient. Jacobi, Billington, and Winters recommended the actions of gargling, spraying, irrigating, vaporizing, and swabbing a variety of substances on the throat, in order to destroy or at least keep in check the pseudo-membrane. When or if patients weakened, all three physicians recommended stimulants. Another important theme present in all three books was the recommendation for complete bed rest and a light diet.

Surgical Intervention: Tracheotomy

Billington devoted a small section of his book to tracheotomy, an operation to be resorted to "when the respiration [was] so seriously interfered with . . . that asphyxia [was] imminent." The operation entailed cutting into the trachea and inserting a tube, in order to open an air passage that had been closed due to the swelling of the pseudo-membrane. He cited a study showing a 28% recovery rate of a total of 21,853 cases, when tracheotomy was performed by physicians from Germany, Great Britain, France, the United States, and other countries. Billington concluded, after analyzing a number of factors, including the method and skill employed in the operation, aftercare, patient age, type of diphtheria, season of the year, and state of the disease, that he would agree with the French clinician Trousseau, a student of Bretonneau: the earlier the operation was performed, the greater were the chances for patient survival.[14(pp 121–138)]

In contrast, Jacobi's experience had taught him that the operation rarely saved lives. He stated that in 50 consecutive operations, performed during the years 1872 to 1874, none of his patients recovered. In a later, milder epidemic, he estimated that 27% of his tracheotomy patients survived.[15(pp 134–142)] Still, Jacobi later stated that he performed at least 700 operations between 1860 and 1887.[21]

Why did he perform these operations, even though he seemed to be conscious of the fact that he was only postponing the inevitable? Perhaps he and other physicians of his time were convinced that it was more humane to try to do something rather than stand by and watch children die of asphyxiation. Adolph Rupp, in a March 1885 article published in New York's *Medical Record*, declared that physicians must determine when to operate, not if they should, in writing that, ". . . no child should be allowed to die from the effects of a persistent progressive laryngeal stenosis [constriction], incidental to diphtheritic croup, if the physician can help it."[21(p 311)]

Billington felt compelled to perform the operation, he said, because even if it failed to save life, it would secure an easier death than one which would result from diphtheria if it spread down into the bronchi. Those cases, he said, were when "death [was] the slowest and most painful of suffocations, and only when septicaemia to the point of stupefaction [was] present . . . [did] the child escape a horrible amount of suffering."[14(p 244)]

In late 1884 and early 1885, Joseph E. Winters entered into a public debate over the operation after publishing his article, "Is tracheotomy in diphtheritic croup dangerous?"[22] In his paper, he encouraged other physicians to perform tracheotomy, even though he understood that some patients in the advanced stages of the disease would die anyway. He made the point that the operation was relatively safe, providing that it was performed with care. "Persistence of the original disease, delay in the performance of the operation, and neglect after, are the causes of failure," he said. He insisted that physicians act quickly, before the patient was so weak that no further intervention would help.

Winters based his essay on his "impressions from reading," and secondarily on "his own personal experience." This infuriated Adolph Rupp, an ear surgeon at the New York Eye and Ear Infirmary, who, believing that he had special skills because he was a surgeon, discouraged other physicians from operating without extreme caution. He responded to Winters' essay with two lengthy and attacking articles, also published in the *Medical Record*.[21,24] Rupp listed numerous complications that could further compromise a patient's already serious condition. He warned his readers that the operation was not so simple as Winters would have them believe.

In April of 1886, the editor of the *Medical Record* summarized contemporary medical opinion on tracheotomy, giving a glimpse of the decisions that practitioners made at the bedside. He said that physicians were "almost unanimous" in their opinion that tracheotomy be performed rather than "let the child die of strangulation," but he also added that because the operation was of "little practical utility," few parents allowed the operation to be performed.[25]

Surgical Intervention: Intubation

In 1858, Paris physician M. Bouchut tried to find a method other than tracheotomy to keep the diphtheritic air passage open. He made a small hollow cylinder, narrower at one end, and when he inserted it into the throats of his patients, he was able to relieve constriction and difficult breathing. Bouchut presented his findings to his colleagues after placing only seven tubes, but since all his patients died anyway, his innovation was not well received, and it quickly fell into obscurity.[14,26]

According to contemporary sources, this earlier work was unknown to Joseph O'Dwyer, who began to experiment with tubes on patients during a 25-year career as an attending physician at the New York Foundling Hospital. In 1881, he tried a 1-inch bivalve speculum which would later be called the "O'Dwyer tube."[1,26] Occasionally, he used the tube on live children, and in 1884, he reported the first recovery from laryngeal diphtheria in a child that he had intubated.[1,27]

This innovation, with definite potential for application to diphtheria cases, received considerable attention from New York City physicians. By 1886, after performing the operation on hundreds of patients, O'Dwyer won the support of Abraham Jacobi, then President of the New York Academy of Medicine. Jacobi opened a discussion of O'Dwyer's tube to the membership, helping to publicize its merits.[28] In 1887, O'Dwyer gave papers before the New York Academy of Medicine and the Medical Society of the State of New York (MSSNY), further broadcasting the use of his invention. In 1877 and 1888, O'Dwyer published papers in MSSNY's journal, then called *The Transactions of the Medical Society of the State of New York.*[29,30] By 1889, news of the tube was widely available. O'Dwyer published an addendum to Billington's *Diphtheria: Its Nature and Treatment,*[14] calling it *Intubation in Croup.*[31]

During the 1880s, a number of physicians in New York City used the O'Dwyer tubes, and news of these experiences spread to others on the local, national, and even international level. Dillon Brown, a resident physician at the New York Foundling Hospital, reported on 15 intubation cases in April 1886.[32] W. P. Northrup, a pathologist at the New York Foundling Hospital, read a report before the New York Academy of Medicine in late 1886 on 165 cases, later publishing it in the *Medical Record.*[26] A. R. Jenkins, of Henderson, Kentucky, responded to Dr Northrup in a letter to the editor in January 1887, questioning advice that Northrup offered in his article.[33] Francis Huber, Clinical Assistant Professor of Diseases of Children at the College of Physicians and Surgeons in New York, published an article on intubation in June 1887, with a detailed table of 47 cases.[34] During the same month, O'Dwyer published "Feeding after intubation of the larynx" in the *Medical Record.*[35] Two other articles in the same issue were contributed by New York physician A. Caille,[36] and a resident physician of Nursery and Child's Hospital of New York, Irwin H. Hance.[37]

In September 1887, Northrup and O'Dwyer attended the International Medical Congress in Washington, DC, both presenting papers on intubation.[38] F. K. Priest, of the city's Willard Parker Hospital, reported on 42 cases in February 1889. And finally, in June of 1890, Dillon Brown went to the second annual meeting of the American Pediatric Society to present a paper on the technique of intubation.[39]

FIGURE 2. Joseph O'Dwyer, MD (courtesy of The New York Academy of Medicine Library).

The O'Dwyer tube seems to have been judged by American physicians as a useful therapy for diphtheria. In 1886, the editor George Shrady remarked that the innovation was desirable because it did away with the use of cutting instruments, but he more succinctly echoed the interests of American physicians when he said that "every new therapeutical [sic] measure must be judged by its results." He went on to quote a number of reports from medical journals in New York, Boston, Chicago, and even the *International Encyclopedia of Surgery,* which all showed a favorable recovery rate from intubation when compared with that of tracheotomy patients.[25] Later studies, such as that by Dillon Brown in 1889, also favorably described recovery from intubation. Brown reported on 350 intubation procedures that he had performed on patients suffering with laryngeal diphtheria, the most serious form of the disease. Of the last 50 procedures, 26% of the patients recovered, which was considered remarkable.[1]

One of the problems associated with the use of the tube was to teach practitioners how to use it. O'Dwyer devoted several pages of his 1889 textbook to addressing this issue. He showed how the nurse or assistant should hold the child in place. Then he told his readers how to place the "Denhardt gag" behind the teeth—with care taken not to do it suddenly or with force. Once the gag was in place, O'Dwyer instructed the operator to stand in front of the patient, holding the apparatus that inserted the tube into the child's throat. He then went on to explain how to push the epiglottis aside and glide the tube down into the larynx before detaching it, or else the child's throat tissues would sustain injury.[31]

O'Dwyer seemed concerned that his written instructions would not suffice. Knowing that practice on human subjects was beyond the reach of most medical students, O'Dwyer suggested that students of intubation try it on the larynxes of small animals. He recommended placing animal heads "upright in the neck of a bottle or other convenient receptacle." Practice was necessary, he said, so that the practitioner would be prepared when he had to quickly insert the tube into the larynx of a struggling, choking child.[30]

Similarly, Northrup discussed practical problems encountered by the clinician while using the tube. He, too, gave instructions on inserting the tube, and he also answered typical questions that practitioners were asking: Was there any danger of the tube slipping down the trachea? Did the tube ulcerate the trachea? Should the tube be regularly removed and cleaned? How was the child to be fed while intubated?[26]

He also discussed practitioners' concerns about interacting with the parents of diphtheria patients. He assured practitioners that it would be their "routine experience. . . to witness the unbounded gratitude of the parents and friends," once they had inserted the tube. He told practitioners to instruct parents of the possibility that the tube may be coughed out, and if so, to place it in water. He assured his readers that difficult breathing would not recur for at least four hours, more than enough time for the physician to return and reinsert the tube. He firmly advised practitioners not to state a prognosis for at least 48 hours after insertion, however. One had to watch for further complications occurring in that period before a favorable prognosis could be made.

Northrup believed that performing intubation instead of tracheotomy was advantageous to building the physician's reputation. When performing tracheotomy, he said, parents had to be convinced to give permission, and then afterwards, the house smelled of ether, there were bowls of bloody towels, and then there was the unpleasant task of cleaning knives. Intubation, on the other hand, was met with little opposition on the part of parents. It required no cutting, and it relieved difficult breathing just as efficiently as tracheotomy. Intubation's most important advantage, he said, was that the physician would avoid hostile reactions from parents if the child died. With intubation, there were fewer regrets that the procedure was performed, and no discredit was attached to the physician.

LABORATORY SCIENCE AND THE PROBLEM OF DIAGNOSIS

Making a proper diagnosis of diphtheria was of great importance to practitioners in New York City in the late 19th century, and this concern was addressed by the introduction of laboratory testing in 1893.[2,5,7] A proper diagnosis was crucial, because if diphtheria were suspected, then local irrigation therapy would be indicated, which was both uncomfortable for the patient and difficult to perform. In an article published in 1889 by T. Mitchell Prudden,[40] the difficulty in diagnosing diphtheria was clearly laid out. The presence of certain clinical symptoms, such as pseudo-membrane in the throat, and fever, did not necessarily mean that the patient was suffering from diphtheria. Therefore, when Prudden posed the question, "Should the clinician consider and treat all pseudo-membraneous inflammations as if they were the local expression of the acute, infectious disease . . . called diphtheria?", he was really asking if every patient who presented with the typical symptoms should be subjected to irrigation treatments.[40]

While at the Carnegie Laboratory in 1889, Prudden made bacteriologic studies on patients with suspected diphtheria, in order to find out if the diphtheria bacillus was present. He found not only the presence of the so-called Loeffler's bacillus, the one that caused diphtheria, but also two types of streptococcus. According to his article, the work concurred with that of other European researchers who were conducting similar studies. Many diphtheria patients were suffering from "mixed infections"; both diphtheria and the so-called false or pseudo-diphtheria. Others presented with the classic symptoms of diphtheria, but when tested had only pseudo-diphtheria, a considerably less dangerous disease.

Based on these findings, Prudden argued for the universal use of irrigation therapy. He told his readers that whether one or more causative agents set up the infectious disease accompanied by pseudo-membrane, in all cases the seat and origin of the infection was local. He concluded with the statement that "all [the] experiments pointed to the paramount importance of efficient local germicidal treatment."[40]

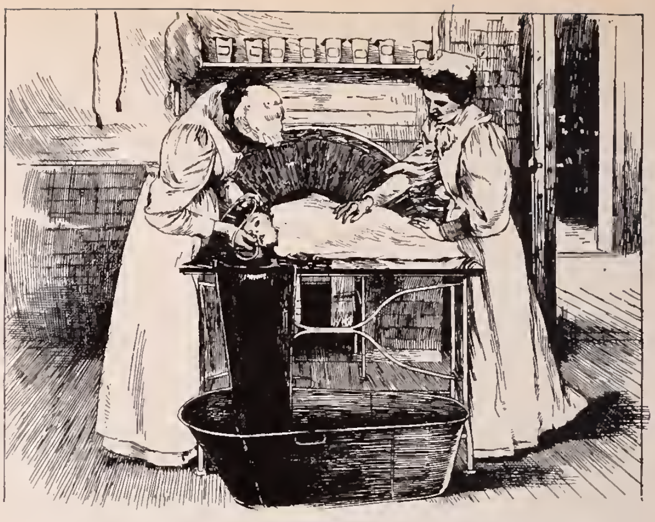
FIGURE 3. Irrigation of the naso-pharynx (reproduced from Berg[48]).

A few years later, Prudden's student, William Hallock Park, would carry this early work to wider use, for both private practitioners and for the city health department. Park, a young man and a nose and throat specialist, was appointed to the city's powerful Division of Pathology, Bacteriology, and Disinfection under Prudden and Biggs. Prior to his appointment, Park had been studying cultures taken from young patients at the Board of Health's Willard Parker Hospital. There he found that approximately one half of the cases were pseudo or false diphtheria.[1]

From May 1893 to May 1894, Park[41] conducted a study on bacteriologic investigations and diagnosis of diphtheria under the auspices of the bacteriological laboratory of the city's board of health.[41] Charles Wilson, a lawyer and then president of the Board of Health, stated six reasons for conducting such work. First, he argued that differentiating diphtheria from other diseases that affect the upper air passages was of great importance for sanitary reasons; in other words, better diagnosis meant better public health. Second, he stated that most practitioners were having difficulty in making diagnoses. They could not determine differences between simple, noncontagious forms of inflammation and diphtheritic or communicable types. Clinical history, anatomic lesions, or both, failed to provide consistent differences that would guide the clinician to a proper diagnosis. Third, he discussed the accuracy of laboratory testing in making such diagnoses. The value of such testing, he went on to say, was in demonstrating how cases of diphtheria, even of the mildest type, were transmitted from infected persons to others. His fourth reason was an appeal to economy: proper diagnosis could save the city money. In the city's hospital for infectious diseases, patients with pseudo-diphtheria as well as true diphtheria had been receiving treatment. Accurate diagnosis would eliminate this waste of resources. Furthermore, the city could save money in fumigations of private dwellings, a service the city was providing in all cases of suspected diphtheria. With proper diagnosis, only the homes with definite cases would be visited. The fifth reason related to the image that the board of health wanted to project: to conduct such testing would represent a medical advance. The sixth reason was for the sake of furthering scientific knowledge. Wilson believed that these examinations would reveal "methods to prevent the extension of the disease."[41]

Apparently, New York City physicians agreed that the study would be useful. Perhaps it was because 1893 marked the beginning of another epidemic, making physicians more open to help in the diagnosis of rising numbers of suspected diphtheria cases. From May until the end of 1893, 2,603 cultures were examined. Among them, 1,801 proved positive; 613 cases were diagnosed as pseudo-diphtheria, and 209 were classified as unknown.[2] By May of 1894, 5,611 cultures were examined.[41] This was accomplished by the New York City Department of Health taking cultures of all suspected cases of diphtheria among hospital cases, as well as making diagnostic kits available to private practitioners through 40 local pharmacies. Throat cultures were collected once daily by messenger and were brought back to the department's laboratory for examination. After a 24-hour incubation period, individual physicians were informed of the results.

One of the study's most important and provocative findings was the fact that seemingly healthy persons could be carriers of the diphtheria bacillus. Researchers realized this because testing showed that persons without symptoms sometimes had the bacillus in their throats. They also noticed that bacilli often remained in the throats of convalescing diphtheria patients long after the disappearance of the pseudo-membrane. Because of these findings, by January 1894, the board of health notified hospital physicians that patients could not be released until a negative bacteriologic examination was proven. The board of health also warned physicians that "members of a household in which a case of diphtheria exists should be regarded as sources of danger, unless cultures from their throats show the absence of diphtheria bacilli." The board of health, in other words, wanted physicians in their private practice to suggest to the family members of a diphtheria patient that they be tested as well. The report also suggested to physicians to recommend that all recovering diphtheria patients be kept isolated until cultures proved negative. In places where cultures could not be made, the board recommended isolation for at least three weeks after the pseudo-membrane had disappeared.[41]

That these recommendations were impractical and also difficult to enforce was clearly understood by Park. Indeed, he even confessed that given the "results of the investigations before us, we can appreciate the difficulty of exterminating diphtheria from a city like New York." On the one hand, he said, cases were scattered all over the city, some too mild to be recognized, and on the other hand, virulent cases were raging in crowded tenements, where isolation was virtually impossible.

In the report's final passage, Park seemed to set the stage for the board of health's next step in fighting diphtheria—the introduction of antitoxin serum treatment. Although he believed that "stricter isolation of patients and. . .systematic supervision of the schools and tenements. . . " would reduce the numbers of cases in the city, the elimination of the disease did not seem probable, he said, "unless we can acquire a new means to combat the disease." That new means, which was not without its problems and controversies, was introduced in New York City within months after Park's report was published.[42-47]

Physicians not only used diagnostic testing for diphtheria during the period of time of Park's study, it became part of normal therapeutic practice. For example, in an article by W. H. Berg, the laboratory diagnosis of diphtheria was discussed with the same ease as traditional therapies.[48] He suggested starting with a proper bacteriologic examination, in order to make a correct diagnosis. He then advised isolating infected patients. Perhaps by having the laboratory diagnosis in hand, physicians could more easily convince parents to remove well children or to isolate ill ones. The testing certainly helped physicians decide whether or not to treat local manifestations with irrigations, which Berg recommended highly. He also advised using calomel fumigations, setting up croup tents, intubating when necessary, and, since this article was published after antitoxin was made available, he also suggested the use of the serum treatment.[48]

CONCLUSIONS: MEDICAL INNOVATION AND DISSEMINATION

The 1880s and early 1890s were critical years in the development of new therapies for diphtheria. New York City was an important locus of much debate on the efficacy of these new innovations. Because intubation was invented by a New York City physician who was connected to a sophisticated medical network, the innovation was rapidly and widely tried and tested for its value as a therapy. Debate surrounding the introduction of intubation centered on whether or not it was helpful at the bedside. Because New York City physicians judged it to be safer and less uncomfortable for patients, news of the treatment quickly disseminated through the medical network in the city, and later throughout the state and country. Although intubation was highly acclaimed by physicians at that time, its contribution would pale when compared to the potential usefulness of knowledge generated by the city's laboratory. While intubation was used to save life in advanced cases of diphtheria, better diagnosis could help prevent the disease in the first place. Testing could be used to determine when or if patients ought to be isolated from others, thus helping to curb further transmission of the disease, and better diagnosis helped practitioners know which treatments to choose. Debates surrounding this innovation similarly centered on usefulness to clinical application.

Although American practitioners utilized knowledge generated by their colleagues in the laboratory, this is not to say that they believed that all of their questions would be answered there. Luther Emmett Holt, another New York City pediatrician of the same era, wrote in his textbook *The Diseases of Infancy and Childhood*,[49] which would become the bible of pediatric medical students for the entire first half of the 20th century, that his readers ought to consider two kinds of evidence in diagnosing diphtheria—both the clinical and the bacteriologic. He carefully delineated the risks in wholly depending on bacteriologic diagnosis to the exclusion of clinical diagnosis. Holt believed that the patient and his surroundings had to be thoroughly examined for symptoms and evidence of the disease. Chances for the disease were greatly increased, for example, if the patient were a young child, if his home was in a tenement or an institution, and if there were other cases in his family or neighborhood. If throat symptoms occurred after high fevers accompanying other diseases such as scarlet fever or measles, he warned of the high probability of diphtheria.[49]

Holt made these points because he knew that the bacteriologic approach had its limitations. Not every physician and patient in America had access to diagnostic laboratories. Not all patients with diphtheria bacilli in their throats were actually sick. Nor were all patients with suspected diphtheria found to have the bacilli present at the time of examination. In other words, insofar as laboratories could help physicians make diagnoses, they could never replace the practitioner's sharp observational powers in helping to unravel the complexities of individual cases. Holt, therefore, urged the honing of clinical skills of physicians.

Holt's thinking, a synthesis of the dual traditions of clinical and environmental observation, plus the new additional use of laboratory science, would dominate pediatrics for years to come. Although Holt seemed to be reluctant in adopting a "chemical approach" to pediatrics, by 1910, he set up a laboratory in his Babies Hospital, funded by the Rockefeller Institute which he helped to found. There he supervised biochemical investigations into infant feeding, and numerous other problems pertaining to diseases of infancy and childhood, yet he never abandoned his belief in the importance of clinical practice.[50,51] Physicians like Holt embodied the American tradition of valuing clinical medicine while at the same time integrating the ideas and practices of laboratory science.

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Hypersensitivity pneumonitis associated with the use of ultrasonic humidifiers

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Hypersensitivity pneumonitis (HP) has been associated with the inhalation of organic dust in farmer's lung, bagassosis, and pigeon breeder's disease.[1,2] HP resulting from the inhalation of antigenic materials carried into the lung by fine water droplets was first reported by Banaszak, Thiede, and Fink in 1970.[3] They described four of 27 workers who developed dyspnea, fever, and chills in an office exposed to a contaminated air conditioner. A thermophilic actinomycete was cultured from this air conditioner, and serum specimens from all patients were found to contain precipitating antibodies against this organism. In addition, inhalation challenge with the extract of the cultured thermophile reproduced the clinical features in one patient. Subsequent reports by these and other investigators indicated that aerosols containing antigenic materials can also be produced by a variety of contaminated heating and humidifying systems.[4-9] We have recently cared for two individuals who developed clinical signs and symptoms of hypersensitivity pneumonitis, apparently caused by the inhalation of a humidifier-generated aerosol.

CASE REPORTS

Case 1. An 80-year-old retired state trooper was well until fever, cough, fatigue, and dyspnea developed. A chest roentgenogram at this time revealed diffuse pulmonary infiltrates. A presumptive diagnosis of an atypical pneumonia with left ventricular failure was made. He was admitted to the hospital and treated with erythromycin and diuretics. His respiratory symptoms and fever subsided in the hospital, and he was discharged four days after admission. However, within 24 hours after his return home, his respiratory symptoms and fever returned.

Three weeks later, he was readmitted because of progressive dyspnea and fever. On physical examination, he had a respiratory rate of 26/min and a temperature of 38.8°C. There was a soft systolic murmur but no gallops on cardiac examination. Inspiratory crackles were heard over the lower part of both lungs. The hematocrit was 39%, and the white blood cell count was 26,200/mL with 67% segmented granulocytes, 23% band-form granulocytes, 5% monocytes, and 5% lymphocytes. Urinalysis and electrolytes were within normal limits. An arterial blood gas drawn while the patient was breathing room air showed a PaO_2 of 60 mm Hg, a $PaCO_2$ of 35 mm Hg, and a pH of 7.48. He was unable to perform spirometry because of cough and shortness of breath. A chest roentgenogram (Fig 1) revealed bilateral interstitial and alveolar infiltrates, unchanged from his previous admission.

The patient's symptoms did not improve with the administration of antibiotics, digoxin, and diuretics. A transbronchial biopsy was then performed. Microscopic examination of the lung tissue showed a prominent interstitial infiltrate composed of a mixture of lymphocytes, plasma cells, and histiocytes (Fig 2). Some areas contained multinucleated giant cells with granuloma formation and organizing intra-alveolar debris. The pathologic findings were consistent with HP. Cultures from the bronchial washings were negative for *Mycobacterium tuberculosis*, *Legionella pneumophilia*, and fungi. Serologic studies were negative for *Mycoplasma pneumoniae*, *Legionella pneumophilia*, and a variety of viruses.

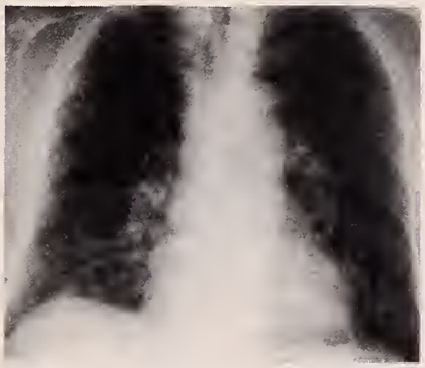
FIGURE 1. Chest roentgenogram showing diffuse alveo-interstitial infiltrates.

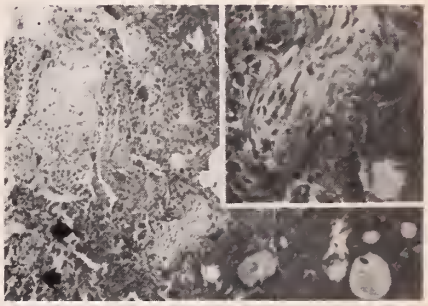
FIGURE 2. Interstitial round cell infiltrates (arrows); organizing intra-alveolar debris (inset) (hematoxylin-eosin stain; original magnification × 10, inset × 20).

On close questioning, the patient indicated that he had purchased two portable ultrasonic humidifiers prior to the onset of his respiratory symptoms and fever. He had used the humidifiers every day, except when he was in the hospital. One humidifier was kept next to the arm of his favorite reading chair, where he spent most of each day. The water reservoir of the humidifier was occasionally rinsed out with tap water, but no effort was made to clean the mechanical parts. Samplings obtained from the water and surface of the mechanical parts revealed a growth of *Cephalosporium sp*, *Trichosporon beigelii*, *Candida guilliermondii*, *Trichoderma*, and aerobic gram-positive bacilli. No thermophilic organisms could be identified. Serologic tests for precipitating antibodies to thermophilic actinomycetes were negative.

The antibiotic therapy was discontinued, and a 60-mg/day dose of prednisolone was administered. The patient's respiratory symptoms and fever abated gradually over the next few days. A repeated analysis of arterial blood, drawn while he was breathing room air, revealed $PaCO_2$, 31 mm Hg; PaO_2, 82 mm Hg; and pH 7.44. Chest roentgenograms showed a significant decrease of the

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pulmonary infiltrates.

The patient was discharged to his home, and the humidifiers were removed. His respiratory symptoms continued to improve, and the dose of prednisolone was tapered and discontinued over the next three weeks. Follow-up pulmonary function tests three months after discontinuing corticosteroids showed a mild restrictive ventilatory defect and a normal diffusion capacity. Follow-up chest roentgenograms showed a complete resolution of the pulmonary infiltrates. He has remained asymptomatic and in excellent health during the past two years.

Case 2. A 59-year-old retired worker was in good health until a nonproductive cough, exertional dyspnea, and mild fever developed. Two years prior to his illness, he had obtained a portable ultrasonic humidifier, which was initially placed in his wife's bedroom. He slept in a separate bedroom and was only occasionally exposed to the humidifier aerosol. During this time he had had no respiratory symptoms. In November 1987 he moved the humidifier to his bedroom. Three days later he noted the onset of dyspnea, cough, and mild fever. He went to a walk-in clinic where a physician diagnosed pneumonia and prescribed erythromycin. A chest roentgenogram at this time showed diffuse pulmonary infiltrates.

The patient's respiratory symptoms deteriorated during the next week despite treatment with erythromycin. He noted a significant decrease in his physical work capacity and could no longer do his regular housework. He continued to use this humidifier after the onset of respiratory symptoms and sometimes leaned over into the rising mist hoping that the humidity might relieve his dry cough. His symptoms worsened.

He was referred to a chest physician who found inspiratory crackles at the base of both lungs. The heart had no murmurs or gallops. There was no edema of the lower extremities nor clubbing of fingers. Urinalysis, complete blood cell counts, and electrolyte studies were normal. The chest roentgenogram showed increased interstitial and alveolar infiltrates in comparison to chest roentgenograms taken one week before. The pulmonary function test revealed a forced vital capacity (FVC) of 2.14 liters (51% of predicted), a total lung capacity (TLC) of 3.39 liters (68% of predicted), and a diffusion capacity of 12.2 mL/min/mm Hg (43% of predicted), indicating restrictive ventilatory and diffusion defects.

Because it was suspected that inhalation of the humidifier mist may have caused his respiratory disorder, the patient was advised to discontinue use of the humidifier. No medications were given. He felt a significant improvement of his respiratory symptoms by the following morning. Within a week, all his respiratory symptoms disappeared, and his physical activities have returned to normal. Chest roentgenograms taken two weeks later revealed a decrease in pulmonary infiltrates, and repeated pulmonary function testings showed an increase in FVC, TLC, and diffusion capacity.

DISCUSSION

Portable ultrasonic humidifiers have been widely sold for use in homes and offices. Water droplets generated from these humidifiers range in size from 0.5μ to 3.0μ,[10] small enough for some of these droplets to reach the distal air spaces. Although the reservoirs for these devices are generally transparent and readily cleaned, contamination of the ultrasonic generator itself or the tower and nozzle assembly which disperses the ultrasonic mist is not readily apparent, and these areas are more difficult to clean (Fig 3).

If the humidifier is not cleaned thoroughly and frequently, organisms may grow in the machine; the organisms may be aerosolized during humidifier operation, and these organisms may be inhaled by individuals who are near the humidifier. If the individual then becomes sensitized to this noninfectious, but potentially antigenic aerosol, that individual may develop a clinically significant hypersensitivity reaction localized in the lung.

The diagnosis of HP usually is made from a combination of characteristic history, an abnormal chest roentgenogram, abnormal pulmonary function tests, and lung pathology consistent with the diagnosis. In the acute form of HP, dyspnea, cough, and fever usually occur four to six hours after exposure to the offending antigen. The symptoms abate gradually with the avoidance of the antigenic agent. In the chronic form of HP, which is frequently due to prolonged exposure to small amounts of antigen, the clinical presentation is usually insidious, and patients may not experience acute respiratory distress after the challenge of antigens.[2]

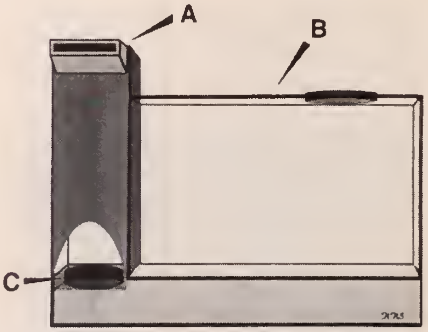

FIGURE 3. Ultrasonic humidifier: A—Aerosol Dispersion Nozzle—this assembly is often contaminated and is difficult to thoroughly clean; B—Water Reservoir—this portion of the humidifier is clear, relatively accessible, and easy to clean; C—Ultrasonic Generator—This portion of the humidifier is recessed in a well, relatively inaccessible, and difficult to clean.

Pulmonary function testing often demonstrates a restrictive ventilatory defect with a decrease in both lung volumes and in the diffusion capacity. Chest roentgenograms reveal diffuse interstitial and alveolar infiltrates.[1,2,11] The pathologic findings are characterized by patchy interstitial infiltrates of mononuclear cells, multinucleated giant cells with granuloma formation, and organizing intra-alveolar debris.[12] Histologic findings of the lung in chronic HP may be that of nonspecific fibrosis.[12] Lung biopsy may not be necessary if the clinical manifestations are classic and the relationship between symptoms and exposure is obvious.[1,12] Bronchoalveolar lavage (BAL) fluid from patients with HP is characterized by an increase in lymphocytes (compared with normal subject BAL fluid) with an increased proportion of T cells.[13] The diagnosis can be established by a positive antigen inhalation challenge that reproduces the presenting signs and symptoms. Corticosteroids may be used, particularly in cases characterized by severe systemic symptoms or serious impairment of pulmonary function.[13] Most acute cases will subside without specific treatment, provided that further exposure to the offending antigen is avoided.

The immunopathogenesis of HP has not been well defined. Immune complex hypersensitivity and cellular reactivity have been considered the major immune alterations induced by antigens in this disorder.[1,11] Interestingly, only a small proportion of individuals exposed to antigen aerosols that are associated with acute HP actually develop the disease.[2,11] Genetic variations in the immunologic response and an increased susceptibility, caused by cigarette smoking, for example, have been postulated to explain the variability in an individual's clinical responses to the same aerosolized antigens.[11,14]

As demonstrated by these cases and others,[2,4,5,9] the diagnosis of HP associated with the use of humidifiers is usually delayed, following repeated exacerbations of respiratory symptoms related to reexposure to aerosols or dust to which the symptomatic individual has become sensitized. Irreversible pulmonary damage may occur if the disorder is not recognized and exposure continues. Since the portions of the ultrasonic humidifiers that are most likely to become contaminated and that generate the aerosol are not readily visible (Fig 3), users may not clean them adequately. The diagnosis of HP caused by the inhalation of an antigenic aerosol from such a humidifier may not be considered, despite the otherwise typical clinical features of the illness, because the association between the use of small ultrasonic humidifiers and HP is not generally appreciated.

A variety of organisms such as *Thermoactinomyces vulgaris*, *Micropolyspora faeni*, and *Bacillus sereus* have been listed as agents that may contaminate the ventilation system and result in HP.[1,2] Thermophilic organisms have been reported most often,[1,2,4,6,15] but these organisms are not usually found in cool humidification systems, because their growth requires a warm environment.[5,15] As shown in case 1 and other reported cases,[2,5,9,15] it is common to grow more than one organism from a contaminated humidifier. Attempts to isolate specific antigens causing patients' disease are frequently not successful.[1,5] In a case reported by Hodges and associates[5] with HP related to a contaminated cool-mist vaporizer, aerosol challenge with preparations from individual fungi and bacteria cultured from the vaporizer failed to reproduce clinical features. However, the patient developed characteristic signs and symptoms after the challenge of water from the vaporizer. The authors concluded that an insoluble particulate antigen not related to the microorganisms isolated from the vaporizer might be the causal agent. Their case illustrates the difficulty in preparing the challenge materials and identifying the responsible agents in humidifier-related HP.

The presence of precipitating antibodies in patients' sera is useful in establishing the relationship between HP and suspected causative antigens.[1,2,11] Unfortunately, the value of this immunologic study in the diagnosis of HP is limited by its relatively low specificity.[16,17] Precipitating antibodies may be absent in some symptomatic patients with exposure history and may be present in asymptomatic but exposed individuals.[11] In a survey conducted by Banaszak and colleagues[16] investigating the relationship between exposure to thermophilic actinomycetes and hypersensitivity lung syndromes, they concluded that symptomatic subjects had a higher frequency of precipitating antibodies against various thermophilic antigens. Poor correlation existed among symptoms, radiographic findings, and the presence of precipitating antibodies. However, identification of specific precipitating antibodies might sometimes lead to investigation of a specific environment to uncover the causes of HP.

We have evaluated two individuals with a noninfectious pneumonitis. Both individuals developed the clinical features of hypersensitivity pneumonitis when regularly exposed to a home ultrasonic humidifier, and both individuals experienced a complete remission when exposure to the aerosol from these devices was discontinued. The first patient had a biopsy which showed features characteristic of HP. He improved, following removal from the ultrasonic humidifier mist, but identical respiratory symptoms developed following reexposure to the same humidifier at home, a de facto inhalation challenge test. Since the humidifier was removed, he has remained well at home for the past two years, strongly suggesting that the critical factor for respiratory disease was his exposure to the mist generated by this humidifier. We confirmed the presence of contaminating bacterial and fungal microorganisms from the well of the humidifier used by this patient. The isolated organisms are not considered pathogenic for humans. The diagnosis of HP in case 2 was based on characteristic findings of symptoms, signs, chest roentgenograms, and pulmonary function tests. That patient's symptoms rapidly subsided following the removal of the humidifier without any medical treatment. Although we did not identify the specific agents responsible for their disease, the relationship between each patients' respiratory symptoms to exposure to the mist generated from a specific ultrasonic humidifier strongly suggests that in each case the patient had developed HP related to the inhalation of antigenic materials in this mist.

We conclude that a syndrome typical of HP, including a characteristic clinical course and pathological findings, may occur in some individuals exposed to ultrasonic humidifiers. Early diagnosis can be made by taking a complete environmental history. Affected individuals improve rapidly when exposure to the aerosol mist generated from the humidifier is discontinued.

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Salmonella arthritis

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Arthritis is a recognized yet uncommon manifestation of salmonella infections. Joint involvement may present as a sterile reactive arthritis or as a septic arthritis. The primary site of infection is rarely identified, but suppurative salmonella arthritis is presumed to originate by hematogenous seeding or by extension from an adjacent focus of osteomyelitis.

CASE REPORT

We recently treated a patient with septic arthritis of a prosthetic hip joint caused by *Salmonella enteritidis.* The case is unusual in that salmonella rarely infects prosthetic joints; the patient possessed sickle cell trait rather than the recognized risk factor of sickle cell disease; and the patient did not respond clinically to appropriate antimicrobial therapy.

A 44-year-old black man with sickle cell trait and chronic renal failure on maintenance hemodialysis was hospitalized in January 1987, following 24 hours of acute onset left prosthetic hip pain, fever, and chills. The patient had begun hemodialysis in November 1976 for end-stage renal disease secondary to post-streptococcal glomerulonephritis. He received a cadaveric kidney transplant in June 1978. Glucocorticoid steroids were administered until 1983 when the transplant failed. Left hip arthroplasty was performed in 1979, and a contralateral arthroplasty was performed in 1980, both for steroid-induced aseptic necrosis of the femoral heads. Hemodialysis was reinstituted in June 1983, and the patient subsequently did well until 1985, when persistent left hip pain developed without fever or local signs. Plain x-ray films revealed loosening of the prosthesis. Serial arthrocenteses performed until December 1986 produced no bacterial growth from aspirated joint fluid. The patient denied preceding trauma to his hip and genitourinary or respiratory complaints. He took no antibiotics. There was no history of gastroenteritis in the patient or in his family members. He was seronegative for the human immunodeficiency virus (HIV).

On admission, the patient was febrile (temperature, 29.2°C). The skin overlying his left hip was tender and warm and the joint's range of motion was restricted by pain. There was no conjunctivitis or urethritis. Hemoglobin was 11.2 g/dL. There was no evidence of hemolysis. The total white blood cell was 4,700/mm^3; blood urea nitrogen, 63 mg/dL; and creatinine, 8.6 mg/dL. A chest film was unremarkable, and radiographs of the left hip only showed loosening of the femoral component of the prosthesis.

Arthrocentesis of the left hip produced purulent synovial fluid with 14,000 red blood cells. There were 52,000 leukocytes/mm^3, with 9% mononuclear cells and 91% neutrophils. Gram stain and stains for acid-fast bacteria and fungi revealed no organisms. Intravenous vancomycin and gentamicin were administered and ceftazidine was added the following day when preliminary cultures of the joint fluid grew a Gram-negative rod.

The patient suffered a cardiopulmonary arrest two days after admission and died on the third hospital day. Autopsy revealed grossly normal heart tissue and valves, a normal arteriovenous fistula, and a rejected cadaveric renal transplant. The left hip joint contained purulent fluid, but no osteomyelitis was evident. Cultures of pre- and postmortem left hip joint fluid, as well as postmortem lung and heart blood, grew *Salmonella enteritidis*. The organism was sensitive to ampicillin, cefazolin, chloramphenicol, trimethoprim-sulfamethoxazole, and aminoglycosides. Antemortem blood, urine, and stool cultures remained negative.

DISCUSSION

Salmonella infections present an increasing challenge to clinicians and epidemiologists. Risk factors for salmonella infection include sickle cell anemia and other hemoglobinopathies,[1] hematologic malignancies,[2] the acquired immunodeficiency syndrome (AIDS),[3] gastric surgery,[4] liver disease,[5] diabetes mellitus,[5] steroid or chemotherapy, and collagen vascular disease.[6]

Salmonella infections can be classified into five, often overlapping, clinical syndromes: gastroenteritis, enteric fever, bacteremia, focal infection, and the chronic carrier state. Bacteremic patients may develop focal salmonella abscesses months to years after their initial salmonella infection. Common focal syndromes include meningitis,[7] osteomyelitis,[8] pneumonitis,[9] endocarditis and arteritis,[10] and arthritis.[11]

Salmonella arthritis manifests in two distinct clinical forms: reactive arthritis and septic arthritis. Reactive arthritis is a sterile, polyarticular process which, similar to the arthropathy associated with shigella and yersinia gastroenteritis, is more common in HLA B27 positive individuals.[12] Pyogenic salmonella arthritis is rare, with an overall incidence of approximately 0.25% of all patients with salmonellosis.[13,14] In their 1976 review of 224 cases of septic arthritis, Goldenberg and Cohen[13] identified only two cases due to salmonella. Theoretically, infections of joint fluid can follow direct inoculation of bacilli into a joint space, spread from an adjacent focus of osteomyelitis or hematogenous spread from a distant site. David and Black[15] reviewed 84 cases of pyogenic salmonella arthritis. None of the cases had evidence of direct traumatic contamination of the joint space. Of the 18 x-rays obtained, only 8 demonstrated an underlying osteomyelitis. Thirty-eight percent of cases had positive blood and/or stool cultures. Physical findings included swelling, erythema, warmth, pain, tenderness, and limited range of motion. Sixty-seven percent of cases were monoarticular, with the knee being the joint most frequently affected and *Salmonella choleraesuis* being the serotype most frequently isolated.[15]

Ortiz-Neu et al[2] published a more recent review of salmonella cases. Nine cases of septic arthritis were identified. Risk factors and physical findings were similar to those found in David and Black's[15] study. Synovial fluid typically was cloudy, with a mean of 80,000 leukocytes/mm^3 and 90% neutrophils, a blood-to-joint fluid glucose ratio of greater than 40%, and a positive Gram stain in 67% of cases.[2] Cohen et al[10] from Duke University reviewed the literature from the antibiotic era and identified 44 cases of salmonella arthritis. Twenty-seven percent of cases oc-

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curred in children. Blood cultures were positive in 65% of patients and stool cultures were positive in 43%. Roentgenographic evidence of osteomyelitis was noted in 68% of patients.

Despite accumulating evidence that salmonella is a frequent cause of bacteremia and gastroenteritis in patients infected with HIV, no cases of salmonella arthritis have been reported in these patients.[16–18] Because of a paucity of cases, no controlled studies on salmonella arthritis therapy have been conducted. Hence, inferences must be drawn from experience with salmonella infections at other body sites and from arthritis caused by other Gram-negative bacilli.

Although most salmonella isolates are sensitive in vitro to cephalosporins and aminoglycosides, only ampicillin, chloramphenicol, and trimethoprim-sulfamethoxazole have proven efficacious in vivo in cases of salmonella enteric fever, bacteremia, meningitis, and osteomyelitis.[5,19]

Chloramphenicol penetrates extravascular spaces well and is a mainstay of therapy of focal salmonella infections. However, it has potentially serious side effects, is rarely bactericidal at achievable serum levels, and does not cure the carrier state. Trimethoprim-sulfamethoxazole enters closed spaces well, but it also rarely achieves bactericidal serum levels. Only ampicillin has been shown to achieve bactericidal blood levels and to cure the chronic carrier state.[19] Certain third-generation cephalosporins and quinolones have excellent in vitro bactericidal activity against salmonella species and have been used successfully in treating certain cases of enteric fever and localized infections caused by salmonella.[19,20–26] However, in light of studies demonstrating lack of correlation between in vitro and in vivo sensitivities of salmonella to other, older antimicrobials, one must be cautious in assuming clinical efficacy of these newer agents.

Despite the lack of prospective controlled studies of salmonella joint infections, it is clear that intravenous antibiotic therapy for two to four weeks as well as adequate drainage of purulent synovial fluid is mandatory for cure.[26,27] Controversy exists as to whether needle aspiration or open surgical debridement and drainage should be the initial surgical approach. According to a recent study by Goldenberg et al[26] comparing needle aspiration and surgery in the treatment of septic arthritis, open drainage was preferable in hip infections because of technical difficulty in performing serial needle arthrocenteses of that joint. Other indications for surgical drainage include evidence for an underlying osteomyelitis, the presence of an infected prosthetic joint, and inability to clear synovial fluid of leukocytes or organisms with serial needle aspiration. When Gram-negative bacilli infect a prosthetic joint, removal of the implant is almost always desirable.[28]

The case presented in this report is unusual in several respects. It is only the fifth reported salmonella infection of a prosthetic joint in the United States.[2,10]

Secondly, the patient possessed no clear risk factors for developing a salmonella joint infection. Steroid therapy had been discontinued three years prior to the onset of his arthritis, and the patient's hemoglobin electrophoresis revealed sickle cell trait. Although sickle cell disease is a significant risk factor for both salmonella osteomyelitis and arthritis, sickle cell trait has only been identified as a predisposing condition for salmonella osteomyelitis.

Although *Salmonella choleraesuis* is the most common serotype to cause a pyogenic arthritis, our patient's culture grew *Salmonella enteritides*. Lastly, our patient did not respond to three days of gentamicin therapy and five doses of ceftazidine, despite favorable in vitro sensitivities of his isolate to these antibiotics.

To date, no prospective therapeutic studies of salmonella arthritis have been conducted, and systemic administration of ampicillin, chloramphenicol, or TMP-SMX with adequate mechanical drainage remains the cornerstone of therapy. Clinical trials with the newer cephalosporins and quinolones are indicated. Meanwhile, salmonella should be considered an unusual yet potential pathogen in Gram-negative infections of both native and prosthetic joints.

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Manual labor metacarpophalangeal arthropathy in a baker

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An occupational arthropathy involving the metacarpophalangeal (MCP) joints has been described in manual laborers who perform sustained gripping with both hands.[1] This disorder has been termed the Missouri metacarpal syndrome. The patients described were noted to be machinists, truck drivers, and masons presenting with bilateral hand disorders in the setting of more generalized osteoarthritis in other joints. We describe an additional case in a professional baker of what we believe to be MCP arthropathy due to manual labor that exemplifies typical features of this disorder.

CASE REPORT

A 64-year-old man presented with bilateral painful swelling and stiffness of the MCP joints of his hands that had been present for many years. He had a history of a traumatic amputation of the fifth digit of the right hand in childhood, as well as degenerative arthritis of the hips and knees. A total hip replacement in 1982 revealed osteoarthritis in the surgical specimen. The patient had worked consistently for 35 years as a professional baker, which involved kneading dough and gripping utensils. His hand complaints persisted until retirement, at which time his symptoms improved.

On physical examination, the patient exhibited a visible enlargement of the second and third MCP joints bilaterally. Soft tissue swelling was not present, and there was no associated redness or warmth. The remaining articular examination was remarkable for decreased internal rotation of both left and right hips and bony enlargement of both knees without warmth or erythema.

Laboratory studies were all normal or negative, including serum iron, total iron-binding capacity and ferritin, cholesterol, T_4, serum calcium, triglycerides, rheumatoid factor, and anti-nuclear antibody. The erythrocyte sedimentation rate (ESR) was 16 mm/h.

Radiographs of the hands showed degenerative changes, with narrowing of the second, third, and fourth joint spaces bilaterally. Amputation of the right fifth finger was noted. Prominent osteophytes of both radial and ulnar surfaces (Fig 1) of the involved MCP joints were seen bilaterally. Cysts surrounded by sclerotic margins were noted in the second metacarpal heads. Radiographs of the right hip and knees identified mild degenerative changes, without chondrocalcinosis.

DISCUSSION

This patient presented a picture of severe osteoarthritic changes involving the MCP joints. The differential diagnoses included osteoarthritis in association with hemochromatosis, calcium pyrophosphate dihydrate crystal deposition disease, or metacarpal arthropathy seen with manual labor. Idiopathic hemochromatosis is an inherited disorder of iron metabolism. The diagnosis is supported by finding elevated serum iron and serum ferritin levels. The MCP joints (especially the second and third), the proximal interphalangeal and distal interphalangeal joints, as well as the wrists are involved in the arthropathy of hemochromatosis. Larger joints (shoulders, hips, and knees) may also be affected.[2] The patient had a joint replacement of the right hip, which lacked histologic evidence of iron deposition.

Half of the patients with calcium pyrophosphate dihydrate deposition disease (CPPD) have progressive joint degeneration which involves the knees, wrists, MCP joints, hips, shoulders, elbows, and ankles. In approximately 10% of these patients, the disease progresses to a chronic symmetric polyarticular form of arthritis resembling rheumatoid arthritis.[3] On joint roentgenograms, calcific deposits occur in characteristic sites. The patient described here lacked radiographic evidence of chondrocalcinosis in the radiocarpal joints, symphysis pubis, or knees. He also had no CPPD-associated metabolic or endocrine diseases. The possibility of crystal deposition disease, however, cannot be absolutely excluded without the benefit of a synovial biopsy, but it is less likely in view of the above findings. The patient was negative for rheumatoid factor and did not have the symmetric inflammatory joint process characteristic of rheumatoid arthritis.

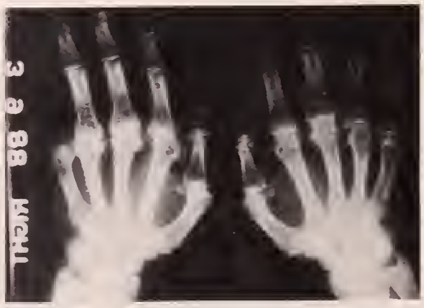

FIGURE 1. Roentgenogram of the hands showing degenerative changes of the second, third, and fourth MCP joint spaces bilaterally.

Studies have suggested that long term occupational stresses or sports activities may precede the development of degenerative joint disease.[4,5] As suggested by Peyron,[6] mechanical stress is one factor among others, such as aging, malalignment, or joint deformity, that interact to produce osteoarthritis of specific joints.[6]

Normal forces across a joint may produce changes if a cartilage abnormality or the cartilage composition makes the joint more susceptible to injury.[7] Biochemical and metabolic changes in the articular cartilage of patients with osteoarthritis have been described.[8] A cartilage abnormality may have played a role in predisposing this patient to generalized osteoarthritis and also facilitated the development of a traumatic arthritis of the MCP joints.

The patient lacked coincident involvement of the proximal interphalangeal and distal interphalangeal joints commonly seen in osteoarthritis of the hands. His hand involvement was bilateral, which points toward usage as playing a role, since his manual labor tasks required use of both hands. The dominant hand had more severe involve-

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ment as seen in the task-related impairments of cotton mill workers.[9]

Mechanical forces across the joint may induce pathologic changes if the forces are abnormal in quantity, or if normal forces are directed in abnormal directions.[7] The occupational arthropathy presented here occurred in the setting of "abnormal" stresses to the joints involved over time. The arthropathy is of interest for its anatomic distribution in a setting of occupationally related manual use over a period of 35 years. The differential diagnosis of degenerative changes involving the MCP joints should include manual labor arthropathy in addition to hemochromatosis and calcium pyrophosphate deposition disease.

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FROM THE LIBRARY

THE PRESIDENT'S PAGE

The medical profession, called by Robert Louis Stevenson "the flower of our civilization," is now on trial. Grave decisions will soon be made, which will determine whether our methods of service, our ideals, our independence will endure.

We are not opposed to change. In fact, we will work continually for change if it is for the benefit of our patients, and if, in the end, that change leaves our individual patient an individual, not a number. The physician is only part of the social picture, and it is certain that the profession will not completely control its own destiny in the economic sphere. Its spiritual destiny, however, will be its own to preserve gloriously, or to lose miserably.

Opposing us are large groups of people of various sorts. Sociologists, labor organizations, government bureaucrats, and even minority groups in the profession itself are doing their utmost to shackle the physician in a system of government-controlled distribution of medical care.

The issue, as expressed by all of these groups, is for the most part an economic one, and is based on the false idea that money can cure the evils that beset mankind. If we add our war debt, the material price of our liberty to date, we are now spending and giving away our great-grandchildren's money, with dogged perseverance, to prove this thesis, but I leave it to you whether the evils seem to be diminishing or multiplying.

The physician, on the other hand, points out that the most important thing is a high standard of medical service to the people. So both sides are not talking about the same thing at any time.

The medical profession is keenly aware of the high cost of medical care. No one is more so. Medical fees are but a fraction of it. It is indeed a very complicated problem. The medical profession will accept whatever is for the good of its patients, provided the control of and responsibility for their care is left with the doctors—where it belongs.

The medical profession will fight this threat to its patients best welfare with all its strength, and with honor and integrity. Our hope is that the people themselves, as this controversy is waged, will distinguish between sound and sound judgement.

The true physician, actuated by his ideals, will ever carry on in fair weather or foul. He would rather carry on as at present, with the ever-increasing potentialities for good for his patient. If, however, he must bow to the whirlwind, his wounds will be on his front and not on his rear, and he will still carry on. If a debacle should come, he will again assume control and carry on. It will be his duty to do so. He knows no other stimulus. It is in his blood.

The people should not be anxious about their doctors. They should be anxious for themselves. If medicine should be socialized, that will be here, as it has been elsewhere, the entering wedge to the collective state—the great leveler. If this nemesis should catch up with our generation, it will sit down with our children and our children's children.

LEO T. SIMPSON, MD
(*NY State J Med* 1949; 49:379)

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Intestinal perforation/fistulization complicating temporal arteritis

TO THE EDITOR: The report by Patsner and associates[1] of three cases of intestinal perforation/fistulization complicating temporal arteritis raises several questions and elicits some comments.

The report once again underscores the frequent nonspecificity of signs and symptoms in the aged, their tendency to be disproportionate to the severity of pathology, and their common absence of localizing value. We cannot be reminded of these features too often, and not only in steroid-treated elders.

One may question whether temporal arteritis was directly responsible for any of these perforations, and the authors have not asserted that it was. The lesions occurred in patients who had no other active manifestations of giant cell arteritis at the time, but this fact and the absence of previous extracephalic manifestations probably cannot be given any weight in deciding the role played by arteritis in the perforations. More significantly, the authors describe no anatomic or histopathologic evidence of vasculitis or vascular occlusion in the arteries supplying the perforated segments, nor of transmural necrosis with the possible exception of the third case. The specimen resected surgically in case two was surely extensive enough to be expected to reveal any responsible lesions. In case three a recurrence of peritonitis seven months after the initial episode is described, but there are no comments on whether the second laparotomy revealed an anatomic cause for the recurrence, nor if any surgical tissue specimens were taken and analyzed at the second operation. Also, the reader infers that autopsy was refused in case three; the anatomic findings would have been of great interest. Although corticosteroid therapy might have obscured or lysed active vascular inflammation in these cases, there would have to be some residual vaso-occlusive lesion, eg, scar, intimal proliferation, or thrombosis, to act as intermediary between arteritis and organ damage. Two of the patients reported had other conditions, common in the elderly, that can be associated with perforation: duodenal ulcer, adhesions from prior intraperitoneal surgery, and diverticular disease. While there exists heated controversy about whether corticosteroid therapy can cause or exacerbate peptic disease, and about its role in gastroenteric perforations, certainly one cannot exclude its having played a significant role in any or all of the cases reported by Patsner and associates; the absence of repeated perforations in two patients (or all three?) while on follow-up tapering corticosteroid therapy does not militate against a major corticosteroid contribution.

The spectrum of extracephalic manifestations of giant cell arteritis is widening all the time,[2] and intestinal involvement appears to be among them. Whatever the answers to the questions raised in this communication, for the reasons cited by Patsner and associates it will behoove the clinician to keep the complication of intestinal perforation in mind in patients with giant-cell arteritis who have abdominal pain: The management will be equally urgent whether the arteritis itself, the necessary therapy for the arteritis, or some other factor underlies it.

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Cervical tuberculosis

TO THE EDITOR: Recent papers on tuberculosis published in the *Journal*[1-4] emphasize the importance of making the diagnosis of this disease through entertaining a high degree of suspicion. We recently encountered a 35-year-old black East African man who had migrated to the United States within the previous five years. A painful mass was noted in the left side of the upper neck suggestive of lymphadenopathy. The mass showed some improvement with oral antibiotic therapy. Subsequent examination showed localized fluctuation with diffuse lymphadenitis in the upper cervical area. A recent chest film did not show any tubercular lesions. At the time of drainage, pus was found and a biopsy of the wall was taken. An initial Gram's stain and cultures showed no bacteria or any growth. No acid-fast bacilli were seen on stains. A biopsy showed granuloma without caseation. Cultures subsequently identified *Mycobacterium tuberculosis*, and the patient was started on triple anti-tuberculous therapy.

It would have been difficult to treat this patient based on a lack of acid-fast bacilli on smear and routine sterile cultures. The chest radiograph was negative, as could be expected in a third of all patients with extrapulmonary tuberculosis.[5] The diagnosis could easily have been missed without a high degree of suspicion.

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The use of a Swan-Ganz catheter and streptokinase in the management of massive hemoptysis

TO THE EDITOR: In the January 1990 issue of the *Journal*, Dr Brandstetter reports how he ingeniously controlled massive hemorrhage from the left mainstem bronchus by tamponading endobronchially with a Swan-Ganz balloon catheter,[1] rather than an embolectomy or thrombectomy balloon catheter, as described by Gottlieb and his colleagues.[2,3] Although, as the author points out, the Swan-Ganz catheter is more readily available in critical care units, embolectomy catheters offer advantages the Swan-Ganz does not. Embolectomy catheters are made with varying sized balloons, so a good match between the balloon and the orifice to be occluded can be achieved without stress on the balloon. Embolectomy catheters can be placed either through the channel of the fiberoptiscope or alongside the scope. (The distal end of a catheter can be grasped by a foreign-body retriever or forceps that has been passed through the channel of a fiberscope and then dragged by the scope into the appropriate spot in the tracheobronchial tree.) Embolectomy catheters can be left in place with the balloon inflated, by clamping the catheter near the hub with a hemostat, cutting off the hub, then plugging the catheter lumen with a pin. Instead of a straight pin as recommended by Gottlieb and Hillberg,[2] I find the tapered, pointed portion cut from a safety pin a more functional plug. Once this is done, if the catheter has been threaded through a fiberscope, the scope can be removed. Likewise, if the patient is intubated, the endotracheal tube can be removed. It is not easy to plug the balloon lumen of a Swan-Ganz catheter.

In the case described by Brandstetter, the endotracheal tube ultimately became occluded by blood clot. A conservative therapeutic option would have been to extubate, and then reintubate the right mainstem bronchus of the patient, perhaps using a bronchoscope or an endobronchial tube changer.[4] If the catheter occluding the left mainstem had been plugged with a pin, perhaps this could have been done.

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In reply. The response to my paper by Dr Nakao is appreciated, especially when one considers the mortality from massive hemoptysis and the limited success of a variety of methods to control bleeding. I am familiar with embolectomy catheters and agree that these can be passed through a fiberoptic scope or alongside it. As with other types of catheters, however, their availability in acute massive hemoptysis may be limited. In addition, familiarity with the correct use of these catheters may also be limited. Indeed, as Dr Nakao points out, he had to modify the original technique of plugging the catheter lumen of these embolectomy catheters as described by Gottlieb and Hillberg.[1] I do disagree with Dr Nakao's comment that plugging the balloon lumen of the Swan-Ganz catheter is difficult. The 2.5 cc syringe which inflates the balloon, or a clamp, will guarantee balloon inflation.

Finally, Dr Nakao appears to have misread the article, or he would not have made the suggestion of removing the endotracheal (ET) tube in order to resolve the blood clot in it. The Swan-Ganz catheter was *in* the ET tube, with the clot adhered to it. Obviously removing the ET tube would have resulted in pulling the Swan-Ganz catheter out, and massive hemoptysis would have recurred.

As Dr Rubin stated in his editorial in the same issue of the *Journal*,[2] cautious but creative ideas in gravely ill patients should be presented and, if possible, published. I thank the *Journal* for publishing this paper, and thank Dr Nakao for contributing his own experience to the matter of life threatening hemoptysis.

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The PRO contract

TO THE EDITOR: Much could be written on the subject of whether or not the Empire State Medical, Scientific and Educational Foundation should have been denied the professional review organization (PRO) contract for the three years beginning December 1, 1989.

Having been actively involved in the peer review process from its inception and having participated in the organization and subsequent administration of the Empire State PRO, I can attest to the fact that one could not find a more dedicated group of physicians and staff than those involved at the foundation. With the ever changing demands and additions to the peer review process imposed by the Health Care Financing Administration (HCFA), the foundation did make some unavoidable mistakes. However, on balance we were a dedicated organization which did a very creditable job to assure the Medicare population the best medical care available to them under the law. Although we disagree with its choice, we realize that HCFA had the authority to award the contract to whomever it wished. What is disturbing is the manner in which it went about it. It was unconscionable for HCFA to have kept the foundation waiting until 3:15 PM on November 30th, the last day of the contract, to be told to cease all work by 4:30 PM that same afternoon. Most assuredly the HCFA contracting office must have reached its decision long before the last day of the expiring contract. Aside from the fact that it was patently unfair to have kept the board of directors in great suspense, it belies the sense of common decency for a loyal staff, which worked far beyond the call of duty, to learn that in an hour and 15 minutes they would be out of a job. The fact that it was just before Christmas compounds the injustice.

Along with many other unanswered questions, this final maneuvering cannot help but focus attention on HCFA's strategy in denying the contract to the foundation.

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The influence of malpractice fears on the practice of medicine

TO THE EDITOR: In the ongoing controversy regarding medical malpractice, there is an oft-stated misconception that needs to be addressed, ie, the belief that the ever present possibility of a malpractice suit will make physicians practice better medicine. The supposition is that physicians, driven by fear of a lawsuit, will take better care of their patients.

Those involved in the practice of medicine know that this is not the case.

All physicians know that medical decisions are often judgment calls and one must have confidence in one's judgment to be able to practice medicine. Though many bits of data may factor into the decision making process, the ultimate choice of what is to be done and how it is to be done is determined by the experience, intuition, knowledge, and sensitivity—in other words, the judgment—of the physician.

The possibility of a malpractice suit, if nothing else, greatly increases the anxiety associated with decision making. Medical decisions are already loaded with much anxiety due to their very nature. The more anxious a physician is about a decision, the more difficult it is for the physician to rely on his or her judgment. It is when one is afraid to trust oneself that one is most likely to go against one's better judgment and, therefore, be most likely to make serious errors.

The threat of malpractice suits does not make for better physicians, only for more anxious, defensive, and insecure ones.

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Fetal heart rate changes during general anesthesia

TO THE EDITOR: Surgical emergencies in the pregnant patient raise major concern in clinical obstetrics. The potential fetal effects of drugs administered to the mother to induce general anesthesia have been the subject of numerous reports.[1,2] We recently monitored the fetal heart rate during general anesthesia for an emergency laryngostomy.

Case Report. The patient, a 36-year-old woman in her 34th week of gestation, was admitted to our institution with an airway obstruction due to laryngeal granulomas and subglottic stenosis. She presented to the emergency room with a history of intubation after a drug overdose six weeks prior to her admission, and was taken to the operating room for excision of laryngeal granulomas under general anesthesia. Fetal heart rate monitoring showed a normal pattern prior to induction of anesthesia and was continued throughout the procedure. After adequate preparation and the intravenous administration of 2 mg of versed, 100 mg of fentanyl citrate, 250 mg of surital, and slow infusion of succinyl choline as a paralyzing agent, decreased beat-to-beat variability was noted as early as three minutes later. Inhaled anesthetics included 0.5% enflurance (ethrane), 50% oxygen, and 50% nitrous oxide.

Twenty minutes later, there was a complete absence of the fetal heart rate beat-to-beat variability and a downward shift of the baseline from 150 beats per minute to 130. Maternal blood pressure also decreased progressively from 152/94 to 110/60.

After the surgical procedure the patient was awakened, and the fetal heart rate pattern and baseline returned to normal. The pregnancy continued to term and a healthy infant was delivered vaginally six weeks later.

Discussion. We have observed the fetal heart rate patterns during general anesthesia administered to a gravid woman. Although central nervous system depressants are readily cleared by the mother during pregnancy, their pharmacologic effects may continue well into the early postnatal period should delivery occur immediately.

In this report, the patient received general anesthesia during a nonobstetric procedure. The observed fetal heart rate patterns are compatible with central nervous system depression secondary to maternal hypoxia as described by Katz.[6] Neonatal central nervous depression predisposes to hypoxia and its long term sequelae. Because no uterine contractions occurred during the observation period, these fetal heart rate changes may indicate that large doses of anesthetics directly interfere with functioning of the fetal central nervous system. In addition, these changes could be secondary to decreased placental perfusion associated with relative maternal hypotension, although it may be argued that the observed decrease in blood pressure was not sufficient to alter placental function. If general anesthesia is administered to the pregnant woman, these fetal cardiovascular changes must be anticipated[1,4,5] and corrected in the event delivery cannot be delayed. It has been concluded that this loss of beat-to-beat variability associated with general anesthesia is probably normal for the anesthetized fetus.[6] However, more studies are needed to establish the clinical relevance of these fetal heart rate changes to the early neonatal period.

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Benzodiazepines: Additional effects of the triplicate program

This article is the second in a two-part series describing the effects of the addition of benzodiazepines to the New York State Triplicate Prescription Program. The first article described the decrease in benzodiazepine prescriptions; this article describes the effect of the law on substitute drug prescriptions, pharmacy practices, drug abuse emergency incidents and diversion.

Benzodiazepines were added to the New York State Department of Health (NYSDOH) Triplicate Prescription Program effective January 1, 1989.[1] This family of drugs was added to reduce the diversion of these drugs into illicit use and to reduce inappropriate prescribing. Results from the first six months show that prescriptions for benzodiazepines have been reduced with concomitant reduction in drug overdoses. Prescribing has also been stable, or has increased, for drugs that have not been abused. An increase in substitute drugs, offsetting the decrease in benzodiazepine prescriptions, has not been observed.

Methodology

As described in an earlier issue of *Epidemiology Notes*,[1] the primary objectives for inclusion of the benzodiazepines into the Triplicate Prescription Program were to reduce the diversion of these drugs into illicit use and to reduce inappropriate prescribing that can compromise patient well being. As part of the effort to measure the success of the objectives, prescribing practices for drugs that were most likely to be substituted for a benzodiazepine agent were analyzed both pre- and post-regulation (January 1, 1989). The analysis included examination of shifts in drug utilization, from a benzodiazepine agent to drugs that were deemed appropriate substitutes and to other less effective and/or more dangerous drugs.

Data sources used in the drug substitution analysis include:

- Medicaid, a federal, state and locally supported program that provides health care to the poor;
- Empire Plan, which provides insurance for over 423,000 state and local government employees, retirees and dependents;
- Elderly Pharmaceutical Insurance Coverage (EPIC) Program, a state cost-sharing program that provides prescription drug insurance to more than 70,000 low- and moderate-income individuals aged 65 or older.

In the Medicaid and Empire Plan analyses, quarterly claims averages were calculated for the four 1988 quarters and for the two 1989 quarters, and the averages were compared. For the EPIC program, data were averaged from three 1988 quarters and compared with each of the first two 1989 quarters.

Diversion, abuse and misuse were analyzed using three additional data sources. These sources are a survey of pharmacies conducted by the Department of Health Bureau of Controlled Substances, data on street prices of selected benzodiazepines in New York City collected by the Street Intelligence Unit of the New York State Division of Substance Abuse Services (DSAS) and an analysis of data from the Drug Abuse Warning Network (DAWN) of the National Institute of Drug Abuse.

The DOH Bureau of Controlled Substances conducted a survey of benzodiazepine dispensing in 80 New York State pharmacies during one week in December 1988 and one week each in January and April 1989. Three of the NYC pharmacies had closed by the April week. The survey consisted of on-site review of pharmacy records conducted by trained investigators.

Twenty-one pharmacies in the Bronx and Manhattan were selected because of their high volume of Medicaid claims or because of intelligence information indicating possible involvement in diversion.

The remaining pharmacies, located in Brooklyn, Queens, Nassau and Suffolk counties and in central and western New York, were selected to be more representative of a cross-section of pharmacies. Although the pharmacies surveyed did not constitute a statistically valid sample, the intent was to establish a barometer of early indicators to assess the general impact of the regulation at the pharmacy level.

Changes in street prices of illicit drugs are used as markers for changes in the availability of the drugs. The Street Intelligence Unit of DSAS collects information regarding illicit availability and sales of drugs in New York City. This information includes the price as well as the methods of sale of illicit drugs.

Approximately 60 hospitals in the New York City Standard Metropolitan Statistical Area participate in the DAWN program by reporting emergency room mentions of drugs. A drug mention is defined as a substance which was mentioned in a drug abuse episode. Up to four substances may be reported for one emergency room drug abuse episode. The US Drug Enforcement Administration (DEA) has analyzed the data and has provided estimates of the number of such emergency room mentions in New York City and the nation for the first four months of 1988 and of 1989.

Findings

Data from Medicaid, the Empire Plan and EPIC show a marginal increase in claims for benzodiazepine substi-

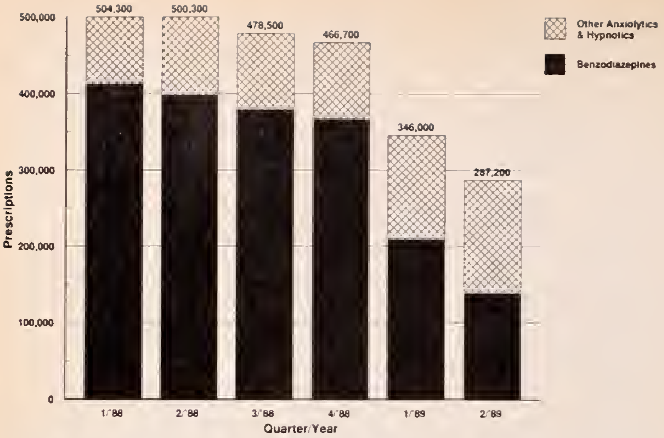

FIGURE 1. Prescriptions for benzodiazepines, other anxiolytics, and hypnotics paid by New York State Medicaid (data derived from MR-53 Quarterly Reports, Department of Social Services).

tute drugs not on the triplicate program. However, this rise has not offset the decline in benzodiazepine prescribing.

Figure 1 illustrates that in the Medicaid program, the decrease in the average number of prescriptions per quarter written for benzodiazepines in the first two 1989 quarters from the average number per quarter in 1988 was 215,497, while the increase in the number of prescriptions for other anxiolytic and hypnotic drugs (barbiturates, chloral hydrate, hydroxyzine, meprobamate, buspirone) for the same time period was 44,580. In the Empire Plan (Figure 2), the average number of prescriptions written per quarter for the anxiolytics hydroxyzine, meprobamate and buspirone increased by 2,700 between 1988 and 1989, while the average number of prescriptions written for benzodiazepines fell by 12,207.

In the EPIC program, the percentage of participants with other anxiolytic claims per quarter remained constant at 2.6% during 1988. That percentage rose to 4.8% in the first quarter of 1989 and to 4.9% in the second quarter (Figure 3). This is compared to a decrease in the participants with benzodiazepine claims from 21% in 1988 to 13.8% by the second quarter of 1989. As a proportion of all claims, other anxiolytics increased to 1% of claims in the first quarter of 1989 and to 1.1% in the second quarter, after remaining a stable 0.6% of claims in each quarter of 1988. At the same time, the proportion of benzodiazepine claims was decreasing from 5.4% of all claims in the second quarter of 1988 to 3.2% in the second quarter of 1989.

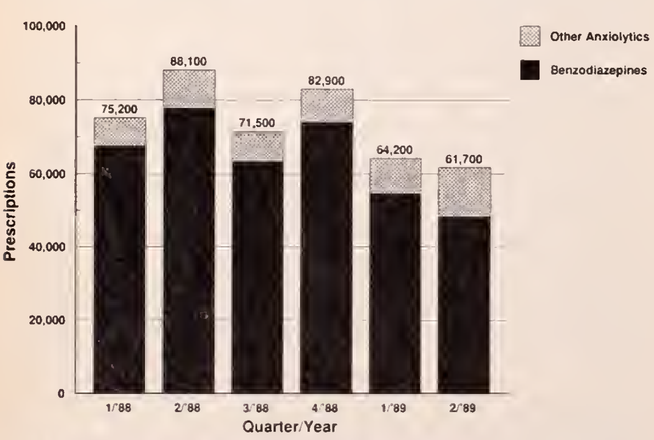

FIGURE 2. Prescriptions for benzodiazepines and other anxiolytics under the Empire Plan.

Pharmacy Survey

The pharmacy survey conducted by staff of the Bureau of Controlled Substances found a statewide reduction of 61% in the number of benzodiazepine prescriptions filled. The number of prescriptions filled in these pharmacies in the December 1988 week was 3,389 compared with 1,315 in the April 1989 week.

In the 21 Manhattan and Bronx pharmacies with high volume Medicaid claims or suspected of drug diversion, prescriptions filled dropped 76% between December and April. In the 59 other pharmacies, the decrease between the December week and the April week was 48%.

Street Intelligence

DSAS Street Intelligence Unit information shows a marked increase in the street value of selected benzodiazepines known to be diverted for illicit use, as well as a change in the marketing techniques of pushers.

Between December 1988 and September 1989, the price per pill for 1 mg of Alprazolam (Xanax) increased from $1.50 to $8.00, while the price for 10 mg of Diazepam (Valium) rose from a range of $2.00–2.50 to $4.50–6.00. Prior to implementation of the regulation, benzodiazepines were widely available on the street. Street sales observed by DSAS now take place one pill at a time.

Drug Abuse Warning Network (DAWN)

The number of times benzodiazepines were mentioned to DAWN as a drug abuse episode leading to an emergen-

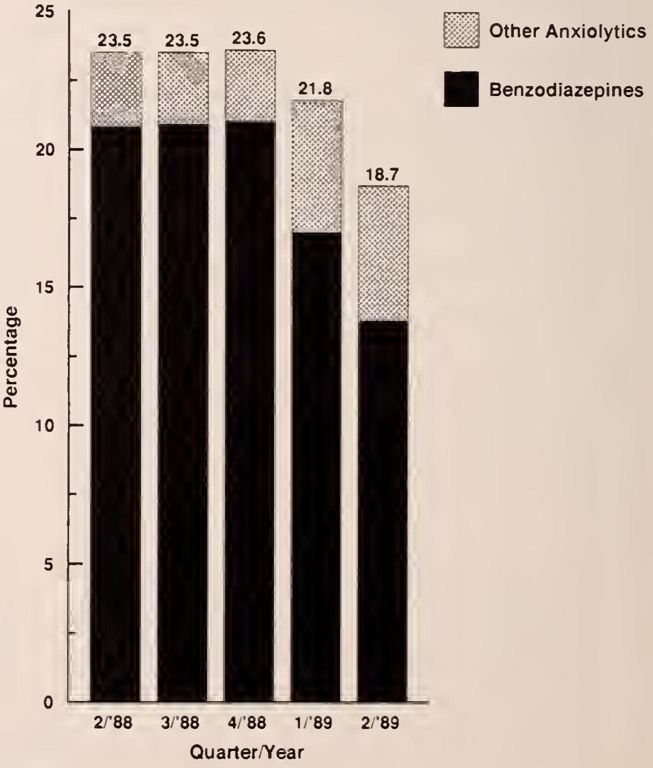

FIGURE 3. EPIC Program participants with anxiolytic claims.

cy room visit in New York City declined 31% between January and April 1989 compared with the same time period in 1988. Led by Diazepam, a total of nine benzodiazepines were among the 25 most commonly mentioned controlled substances in emergency room visits in the first four months of 1988, with a total estimate of 688 mentions. In 1989, the total number of mentions declined to 474 and three of the nine benzodiazepines from 1988 were not among the top 25. Simultaneously, the estimated number of benzodiazepine mentions nationwide in DAWN rose by 11% from 17,219 in the first four months of 1988, to 19,142 in 1989.

Discussion

Substitution of drugs not on the triplicate program has increased, but has not offset the decrease in benzodiazepine prescribing. DAWN data indicate the regulation's impact on abuse and possible illicit use of benzodiazepines. The data show a 31% decline in the number of emergency room mentions for benzodiazepines during the first four months of 1989 compared to the same time period in 1988, while the nationwide mentions increased by 11%.

The street price of a drug is an effective measure of its availability for illicit use. At a time when prices for crack, heroin and other drugs have remained stable or have been decreasing due to increased availability, street prices of benzodiazepines have shown a marked increase. The price increase suggests there has been a decrease in supply of these drugs since implementation of the regulation. These price increases also indicate that illicit demand has not been met by substitution of other drugs on the street. If it had been, the demand would have switched to the other drugs with prices for benzodiazepines remaining at the lower level. The DSAS intelligence unit has found evidence of the lack of alternatives available.

The distinct difference between the magnitude of the decrease in benzodiazepine prescriptions and the magnitude of the increase in the substitute prescriptions, coupled with the decrease in the benzodiazepine prescriptions in the high volume pharmacies, the increase in the street price of benzodiazepines and the decrease in DAWN drug mentions, indicate that the goal of reducing diversion and abuse of benzodiazepines is being achieved. To the extent that physicians are prescribing less effective or more dangerous drugs rather than have their benzodiazepine prescribing monitored by the Department of Health, appropriate peer counseling or Department of Health review may be needed. For further information regarding the Triplicate Prescription Program, contact the Division of Public Health Protection, (518)474-3276.

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ANORECTAL, PRESACRAL AND SACRAL TUMORS: ANATOMY, PHYSIOLOGY, PATHOGENESIS AND MANAGEMENT

By S. Arthur Localio, MD, Kenneth Eng, MD, and Gene Francis Coppa, MD. 356 pp, illustrated. Philadelphia, Pa, WB Saunders Co, 1987. $78.95 (hardcover)

Anorectal, Presacral and Sacral Tumors is a 356-page nicely printed compendium that addresses the sometimes complex problem of management of tumors in this area. Dr Localio is the Johnson & Johnson Distinguished Professor of Surgery at the New York University School of Medicine. His collaborators, Drs Eng and Coppa, are also well-known authorities. Chapter 1 is a fascinating illustrated guide of the history of surgery for carcinoma of the rectum. In a series of 16 illustrations, the reader is guided from the early 1930s attempts to extirpate rectal cancer to the Turnbull-Cutait procedure of the 1960s. Chapter 2 provides detailed, informative, multicolored illustrations of the pertinent anatomy. The remaining 16 chapters cover diagnosis and treatment of tumors of the anus, anal canal, rectum, and colorectum. Preoperative preparation is discussed, followed by details of the treatment options. Management of malignant extension to adjacent organs is discussed. It concludes with consideration of metastatic disease, radiation therapy, chemotherapy, postoperative follow-up and recurrence, and rehabilitation of the stoma patient. This book is extremely well written. The editing assured remarkable consistency with minimal overlap between chapters. While other textbooks contain similar information, *Anorectal, Presacral and Sacral Tumors* provides a wealth of material in a reasonably sized volume. References cited are voluminous and mostly current. This text fulfills the authors' objective to "place in one volume our present understanding and experience with these diverse neoplasms." I recommend it to surgeons whose practice includes management of neoplasms in the anorectal, presacral, and sacral areas.

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TOWARD A NATIONAL POLICY ON DRUG AND AIDS TESTING

Edited by Mathea Falco and Warren I. Cikins. 81 pp. Washington, DC, Brookings Books, 1989. $10.95 (paperback)

Two fundamental public health problems, the epidemic of HIV infection and the epidemic of drug abuse, are inextricably linked. This linkage goes beyond the reality that drug use is a key pathway to the broadening of the AIDS epidemic. New technologies have given us the tools to detect both HIV as well as the presence of certain illicit substances. As a result there have been calls from both the private and public sector to expand testing as a means of controlling both these epidemics. Others have viewed such measures as the ultimate abrogation of our personal freedoms as well as limiting our ability to deal with these two problems. This text summarizes the discussion held at two meetings which were held in late 1987 and early 1988. These conferences were sponsored by the Brookings Institution to examine the critical issues raised by drug and AIDS testing. Participants in these discussions represented the private sector, experts in law and medicine, as well as federal, state, and local government.

This report is divided into four chapters. The first, by Mathea Falco, an attorney in New York, summarizes the legal and philosophical issues raised by the implementation of testing in various settings. This chapter concludes by setting forth three possible scenarios for applying increased testing. These include expanded testing, mandatory testing in some circumstances, or permitting voluntary HIV testing while eliminating drug testing. In the second chapter, June Osborn of the University of Michigan, School of Public Health, provides the medical background of testing. Eighty percent of this chapter revolves around HIV testing, which is unfortunate since many of the issues she raises (lack of therapeutic options for seropositive individuals and issues of antibody test reliability) have now been clarified. There is no discussion of reliability issues with regard to drug testing. In the third chapter, private concerns are laid out by a panel including representatives of the insurance industry, the corporate sector, and organized labor. While it was agreed that HIV testing has no specific role in the workplace, issues of drug testing are not clarified. The fourth chapter, edited by Norman Zinberg, addresses the issues raised by mandatory testing.

This rather slim volume clearly raises many of the issues raised by mandatory testing. Nonetheless, the attempt to tackle both HIV testing as well as drug testing in a single text ultimately fails. Although linked, testing for HIV serves a different purpose than testing for drug use. As the book itself points out, these two processes are fundamentally different in that HIV infected individuals do not know they are infected, while drug testing does not provide new information to the person tested, only to the testing agency.

In summary, this text may be useful in outlining some of the elementary issues. If the reader is truly interested in dealing with these issues on a more substantial basis, other resources will be necessary.

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OTOLARYNGOLOGY: PROBLEMS IN PRIMARY CARE

Edited by D. Thomas Upchurch, MD. 350 pp, illustrated. Oradell, NJ, Medical Economics Co, Inc, 1989. $42.95 (softcover)

This book presents otolaryngologic disorders in a succinct fashion, with emphasis on differential diagnosis, geared specifically toward primary care physicians, medical students, and other health care personnel who need "to know more about otolaryngology" and "a place to start to learn." Information regarding management within the scope of nonspecialty physicians is given for routine as well as difficult and emergency problems. The purpose of the book is to impart basic, useful understanding of the entire spectrum of diseases that may be encountered in otolaryngology.

Structured after a course for family physicians sponsored by the Department of Otolaryngology of the University of Tennessee, the book is edited by

Dr D. Thomas Upchurch, clinical assistant professor in the department. Coauthors include Dr Richard W. Babin, professor and chairman of the Department of Otolaryngology and a recognized authority in the field, and Dr Christopher J. Harrison, assistant professor of pediatrics from the Children's Hospital Medical Center in Cincinnati, Ohio, an active researcher and clinician.

This book is especially useful in delineating those circumstances in which the primary care practitioner may diagnose and definitively treat specific diseases, as opposed to conditions that require referral or consultation once a preliminary diagnosis is made or emergent treatment is initiated.

In the first section, entitled "Assessment," anatomy, physiology, and physical examination techniques are reviewed. Under "Problems," differential diagnostic possibilities are outlined based upon symptoms, signs, and tests such as audiograms. Next, otolaryngologic disorders are described in alphabetical sequence in the "Diseases" section. Finally, in "Emergencies," urgent conditions and their management are detailed with minimal cross-reference to the other sections so that it may serve as a quick-reference handbook.

While the writing style enhances readability and the content is in general commendably comprehensive and current, there are several instances of inaccuracy and inconsistency. The recommendation to perform caloric testing with ice water in the awake and alert patient cannot fail to elicit severe vertigo and vomiting in a sizable proportion of patients with functional vestibular systems. Surgical treatment for positional vertigo is rarely indicated and is associated with a significant incidence of hearing loss as a complication, yet the "Diseases" section implies that positional vertigo of traumatic origin "usually requires . . . destructive surgical procedure." The book advocates use of magnetic resonance imaging and ultrasonography in the evaluation of nasal and sinus disorders, both of which techniques are relatively unproven and controversial. Likewise, CT sialography is no longer a helpful tool with present generation CT scanners. Additionally, arrows for two of the branches erroneously label structures in a diagram outling the facial nerve. The aggressive surgical debridement mentioned in the discussion of malignant external otitis has not proved useful and is not indicated. Viral labyrinthitis has been omitted for unclear reasons from the differential diagnoses of conditions with vertigo and hearing loss. Otitis externa is not mentioned as a cause of periauricular adenitis. Established etiologies such as sarcoidosis and bulimia are not included in the differential diagnoses of parotid enlargement. Antithyroid antibodies are incorrectly attributed to acute rather than chronic thyroiditis. Fundic exam is not included in the diagnostic work-up of subarachnoid hemorrhage. In the "Diseases" section, otolaryngologic consultation is advised for Ludwig's angina only if airway compromise arises or initial treatment is unsuccessful, whereas the "Emergencies" section treats this potentially fulminant infection which may require tracheotomy in concert with surgical consultation. Another inconsistency arises in the management of blisters in frostbite, with one chapter advocating breaking the bullae, and another warning against it (blisters are usually viewed as sterile protective barriers to infection that should not be broken).

There are few books in the field directed toward primary care physicians which have been as ambitious in their scope and detail. The book comes close to fulfilling the objectives of the authors despite the aforementioned flaws.

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YANKEE SURGEON: THE LIFE AND TIMES OF USHER PARSONS 1788–1868

By Seebert J. Goldowsky, MD. 450 pp, illustrated. Boston, Mass, The Francis A. Countway Library of Medicine, 1988. $24.50 (hardcover)

This book depicts the life and times of a 19th century physician and surgeon named Usher Parsons. He and the author, Dr Goldowsky, had much in common. They both practiced in Rhode Island, served on the faculty of Brown University, and received their medical degrees from Harvard.

Dr Parsons spent ten of his early years as a naval surgeon and became famous for having participated in the Battle of Lake Erie during the War of 1812. He was 30 when granted a medical degree. It marked the beginning of 50 more years as a medical practitioner, prolific writer, articulate speaker, and very diligent delegate to the American Medical Association. He was reelected several times and rose to the position of vice-president. Dr Parsons was married to the sister of Oliver Wendell Holmes, Sr, who bore his only son, Charles, who died of unknown causes at the age of 23.

This well-referenced book is difficult to read and review. It is both a detailed biography of an interesting man and a historical account of the period in which he lived (1788–1868). Apparently the author was strongly motivated by his respect and love for his native state—Rhode Island—and his subject. He used the logs, diaries, letters, books, and articles produced by Dr Parsons to write this epic. I believe it is over-long.

This book's appeal will be limited to readers interested in early American history, especially natives of Rhode Island, and the relatively few of us who played an active role in the affairs of organized medicine.

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THEORIES OF CARCINOGENESIS

Edited by Olav Hilmar Iversen, MD. 327 pp, illustrated. New York, Hemisphere Publishing Corp, 1988. $59.95 (hardcover)

This book consists of the proceedings of a conference held in Norway on August 16–20, 1986. The purpose of the conference was to get together experts in the field of carcinogenesis critically to discuss current concepts that are used by scientists in studying the process of carcinogenesis. It was thought that this was necessary because of the sometimes confusing use of terms and phrases by different people working in this area. It is clear that Dr Iversen got together experts in the various fields of carcinogenesis research and managed to put together a clear and concise volume of the "current" state of affairs in carcinogenesis research. There is no doubt that this book will be useful for a number of disciplines, in particular for scientists working in carcinogenesis. It will also be useful for the medical profession, in particular for those who deal with the treatment of cancer patients, since the current research at the molecular levels of oncogenesis is starting to have an impact on chemotherapy. The problem, of course, in writing a book like this involves the difficulties in keeping the information up to date since progress in both basic and clinical research in oncology is tremendous. For example, since the time of this conference (1986), a number of important new leads and discoveries inevitably have occurred. Perhaps the most useful thing that Dr Iversen has done is to get people together to discuss concepts, definitions, and paradigms that are currently used in the field. As such, the book does provide a foundation for further exploration and discussion among those who are involved in research and those who would want to get into the field of carcinogenesis research.

The book is divided into six parts. The first part is entitled "Philosophy of Science: The Impact of Paradigms"; the second, "Oncogenes: Positive and Negative Growth Factors and DNA Repair"; the third, "The Two-stage Theory"; the fourth, "Aging, Endogenous Production of Electrophiles, Oxidative DNA Damage"; the fifth, "Phenotypic Cellular Changes: Evidence from Liver Carcinogenesis"; and the sixth, "Growth Control, Cell Proliferation, Hormonal Carcinogenesis, Clonality." These parts are well covered. In particular the two-stage model of carcinogenesis is discussed in depth, and provides a foundation for further discussion and possibly some new ideas to be explored in both clinical and basic research.

The organization and style of the book are good and there is no doubt that the book has achieved the objective set out by the editor in compiling a concise treatise on theories of carcinogenesis. This book greatly complements the more detailed in-depth reviews that currently can be found in the literature.

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JURY OF MY PEERS: A SURGEON'S ENCOUNTER WITH THE MALPRACTICE CRISIS

By Howard C. Snider, Jr, MD. 292 pp. Montgomery, Ala, Fountain Press, 1989. $19.95 (hardcover)

It is unusual to be a competent practitioner, an articulate speaker, and a fine writer. Dr Snider's book is solid evidence that he has accomplished all three. His use of words and phrases vividly conveys his innermost thoughts and feelings when he was accused, with a partner, of medical malpractice.

After receiving his medical degree from the University of Alabama, Dr Snider served an internship at Johns Hopkins and a surgical residency in Alabama. In 1973 he entered private practice in a partnership, and in 1981 became involved with one of the partners in a very difficult surgical procedure on a young woman whose pelvic organs were completely frozen together by a cancerous lesion that originated in the colon. The details of the case are not unique but the exquisite story telling by the author will fascinate you, as it did me. I could actually feel the pain he experienced, and his words describe vividly his frustration and emotional distress. Dr Snider's doubts, fears, and periods of depression and elation are real and will make the reader believe that "there, but for the Grace of God, go I."

Having spent many hours since 1970 thinking about our professional liability problem, writing editorials, and attending national meetings in a futile search for possible solutions, I thought I knew a great deal more than the average physician. But in over 40 years of practice and teaching, never having been sued or threatened with a suit, I never truly imagined the devastation it produces. The author makes one feel involved in the case from the initial charge through the trial in 1985.

I share Dr Snider's feelings about jury trials and decisions made by 12 of his "peers." His call for reform and lack of faith in the present tort system are right on target. To be effective, this book must be read by all practitioners, medical students, politicians, insurance executives, and lawyers. It may help stem the tidal wave that is engulfing all of us.

In the judge's charge to the jury he included two definitions that are worth repeating here: "Negligence is the failure to do what a reasonably prudent person would have done under the same or similar circumstances, or the doing of something which a reasonably prudent person would *not* have done under the same or similar circumstances," and "Proximate cause of an injury is that cause which, in the natural and probable sequence of events and without the intervention of any new or independent cause, produces injury, and without which such injury would not have occurred."

The findings of the jury will surprise you.

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THE FAMILY FITNESS HANDBOOK

By Bob Glover and Jack Shepherd, 415 pp. New York, Penguin Books, 1989. $9.95 (paperback)

This book's purpose is to serve as a comprehensive guide and reference source for people of all ages. It specifically addresses many important health issues as they pertain to contemporary families. The theme, the involvement of children from a very young age through adulthood with regular exercise and good nutrition, is introduced by the author using examples of personal experiences with his son. Throughout the book the author shares situations and events he and his family have participated in together.

Bob Glover has a great deal of experience with program development, implementation, and supervision. His primary focus is on running, exercise and wellness programs, and youth organizations. He has spent much of his life helping Americans of all ages get fit. He uses these experiences and some of the latest research to provide good, sound advice and recommendations for health educators, practitioners, and, most important, parents.

This book is strongly recommended to anyone who works with or has children of any age. It can benefit clinicians in their practice by explaining the importance of developing these habits at a very young age. The book can also assist in communicating the key roles regular cardiovascular exercise and good nutrition play in helping to prevent many major diseases.

The book's main objective is the involvement of children in regular exercise, both at home and in schools. The author gives a brief history of fitness and how our society has changed with the advance of technology. He also discusses the "unfitness revolution," building a case that today's youth are in much poorer physical condition than prior generations. He then defines fitness, giving examples of different types of exercise, sports, and activities. The benefits of both exercise and sport are elaborated on, the author pointing out that variety is very important for longevity.

Several chapters are devoted to parent, child, and family interactions. Much is said about role modeling of parents, interest and motivation of children, the child's environment both at home and school, and his or her peers. Various types of games and activities are discussed based on their aerobic content.

Another large section discusses the fundamentals of getting fit. It explains such physiological principles as: overload, recovery, consistency, specificity, adaptation, and others important in understanding a complete program. There are discussions on the various components that make up a comprehensive program: warm-up, flexibility, strength, cardiovascular endurance, and cool down. Examples of "fitness games" are also numerous and very well explained. The author is very helpful in recommending games and activities through a child's developmental years tailored to specific age categories, ie, pre-school, elementary school, teenager, etc.

The rest of the book discusses the importance of lifetime participation in regular exercise and the concept of total wellness.

The quality, organization, and style of the book is very good. The material is presented in a clear and concise manner with good explanations and examples. The one area that is somewhat lacking is that of technical features. Several studies are mentioned and quoted, but there is no bibliography, reference list, or footnotes citing original investigations. It is quite obvious the author has done the necessary research for the book's content. However, a bibliography would have been helpful for those interested in pursuing some of the topics in depth.

This book is very comprehensive, and discusses many topics not touched on in publications with similar titles and objectives. The goals and objectives set forth by the author have been achieved in an organized manner and in language that is easily understood.

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FOREVER YOUNG: 20 YEARS YOUNGER IN 20 WEEKS. DR BERGER'S STEP-BY-STEP REJUVENATING PROGRAM

By Stuart M. Berger, MD. 254 pp, illustrated. New York, William Morrow & Co, 1989. $17.95 (clothcover)

In the never-ending quest for everlasting youth, we now have *Forever Young* by Stuart Berger, MD, author of several books including *The Immune Power Diet* and *What Your Doctor Didn't Learn in Medical School. Forever Young* is a self-help book implying that by following the advice given, the prize will be increased longevity and prolonged youthfulness.

Dr Berger has extensive clinical experience in the use of nutrition to control weight, allergies, and other nutritionally related illnesses. He has contributed significantly to the public's awareness of the importance of nutrition in obtaining and maintaining good health.

Forever Young contains sections on keeping a youthful skin, taking care of one's heart through proper diet, the physiological costs of smoking, protection of the skeletal system through exercise, diet, and nutritional supplementation, information on keeping the immune system in top shape, and protecting brain functioning. In the appendix a modified 21-day "very low (1,600) calorie diet" is outlined. Each of the chapters on the individual organ systems has a brief, intriguing description of promising research and developments.

Forever Young is written in a friendly, chatty style. There are numerous self-tests for evaluation and summaries for easy reference. The only references not provided are those which refer to the stated facts and advice throughout the book, although we are told that a computer search of "aging" resulted in 45,000 references.

With *Forever Young*, Dr Berger once again has contributed to the further popularization and awareness of the value of active intervention in maintaining one's good health. The information given is easily understood and, if applied diligently, should positively affect the quality of life and health of the individual. Whether following Dr Berger's advice will actually prolong life and youthfulness cannot be judged for another 50 or more years. Dr Berger is optimistic. He invites every reader to a "gala celebration of long life, strong health, and extended youth," Sunday, January 1, 2045, 12:00 noon, place to be announced. See you there?

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Two new infection-fighting drugs come to market

Fungal infections are a cause for concern in any patient, but for patients with immunosuppression, fungal infections carry a high risk of morbidity and mortality. Fluconazole (Diflucan®) is the first drug on the market in the United States derived from a new class of broad spectrum triazoles to treat and prevent relapse of fungal infections.

Fluconazole is a highly selective inhibitor of ergosterol, a vital component of cell wall formation in fungi. Its enhanced efficacy combined with low toxicity provide advantages over existing antifungal agents. Another benefit is its dual mode of administration; it is available in both oral and intravenous formulations. Roerig, division of Pfizer Pharmaceuticals (New York, NY), the manufacturer of Diflucan, boasts a 90% bioavailability of the oral form. As concentrations of fluconazole in the tissues and fluids compare favorably with those in the serum, the drug can fight fungal disease in almost all parts of the body, including the central nervous system.

Fluconazole has been approved by the US Food and Drug Administration (FDA) for treatment of fungal infections such as oropharyngeal and esophageal candidiasis, systemic candidiasis, including urinary tract infection, pneumonia, and peritonitis, and for the treatment and suppression of relapse of cryptococcal meningitis. It will be especially beneficial for immunocompromised patients, such as those with cancer or AIDS, as well as organ transplant recipients.

Mefloquine hydrochloride (Lariam®), a new antimalarial agent produced by Roche Laboratories, division of Hoffmann-La Roche, Inc (Nutley, NJ), is effective against drug resistant *Plasmodium falciparum.* Although chloroquine continues to be an effective prophylactic agent against some species of malaria, during the past 25 years, there has been an increase in chloroquine-resistant strains of *Plasmodium falciparum.* How mefloquine protects against malaria is not yet completely understood, but the drug appears to prevent malarial parasites from invading red blood cells.

Chloroquine-resistant malaria has become especially prevalent in southeast Asia, sub-Sahara Africa, the interior of South America, and in New Guinea. The Centers for Disease Control (CDC) recommend the use of mefloquine by travelers to any region of the world known to have chloroquine-resistant malaria. For travelers to regions where chloroquine is effective, the CDC promotes the use of chloroquine, rather than mefloquine, so as to lessen the risk of development of mefloquine resistance.

Diagnosing Lyme disease in seronegative patients

The spirochete *Borrelia burgdorferi* causes Lyme disease, a multisystemic disorder with symptoms that mimic a variety of other diseases. The diagnosis of Lyme disease is often difficult; even when patients present with the erythema chronicum migrans (ECM) that is characteristic of the disease and which occurs in about 50% of affected individuals, these patients may be seronegative. Seronegative patients with classic symptoms of Lyme disease lead to a diagnostic dilemma.

Schutzer et al have proposed that the inability to detect antibody to *B burgdorferi* "despite clinical features of the disease may perhaps be due to sequestration of the antibody in complexed form . . . , in which immune complexes (IC) often contain the relevant antigen and antibody, even in the absence of the free component" (*Lancet* 1990; 335:312–315). The authors tested this theory by analyzing immune complexes from seronegative patients with Lyme disease.

There were 22 seronegative patients in the study population with symptoms of Lyme disease who exhibited no antibody to *B burgdorferi* based on a commercial enzyme linked immunosorbent assay (ELISA). All of these patients were from areas of New Jersey endemic for Lyme disease.

Seronegativity was first reconfirmed by ELISA. Then two different enzyme immunoassays were employed to detect immune complexes. The technique of polyethylene glycol-borate dissociation of the ICs enabled analysis of the components by ELISA and confirmatory Western blotting for *Borrelia* antibody and antigen.

The authors found near normal IC levels in both the test and control patient populations. All ten patients with seronegative Lyme disease who had erythema chronicum migrans had *B burgdorferi*-reactive antibody in their immune complexes. Four of the other 12 seronegative patients with possible Lyme disease had ICs containing anti-*B burgdorferi* antibody. Complexed anti-*B burgdorferi* antibody was also detectable in ICs from 20 of 21 (95%) seropositive patients with symptoms of Lyme disease. Complexed antibody was absent from ICs of healthy controls having no symptoms of Lyme disease, as well as control patients with chronic fatigue or other diseases.

Soluble CD4: A new antiviral treatment for AIDS

Human immunodeficiency virus 1 (HIV-1), the retrovirus that causes the acquired immunodeficiency syndrome (AIDS), has been isolated, tested, and evaluated in laboratories throughout the world. The specific stages required for HIV-1 replication are now well understood, and scientists are assessing each step in the virus' replicative process in their goal to develop inhibitory agents that would halt the progression of HIV infection.

The first antiretroviral drug licensed to combat HIV-1 was zidovudine (AZT), a type of nucleoside analogue. Other nucleoside analogues, such as dideoxycytidine and dideoxyinosine, are yielding promising antiviral activity in early phase testing in humans. These drugs are also highly toxic and can produce severe adverse effects at therapeutic dosages.

Recombinant soluble CD4 (rsCD4) is a new antiretroviral agent now undergoing preliminary testing in HIV-infected persons. Early results of toxicity and pharmacokinetics studies have been promising, and there has been evidence of the drug's antiviral activity at tolerable doses.

Recombinant soluble CD4 inhibits HIV-1 production at a different stage of the viral replicative cycle than do

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NEWS BRIEFS are compiled and written by Vicki Glaser.

nucleoside analogues. Combination therapy with multiple antiviral agents having different modes of action may prove to be the most effective and least toxic method for conquering the human immunodeficiency virus.

Based on the knowledge that the HIV-1 envelope glycoprotein (gp120) binds to the CD4 glycoprotein on the surface of mature T4 lymphocytes in order for the virus to enter the cell, researchers sought to block the virus at this step, before it could penetrate the cell (*Ann Intern Med* 1990; 112:241, 247–253, 254–261). This goal led to the development of recombinant soluble CD4, a genetically engineered fragment of the CD4 molecule that can bind gp120 of HIV-1, thus effectively competing with the cell surface receptor for circulating HIV-1.

Preliminary safety studies employed escalating dosages of rsCD4, from 0.9 mg to 30 mg, administered intramuscularly or intravenously, to test for toxicity in HIV-infected subjects. "No significant differences in hepatic, renal, or hematologic measurements were noted during rsCD4 therapy for patients at any dosage . . . .No patient in our study developed severe or life threatening toxicities related to rsCD4 therapy." Reactions included transient erythema at the injection site, a maculopapular eruption which disappeared with cessation of treatment, and in one patient, the development of antibodies that bound rsCD4. This antibody response did not cause any negative clinical effect. The authors concluded that "rsCD4 is well tolerated by patients with AIDS or AIDS-related complex when given in doses up to 30 mg/d for as long as 28 consecutive days." The drug's long term effects require further testing.

Measurements of the subjects' serum HIV-1 p24 antigen levels taken before and after rsCD4 therapy suggested antiviral activity. There was a significant ($p = 0.021$), 23% reduction in p24 antigen levels, when comparing pretreatment (1,341 pg/mL) and post-treatment values (789 pg/mL) for subjects receiving 30 mg/d of rsCD4. This study did not determine the maximum tolerated dosage of rsCD4. Therefore, additional tests are needed to assess whether higher dosages or a longer course of treatment would further enhance antiviral activity.

Recombinant soluble rsCD4 could be an important contributor to the fight against AIDS. It could prove important in combination antiviral regimens, or as a transporter of toxic substances that target the HIV virus for destruction. Its most effective role awaits further study.

Cigarette smoking has a reversible, adverse effect on olfactory function

In a study performed at the University of Pennsylvania's Smell and Taste Center (Philadelphia, Pa), Frye et al demonstrated "a clear adverse effect of cigarette smoking on olfactory function that is dose related and present in past smokers. This effect is reversible, at least to some degree, and the time course of this reversibility is dependent on the duration of cessation from smoking and the amount of prior smoking activity" (*JAMA* 1990; 263:1233–1236).

Using the University of Pennsylvania's Smell Identification Test, the authors assessed olfactory function in a group of current smokers, nonsmokers, and subjects who had previously smoked. Based on cumulative cigarette smoking dose for current and previous smokers, Frye and colleagues found an inverse relation between pack-years and olfactory function. Furthermore, there was a significant ($p = 0.04$) correspondence between the length of time since a smoker had quit smoking and the improvement in olfactory function. A previous smoker does not quickly regain his or her sense of smell.

Man's sense of smell serves many functions, from detecting hazards such as natural gas leaks, smoke, or spoiled food, to enjoying the smells of flowers, spices, and scents. According to this study, "persons who currently smoke are nearly twice as likely to have an olfactory deficit than persons who have never smoked."

Promising new arthritis drug awaits testing in humans

Researchers at the University of Colorado Health Sciences Center (Denver) and Synergen (Boulder, Colo), a small biotechnology company, have discovered a protein that prevents arthritis-associated inflammation in rabbits and rodents. The protein also appears to stop the destruction of cartilage in rheumatoid arthritis, in which a patient's immune system mistakenly attacks healthy cells in the joints.

The research team of Thompson et al reported the purification of this protein, an interleukin-1 antagonist, in the journal *Nature* (1990; 343:336–346). Interleukin-1 plays a major role in the body's response to injury and infection. The release of interleukin-1 from monocytes and its subsequent binding to receptors on white blood cells stimulates the body's infection-fighting machinery and, in the process, produces pain and inflammation. Thompson and colleague's protein is produced by the same cells that manufacture interleukin-1 (IL-1), and it acts as a highly specific IL-1 antagonist by blocking the IL-1 receptors without activating an IL-1-like response.

The IL-1 antagonist appears to prevent the inflammation and tissue destruction caused by arthritis, rather than merely reducing the symptoms of arthritis while allowing the disease to progress, as do current treatments such as painkillers and anti-inflammatory agents. Synergen and Hoffman-La Roche are collaborating to develop a synthetic form of the drug, which could be manufactured more easily, inexpensively, and in greater quantities. Human testing with the IL-1 antagonist is planned for later this year.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1988; 88:456–462). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

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1. Brantly ML, Paul LD, Miller BH, et al: Clinical features and history of the destructive lung disease associated with alpha-1-antitrypsin deficiency of adults with pulmonary symptoms. *Am Rev Respir Dis* 1988;138:327-336.
2. Wewers MD, Casolaro MA, Sellers SE, et al: Replacement therapy for alpha$_1$-antitrypsin deficiency associated with emphysema. *N Engl J Med* 1987;316:1055-1062.

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AIR FORCE

(*continued from p 7A*)

MISSOURI

June 1–2. **Third Annual Contact Lens Course.** *June 6–9.* **Facial Rejuvenation.** 17 Cat I Credits. Ritz-Carlton St Louis Hotel, St Louis. *June 7–9.* **Frontiers in Endosurgery: Flexible Endoscopy, Laser Surgery, and Endourological Techniques.** Washington University Medical Center, St Louis. Contact: Cathy Caruso, Office of Continuing Medical Education, Washington University School of Medicine, 660 S Euclid, Box 8063, St Louis, MO 63110. Tel: (314) 362-6893.

NEW MEXICO

June 28–30. **Highlights in Women's Health Care.** 16 Cat I Credits. Contact: Sharon Stermer, Office of Continuing Medical Education, Scott and White, 2401 South 31st St, Temple, TX 76508. Tel: (817) 774-4073.

OHIO

June 7–10. **AIDS: Living in Recovery Conference.** Omni Netherland Plaza, Cincinnati. Contact: James R. Swearingen, Communication Coordinator, AIDS: Living in Recovery Conference, PO Box 14949, Cincinnati, OH 45250. Tel: (513) 541-7022.

SOUTH CAROLINA

June 11–16. **Intensive Review in Family Medicine.** 38½ Cat I Credits. Westin Resort Hotel, Hilton Head. Contact: Gerri LaVia, Department of Family Medicine, Medical University of South Carolina, Charleston, SC 29425. Tel: (803) 792-2426.

TEXAS

June 13–17. **Issues in Care: Pain.** 16 Cat I Credits. South Padre Hilton Resort, South Padre Island. *June 21–22.* **Pediatric Advanced Life Support (PALS) Provider Course.** 13 Cat I Credits. Temple. Contact: Office of Continuing Medical Education, Scott and White, 2401 South 31st Street, Temple, TX 76508. Tel: (817) 774-4073.

AROUND THE WORLD

CANADA

June 23–24. **Fifth Conference on Health Problems of the Chinese in North America.** Four Seasons Toronto. Contact: Dr John H. C. Chiu, 50 Michael Dr, Willowdale, Ontario, Canada, M2H 2A5. Tel: (416) 635-2550.

June 25–28. **Molecular and Cellular Mechanisms of Alcohol and Anesthetics.** University of Calgary School of Medicine, Alberta. Contact: Conference Director, The New York Academy of Sciences, 2 E 63rd St, New York, NY 10021. Tel: (212) 838-0230.

FRANCE

June 14–18. **New Approaches to Pulmonary Ventilation.** L'Hôpital de la Pitié-Salpetrierè, Paris. Contact: Professional Seminars Inc, PO Box 012318, Miami, FL 33173. Tel: (305) 547-6511. □

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Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
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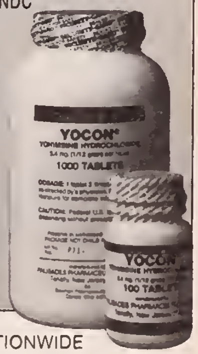

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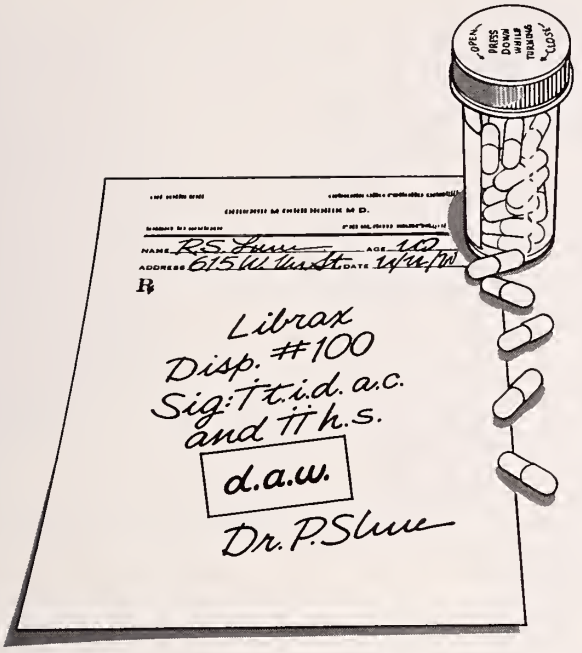

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Please consult complete prescribing information, a summary of which follows:

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows: "Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. After extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

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COMMENTARIES

Mainstreaming the minority and assumptions of rightful place 283
NAOMI R. BLUESTONE, MD, MPH

Women, families, and academic medicine 284
NANCY M. BENNETT, MD, KATHERINE G. NICKERSON, MD

Screening for breast cancer: Are physicians doing enough? 285
DONALD H. GEMSON, MD, MPH

Granulomatous mastitis 287
PATRICK L. FITZGIBBONS, MD

RESEARCH PAPERS

Breast cancer screening: Changing physician practices and specialty variation 288
DOROTHY S. LANE, MD, MPH; MARY ANN BURG, MSW, PHD

Senior women physicians: The question of retirement 292
ALLISON J. BATCHELOR, MD

REVIEW ARTICLES

Primary female syndromes: An update 295
ELLEN YANKAUSKAS, MD

Women in medicine since Flexner 302
MARY ROTH WALSH, PHD

CLINICAL CONFERENCE

Deep vein thrombosis in pregnancy 309

SPECIAL ARTICLES

Perspectives on women physicians in New York State: Past, present, and future 317
VERONICA C. SANTILLI, MD

An interview with Nancy E. Gary, MD, a medical school dean 321
LILLIAN GROSS, MD

History of the formation of the Committee on Women of the Westchester County Medical Society 324
TONI G. NOVICK, MD

Parenthood and practice 326
ADELAIDE T. SCANLON, MD

Jane V. Williams, MD (1924–1989): An appreciation 327
NAOMI R. BLUESTONE, MD, MPH

CASE REPORTS

Unexpected, unattended deliveries 330
RITA G. HARPER, MD; ELIZABETH SEATON, MD; REGINA SPINAZZOLA, MD; JERROLD S. SCHLESSEL, MD

Sarcoidosis of the breast coexisting with mammary carcinoma 331
AJAY K. SHAH, MD; LYDIA SOLOMON, MD; MILTON A. GUMBS, MD

LETTERS TO THE EDITOR

Responses to "Parenthood and practice" 334
VALERIE KAISER, MD
JESSICA R. JACOB, MD

An introduction to a current bibliography on women in medicine 335
RENEE A. FLEISCHER, MD, ELLA ABNEY

Substance abuse during pregnancy 336
JOHN T. PARENTE, MD; BENJAMIN GAINES, MD; ROBERT LOCKRIDGE, MD; STANLEY BAUER, MD

LEADS FROM EPIDEMIOLOGY NOTES 338

NEWS BRIEFS 341

BOOKS RECEIVED 343

MEDICAL MEETINGS AND LECTURES 3A

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References
1. *USP DI Update, September/October 1988, p 120.*
2. *Br J Clin Pharmacol 1985;20:710-713.*
3. *Data on file, Lilly Research Laboratories.*
4. *Scand J Gastroenterol 1987;22(suppl 136):61-70.*
5. *Am J Gastroenterol 1989;84:769-774.*

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Brief Summary. Consult the package literature for complete information.

Indications and Usage: 1. *Active duodenal ulcer*—for up to eight weeks of treatment. Most patients heal within four weeks.

2. *Maintenance therapy*—for healed duodenal ulcer patients at a reduced dosage of 150 mg h.s. The consequences of therapy with Axid for longer than one year are not known.

Contraindication: Known hypersensitivity to the drug. Use with caution in patients with hypersensitivity to other H_2-receptor antagonists.

Precautions: *General*—1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy

2. Dosage should be reduced in patients with moderate to severe renal insufficiency.

3. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

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Drug Interactions—No interactions have been observed with theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450 enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increased serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility—A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of varying durations included almost 5,000 patients. Among the more common adverse events in domestic placebo-controlled trials of over 1,900 nizatidine patients and over 1,300 on placebo, sweating (1% vs 0.2%), urticaria (0.5% vs <0.01%), and somnolence (2.4% vs 1.3%) were significantly more common with nizatidine. It was not possible to determine whether a variety of less common events was due to the drug.

Hepatic—Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to three times the upper limit of normal, however, did not significantly differ from that in placebo patients. Hepatitis and jaundice have been reported. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS—Rare cases of reversible mental confusion have been reported.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with equal frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic—Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental—Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity—As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Because cross-sensitivity among this class has been observed, H_2-receptor antagonists should not be administered to those with a history of hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance by approximately 84%.

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July 9. **Update on Interventional Cardi-**

(*continued on p 9A*)

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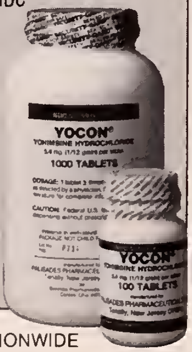

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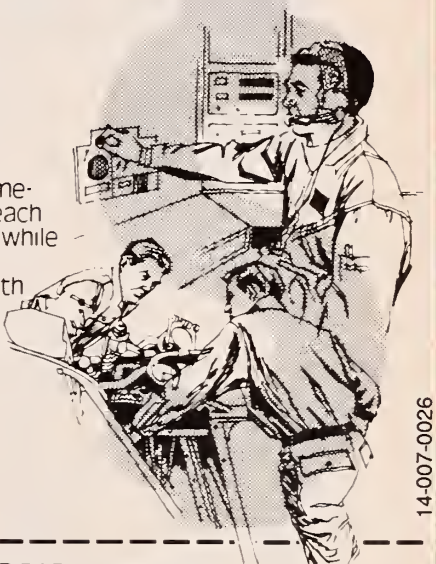

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NEW YORK STATE
JOURNAL OF MEDICINE
June 1990 • Volume 90, Number 6

COMMENTARIES

Mainstreaming the minority and assumptions of rightful place

Those of us educated before the explosion of women entering the medical profession during the past decade and a half remember well the professor who welcomed the incoming freshman students with the ancient warning, "Gentlemen, look to the right of you! Look to the left of you! One of you will not be here when the semester ends!" An updated version of that grim reminder of the toll of medicine should now be, "Students! Look to the right of you! Look to the left of you! One of you is now a woman, and the other soon will be!"

One third of the freshman class has been female for quite a while now, resulting in one fifth of New York State's practicing physicians being female. The projections are for a continued heavy upsurge of women in medicine. We therefore considered it more than timely for an issue devoted primarily to the issues of women physicians, and, inevitably, the women who are pleased to seek them out as personal physicians. The *Journal of the American Medical Women's Association* has been addressing their concerns for decades, but only recently has the "male establishment" joined their chorus. Our colleagues at the *Western Journal of Medicine* produced a fine issue on this subject in 1988.[1] And the numbers of women submitting articles for publication in the medical journals of all specialties have been increasing. We further note the concomitant rise in the numbers of women in other professions that intersect medicine, such as the law.

The growing number of female physicians has in many ways intensified the difficulties of women entering this profession. The problems they have always faced have not been solved by their numbers, and in fact, their very magnified presence has only served to accentuate the unresolved issues attending those who wish to enjoy the benefits not only of a career, but of traditional family life as well. When women were few in numbers, they could embrace the professional life in full service to society, and let the other go, as a necessary prerequisite to the superior performance demanded of them. The pre-21st century demand that women "have it all" has compounded their dilemmas even as it has expanded their horizons.

Most of the literature dealing with how women are adapting to their new role comes not from women physicians themselves, but from social scientists of all kinds, primarily those whose intellectual pursuits lie in the area known roughly as "women's studies." Psychologists, sociologists, anthropologists, psychometricians, economists, specialists in organizational theory, even political scientists now find the growing role of women in all the professions not only worthy of study, but vital to the interests of the nation. Much of the literature on women physicians is now unfortunately buried in larger works dealing with women professionals in general, for, as medicine now more closely approximates the work of teachers, lawyers, etc, the interests and concerns of female professionals all merge. Women are concerned with professional advancement, reorganization of the power elites, equitable reimbursement, recognition of the validity of their simultaneous responsibilities as wives and mothers, maintenance and recognition of their "mind-set" as women with an alternative view of the universe, and flexibility of training schedules which recognize their unique contributions in the dual role. They have shown themselves to be profoundly concerned with issues of ethics, social justice, empathic listening, and other intangibles we toss into the inappropriately-named rubric, "humanism in medicine" or "medical humanities."

The emergence of women into this profession coincides with possibly the greatest period of stress that physicians have known in this country. The very roots of how we practice, why we practice, and for whom are undergoing massive transplantation. Women no longer have precedents to follow, a factor which may serve them well or ill, as only time can prove. They are well aware of the dangers. It behooves us all to work together for those values which remain immutable, no matter what the circumstances under which we work.

We are all indebted to Dr Carola Eisenberg for her recent unequivocal statement in the *New England Journal of Medicine*,[2] and to Dr Arnold Relman's editorial in the same issue,[3] which calls the reader's attention to the concept of "critical mass" in the profession. By pointing out that the numbers of women have increased to the point where formerly hard-won concessions are now across-the-board necessities if the profession as a whole is to continue to function, she requires all physicians to accept that women are no longer merely welcome guests but now hosts and co-owners of the establishment. They need no longer slip away in guilt to attend to their duties at home, nor can their male colleagues continue to view these activities as unavoidable evils that detract from a woman's worth as a physician. Those realists who heed her call and take it to heart may be responsible for the most massive transformation in the internal structure that the profession has seen in almost a century.

Those of us who have watched and nurtured the growth and progress of women, particularly during these several past explosive decades, are being amply rewarded for our belief that women would someday assume their rightful place in this most blessed of professions. We will continue to monitor the steady unfolding of this process.

The articles in this issue represent an eclectic mix of some of the issues which have been of concern to women physicians and their patients, and which bear their continuing attention. We applaud the growing trend recognizing that women are not just smaller men, and note that reliable and accurate health indices cannot be established using only the data culled from research based on men. We happily view progress towards the goal we had as medical students . . . that someday, with more women physicians, we would finally make dysmenorrhea a diagnosis as extinct as diphtheria. We encourage further work on those pathologies afflicting only women, from breast cancers to parking lot deliveries.

We acknowledge not only our contributors, but the tireless planning committee of women physicians who met faithfully for a full year (despite two successful pregnancies and happy deliveries): Karyl Cole, MD; Renee Altman Fleischer, MD; Lillian Gross, MD; Mary Parisi, MD; Claudia Thomas, MD; and Ellen Yankauskas, MD.

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Consulting Editor

Special Editor
"Women as Physicians—Women as Patients"

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Women, families, and academic medicine

Now that women comprise more than one third of the graduating classes of medical schools across the country,[1] many of us in academic medicine are committed to assuring that they advance and contribute to research and the teaching of medicine in commensurate numbers. However, there are reports throughout the academic medical community of young women dropping out of or never entering tenure-track positions because they believe that the rigorous pace necessary to achieve tenure is incompatible with maintaining a household and raising children. Many gravitate towards non-tenure clinical tracks which are becoming increasingly dominated by women,[2] thus constituting, in essence, "Mommy tracks" in academic medicine. However, male junior faculty, as well as female, are expressing growing discontent with the traditional structure of title and tenure and with departmental expectations that junior faculty have few or no personal responsibilities that might interfere with their research. Many male academic physicians are the husbands of working women and therefore must share in the tasks of running a household and caring for children. It is no longer possible for them to devote all their waking hours to research and teaching.

While working class families have long been burdened by the need for both parents to work and the absence of societal supports such as adequate child care, the old paradigm for professionals split the home world from the work world by sex. The men were available to work long hours at their profession, while women bore primary responsibility for the home and children. Occasionally these arrangements were disrupted by a woman's working, out of choice or necessity. In either case, the female professional, in order to succeed, had to present herself to the work world as if she were unencumbered by family or household commitments. This was understandable since she had to compete with male professionals, the majority of whom had full-time support at home and were truly unburdened by these responsibilities.

This paradigm no longer exists in many of our private lives. However, the work world has yet to recognize that the trade-off for having talented women in the professions is a loss of some degree of individual productivity. Instead of dividing tasks by sex, we now require commitments to both home and work from each individual. This new paradigm is more fulfilling for all and ultimately more productive for society. Men can enjoy their children more and no longer feel like strangers in their own homes; women no longer feel frustrated by their financial dependence nor by their exclusion from the work world; and children benefit from the full participation of both parents in their upbringing. Less talent goes untapped than did in our mothers' generation.

Unfortunately, because the old paradigm is still with us, and the demands of the work world are often unrealistic, many women feel forced to choose between the two worlds and therefore sometimes yearn for the old "simple" separation of sexual roles. In the case of academic medicine, this problem is magnified because "full-time" means 60–80 hours a week, a time commitment incompatible with the maintenance of a household or the raising of children. Thus, just as opportunities are expanding for them, women are inclined to drop out because they still believe that when conflict arises (between work and home or between the demands of their jobs and the demands of their husbands' jobs), it is primarily their responsibility to resolve it. Their talents are lost to medical research and teaching, and they do not serve as the successful role models so desperately needed by their younger colleagues. Men may also be forgoing careers in academic medicine for the same reasons. They are certainly grumbling privately about juggling the multiple personal and professional demands of their lives.

Academic medicine needs to be restructured so that the education, creativity, and experience of all its members can be fully utilized. It is reasonable that tenure be reserved for those who excel but there is no particular need for that excellence to be achieved in the seven years that most frequently coincide with the start of a faculty member's family. More realistic demands during the early years of academicians' lives will enable them to develop the personal and professional maturity that will guarantee productive careers. There is no need for a "Mommy track" or even a "parent track." The need for a more rational system is universal. Flexibility in the pace of advancement is necessary for all young people in academic medicine; speed is not always predictive of greater overall productivity. Too often, the pressure to produce quickly gives rise to reams of useless, repetitious publications at best, and scientific fraud at worst.

Both men and women will benefit from the development of more rational policies towards maternity and paternity leaves and towards child and dependent care. The United States and South Africa are the only developed countries in the world which have no national maternity leave policy and lag behind most European countries in the quality and quantity of child care available.[3] Academic centers should take the lead in establishing realistic maternity and paternity leave policies and in providing comprehensive child care, either on-site or in the surrounding community. Such programs can benefit child study, psychiatric training, and research programs as well as providing a means of sharing resources with the surrounding community to improve "town-gown" relations.

Many will respond to these points by saying that the brightest and most dedicated physicians are not unhappy with the current demands of academic medicine and that they will continue to sacrifice their private lives to their science. While it is true that there will always be some people for whom the 60–80-hour work week is not a problem, one cannot ignore the fact that fewer bright young people are going into medicine in general and academic medicine in particular, and that many who have chosen to do so are becoming angry at the insensitivity of their institutions to the realities of their lives. The pool is shrinking and will continue to do so if academic medicine does not become a more supportive environment for young people who demand fulfilling professional and personal lives.

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Screening for breast cancer: Are physicians doing enough?

The paper on breast cancer screening by Drs Lane and Burg[1] in this issue of the *Journal* emphasizes the critical role of the primary care physician in the early detection of breast cancer. Breast cancer is the most common cancer in women, and is second only to lung cancer as the leading cancer cause of death in women.[2] Although knowledge is rapidly accumulating about the epidemiology of breast cancer,[3] not enough is currently known to significantly reduce the incidence of the disease, and treatment advances are unlikely to result in substantial reductions in mortality in the near future. Secondary prevention, or early detection through screening, offers the best hope of reducing breast cancer mortality.[4]

Breast cancer is the only cancer for which evidence from a single site randomized, controlled trial has established the effectiveness of screening in reducing mortality.[5] The Health Insurance Plan of Greater New York (HIP) study demonstrated that cancer deaths in women who were offered annual mammograms and physical examinations were significantly lower than in a control group.[6] The American Cancer Society, the National Cancer Institute, and nine other medical organizations recently issued a consensus report endorsing periodic screening mammography and clinical examination of the breast for women beginning at age 40,[7] although the recommendation for mammography screening for women in their 40s remains somewhat controversial.[8]

Despite the evidence supporting the routine application of mammography and physician breast examination, many physicians have remained unconvinced or uncertain about guidelines for breast cancer screening, particularly with respect to mammography.[9,10] Although mammography is only one component of a three-pronged strategy for early detection of breast cancer—clinical breast examination and breast self-examination are the others—evidence indicates that physicians are less likely to recommend mammography than to perform clinical breast examination or to recommend breast self-examination.[10] Mammography may be the most effective modality for detecting treatable lesions.[4]

Of particular interest with respect to mammography screening is the finding by Lane and Burg that the most frequently cited reason by women for never having had a mammogram was that their "doctor never recommended it."[1] Several other studies have underscored the importance of the physician's recommendation in persuading patients to undergo mammography. Zapka et al[11] found that women who had a regular physician were more likely to obtain mammographies than those who did not. Coll et al[12] found that 88% of women who recalled their physician advising a mammogram had obtained one, whereas among those who could not recall their physician advising a mammogram only 7% had had one.

Physicians' recommendations for mammography may also play a role in the lower five-year survival from breast cancer seen in black women compared to white women.[13] A survey of physicians in New York City found that only 7% of practitioners with predominantly black and Hispanic patient populations recommended screening mammography for their patients, compared with 23% of physicians with predominantly white patient populations who recommended mammography for their patients.[14] Analysis of data from the HIP study found that there was no difference in survivorship between black and white women in the treatment group (ie, where every woman was offered mammography and physician breast exam), prompting the authors to conclude that secondary prevention measures might succeed in closing the gap in breast cancer survival between white and nonwhite women.[15] The difference in survivorship between black and white women for breast cancer may thus possibly be explained, at least in part, by a failure of black women's physicians to recommend the appropriate screening test.

Why don't more physicians recommend screening mammography for their patients? Lane and Burg[1] found that fewer than half of the physicians in their sample (48%) reported ordering regular screening mammograms, as have other physician surveys.[9,10,16] Explanations often center around the cost of the test, although recent legislation in New York and 24 other states which requires health insurance carriers to cover screening mammography should mitigate this concern somewhat.[17] Other factors that may contribute to physician reluctance to refer patients for screening mammography include concern about radiation exposure, patient anxiety, and false positives and their associated unnecessary work-ups. An additional deterrent behind a failure to recommend screening mammography may simply be a lack of familiarity with the indications for the test. A 1984 survey found that fewer than one in ten physicians were able to recall the American Cancer Society (ACS) guidelines for mammography screening, although almost three quarters of the physicians surveyed cited the ACS as a source of information for prevention guidelines.[10] These data emphasize the need for continuing education for physicians about the indications, risks, and benefits of screening mammography. The fact that Lane and Burg[1] found that obstetrician-gynecologists were significantly more likely to comply with guidelines for breast cancer screening than were internists and family practitioners, a finding replicated in a recent survey of primary care physicians in Pennsylvania,[16] suggests that some specialties require additional efforts to educate them about the importance of screening for breast cancer.

Age-adjusted cancer mortality rates have not declined appreciably in the United States in the past 35 years, and prevention may offer the greatest potential for achieving a meaningful reduction in cancer deaths.[18] The HIP study provided clear evidence that secondary prevention of breast cancer saves lives, but a minority of women undergo annual mammography.[1] Should educational programs succeed in getting more physicians to recommend screening mammography, the data suggest that many patients would comply. Physicians can thus play a pivotal role in contributing to a reduction in mortality from breast cancer. By documenting the extent to which primary care physicians are currently recommending mammography and the willingness of patients to comply with their physicians' recommendations, the paper by Lane and Burg[1] serves to remind us of the essential role of the primary care physician in enhancing early detection of breast cancer. Physicians can do more, and should.

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Granulomatous mastitis

The most important step in the management of a patient with a breast mass is to determine the presence or absence of malignancy. In most cases, biopsy of a palpable breast mass reveals common benign or malignant breast lesions, and decisions regarding treatment generally follow established guidelines. Occasionally, a biopsy performed in a patient with a clinically suspicious breast mass reveals granulomatous inflammation. This finding is usually unexpected and raises a series of questions regarding the etiology of the disease and the proper management of the patient.

Mammary granulomas can be divided into infectious and noninfectious types. Infections such as tuberculosis, syphilis, and fungal infections may give rise to a palpable breast mass, but these are much less common than noninfectious granulomas. Noninfectious granulomatous mastitis may be an isolated finding without evidence of extramammary disease, or it may be part of a systemic, immunologic disease.

A common cause of isolated noninfectious granulomatous inflammation of the breast is mammary duct ectasia (periductal mastitis). On clinical evaluation, this disorder closely simulates carcinoma and is characterized by marked ductal dilation with stasis of ductal contents and an extensive periductal inflammatory infiltrate. Initially, the inflammatory infiltrate is rich in plasma cells (so-called plasma cell mastitis), but in later stages, a poorly circumscribed, periductal, granulomatous response develops. Although the inflammation is thought to represent a host reaction to leakage of ductal secretions, the etiology of mammary duct ectasia remains unknown. Another, much rarer, form of "idiopathic" granulomatous mastitis is not associated with duct dilation.[1] In this condition, granulomas are confined to lobules and are associated with distinct microabscesses. This form of granulomatous mastitis may persist if untreated, but there is some evidence to suggest that these cases respond to steroid therapy.[2]

Systemic diseases in which breast involvement may be observed include Wegener's granulomatosis, giant cell arteritis, and sarcoidosis. Sarcoidosis most commonly involves the lung, lymph nodes, and skin, but any tissue may be involved, and the pathologic findings are similar in each. These findings include the presence of well-circumscribed, noncaseating granulomas usually not associated with acute inflammation, necrosis, or other evidence of overt tissue damage. Asteroid bodies and Schaumann's bodies (thought to be degenerated cell organelles) are commonly found but are not pathognomonic of sarcoidosis. Granulomas in mammary sarcoidosis are not restricted to lobules or ducts. In contrast to Wegener's granulomatosis and giant cell arteritis, mammary sarcoidosis does not typically show significant arteritis. Interestingly, a small group of patients has recently been reported in whom granulomatous inflammation of the breast presents in a pattern similar to that seen in sarcoidosis.[3] In each of these patients, however, the biopsies also revealed small vessel vasculitis. The relationship between this disorder and other forms of noninfectious granulomatous inflammation is unclear.

Many hypotheses regarding the pathogenesis of sarcoidosis have centered around inhaled antigens. While a specific antigenic stimulus has not been identified, numerous studies provide evidence for a complex disorder involving T lymphocytes and histiocytes. Of interest is the finding of cancer in a patient with sarcoidosis. It is well known that carcinomas may be associated with granulomatous inflammation either adjacent to the tumor or in a regional lymph node, but sarcoid-like granulomas in patients with breast cancer are relatively rare. In this issue of the *Journal*, Shah and colleagues[4] report one of the few well-documented cases of mammary sarcoidosis in a patient with breast cancer. The diagnosis of pulmonary sarcoidosis had been previously established in this patient, and mammary sarcoidosis was subsequently discovered in the contralateral breast. Thus, in this patient, the granulomas do not appear to represent a response to carcinoma. Previous studies have suggested an increased frequency of malignancies in patients with sarcoidosis, but statistical proof for this is lacking.[5] The coexistence of these two lesions may simply be coincidental.

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Breast cancer screening

Changing physician practices and specialty variation

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ABSTRACT. Baseline surveys of primary care physicians and women on Long Island were conducted to determine breast cancer screening practices prior to introducing educational interventions to increase the use of mammography and breast examination in women aged 50 and older. Eighty-seven percent of the physicians (323) and 74% of the women (1,440) responded to postal surveys. Forty-eight percent of the physicians reported ordering regular screening mammograms on all asymptomatic female patients in this age group, with a significantly higher percentage reported by obstetrician/gynecologists than by family practitioners or internists. Only 24% of the women reported having had a mammogram within the past year. Lack of physician recommendation was the reason most frequently cited by women for not having had a mammogram. Findings point to the need for continuing medical education about consensus breast cancer screening guidelines, particularly the importance of combining both physical examination and mammography, and the need for patient education to minimize patient concerns.

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In contrast to concerns about over-utilization of modern medical technology, the 1980s were marked by reports of underutilization of mammography for breast cancer screening. In a survey of primary care physicians conducted by the American Cancer Society (ACS) in 1984, only 11% reported following accepted guidelines for annual breast examination and mammography.[1] In a Gallup survey of women aged 50 years or older published in 1983, only 41% of respondents reported having *ever* had a mammogram for either diagnostic or screening purposes.[2] The results of a study on Long Island conducted by one of us (DL) in the late 1970s and early 1980s highlighted low compliance rates among women referred by physicians for mammography from a breast cancer screening project, even when the examination was provided without charge.[3]

A consensus report on breast cancer screening guidelines was released in 1989 by the National Cancer Institute, the American Cancer Society, and 19 other medical organizations.[4] These consensus guidelines reiterated the longstanding and already widely endorsed recommendation for annual mammography and breast examination screening of women 50 years of age and older.

Because of national recognition of the need for better utilization of effective screening methodologies, a study on Long Island was funded by the National Cancer Institute as part of a special initiative to increase the use of mammography and breast physical examination in women 50 years of age and older through public and physician education. The higher breast cancer mortality rate on Long Island (Nassau and Suffolk counties), when compared to New York State as a whole, also gave the project particular local priority. Notani et al[5] reported that the age-adjusted annual breast cancer mortality rate per 100,000 white female population for 1970–1977 in New York State, excluding New York City, was 31.9. County rates ranged from 18.4 to 36.8, with Nassau County at 35.9 and Suffolk at 33.1, and variation was associated with population density. A forthcoming report from the Long Island Breast Cancer Study will examine risk factors associated with breast cancer on Long Island.

METHODS

The research methodology of the entire project is described in detail elsewhere.[6] The design includes measurement of the impact of educational interventions through baseline and post-educational intervention surveys of women and physicians in selected control and study townships on Long Island over the course of four years. This paper will focus primarily on the findings of the first phase of the project, the completed baseline, pre-intervention surveys of physicians and women, which indicated some important differences from earlier studies.

The physician education intervention and evaluation is targeted specifically at primary care physicians in the specialties of family practice, general internal medicine, and obstetrics and gynecology. There are a total of 370 physicians in these specialties practicing in the townships targeted for intervention in Suffolk County, Long Island. The physician survey was conducted from December 1987 through March 1988 to determine current screening practices prior to the educational intervention. It was limited to physicians on the staffs of the five community hospitals and/or university hospital located in the participating towns. Since in this area virtually everyone in active practice requires such a hospital affiliation, we felt that this was the best method for identifying actively practicing primary care physicians and their office locations.

The survey instrument required approximately five minutes to complete and no financial incentive was provided. The physician survey instrument collected several types of data:

- Physician characteristics (eg, sex, specialty, board certification or eligibility, type of practice, years since graduation

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from medical school)

- Knowledge of women's lifetime risk of breast cancer
- Breast cancer screening practices with regard to asymptomatic female patients between the ages of 50 and 75 (eg, approximate percent referred for screening mammograms or having breast examination, and intervals for such screening)
- Office practices with regard to advising/instructing patients to perform breast self-examination and reminder systems for recall for breast cancer screening
- Physician concerns that affect ordering of screening mammograms (eg, cost, radiation, etc) or performing breast examination (eg, patient embarrassment, lack of confidence in effectiveness of technique, etc).

Physicians were asked to rate their concern on a scale from "1" ("no concern") to "5" ("a great deal of concern").

The overall response rate for the physician survey was 87% (323/370). The survey was completed primarily at hospital department meetings (69% of respondents). Those not in attendance at the meeting were mailed the survey, and three attempts were made to obtain a response by mail, the third time by registered mail.

The women's survey was conducted by mail and included an incentive prepayment of two dollars. Three attempts were made to obtain a response, the final attempt also sent by registered mail. Women in the target age group were reached through a listing from the New York State Department of Motor Vehicles of licensed female drivers aged 50-75 residing in the study towns.[6] The women's survey instrument included data on demographic characteristics, utilization of mammography and breast physical examination screening, reasons for never having had a mammogram, concerns that would affect obtaining a mammogram or breast physical examination, and frequency of physician visits when not ill (for a check-up). Women were similarly asked to rank their concerns on the above scale from 1 to 5.

The response rate to the women's survey was 68% (1,440 out of 2,118), excluding those who were known to have died or moved out of the area. After additionally excluding those who did not receive the survey because they moved and had no forwarding address, the response rate increased to 74%. Respondents who had had breast cancer and/or those of unknown age were excluded from the analysis.

Statistical analysis of the survey data was performed to identify factors associated with use of breast cancer screening. Chi-square tests were used for examining significant differences between physician specialty groups.

RESULTS

Physician Characteristics. The majority of physician respondents were male (84%), with a mean age of 47 years (SD = 10.7 yr), board certified or eligible (92%), in private solo or group practice (86%). There was considerable variation in years since graduation from medical school, and all three target specialties were well represented. The overall response rate of 87% varied to some degree by specialty, and was lower for family practitioners (FPs), 80%, than for obstetrician/gynecologists (Ob/Gyns), 92%, and general internists (IMs), 91%.

TABLE I. Physician Reporting of Regular Screening of Asymptomatic Female Patients Aged 50-75 Years

	Physician Reporting			
	Mammography Referral (N = 310)		Breast Examination (N = 280)	
% Referred or Examined	N	%	N	%
0	11	3.5	6	2.0
1-49	51	16.5	13	4.6
50-74	45	14.5	31	11.1
75-99	55	17.7	55	19.6
100	148	47.7	175	62.5

TABLE II. Physicians Regularly Performing Breast Cancer Screening on All Asymptomatic Women Aged 50-75 Years by Specialty

	100% Referred for Mammography (N = 310)				100% Examined (Breast Physical) (N = 280)			
Physician	Yes		No		Yes		No	
Specialty	N	%	N	%	N	%	N	%
Obstetrics/ Gynecology	81	86.2	13	13.8	84	97.7	2	2.3
Family/General Practice	30	29.7	71	70.3	43	45.7	51	54.2
Internal Medicine	37	32.2	78	67.8	48	48.2	52	52.0
Total	148	47.7	162	52.3	175	62.5	105	37.5
	$\chi^2 = 79.983$, df = 2 $p < 0.0001$				$\chi^2 = 65.629$, df = 2 $p < 0.0001$			

Breast Cancer Screening Practices of Physicians. Approximately 48% of the responding physicians reported that they refer all their asymptomatic female patients aged 50-75 for regular screening mammograms (Table I), whereas about 20% reported referring less than half of their patients for the examination. In addition, about half of the respondents reported that they refer some or all of their female patients in this age category for screening mammograms on an annual interval. Approximately 33% refer such women every two years, and an additional 2% refer them every one or two years, for a total of 85% referring annually and/or biannually.

Mammography referral rates did not differ significantly by physician gender, age, or years since graduation from medical school. Physicians who were not board certified or eligible (N = 26) reported lower referral rates than board certified or eligible physicians, but their number is too small to draw inferences about differences between the two groups.

Ob/Gyns were significantly more likely than FPs and IMs to refer for screening mammography (Table II). Eighty-six percent of Ob/Gyns reported referring all asymptomatic female patients aged 50-75 for regular screening mammograms, compared to 30% by FPs and 32% by IMs. Similarly, an annual frequency for mammography was recommended for this age group by a significantly greater proportion of Ob/Gyns (71%) than by FPs (44%) and IMs (40%) (Table III).

With regard to physical breast examination practices, 63% of all physician respondents reported that they regularly perform such examinations on *all* their asymptomatic female patients aged 50-75 (Table I). Eighty-three percent of the respondents followed an annual interval for such examination.

Virtually all (98%) Ob/Gyns reported that they perform regular

TABLE III. Physician Report of Annual Interval for Breast Cancer Screening of Asymptomatic Women Aged 50-75 Years by Specialty

	Annual Referral for Mammography (N = 313)				Annual Breast Physical Exam (N = 316)			
Physician	Yes		No		Yes		No	
Specialty	N	%	N	%	N	%	N	%
Obstetrics/ Gynecology	67	71.3	27	28.7	90	95.7	4	4.3
Family/General Practice	46	44.2	59	55.8	88	83.8	17	16.2
Internal Medicine	46	40.2	69	60.0	85	72.6	32	27.4
Total	159	50.8	154	49.2	263	83.2	53	16.8
	$\chi^2 = 23.184$, df = 2 $p < 0.001$				$\chi^2 = 19.955$, df = 2 $p < 0.001$			

TABLE IV. Physician Level of Concern About Items Affecting the Ordering of Screening Mammograms by Specialty

	Level of Concern				
	Obstetrics/Gynecology (N = 95)	Family/General Practice (N = 108)	Internal Medicine (N = 120)	Total (N = 323)	
Items	Some	Some	Some	Some	None
Inadequate insurance coverage*	38.0%	67.9%	54.2%	54.0%	46.0%
Equivocal radiology reports	41.3%	61.5%	52.9%	52.9%	47.1%
High price of mammograms*	34.8%	69.2%	47.5%	51.3%	48.7%
Radiation exposure	32.6%	37.7%	37.2%	36.1%	63.9%
Possibility of unnecessary biopsies	26.1%	35.2%	37.2%	33.3%	66.7%
Not cost effective*	17.4%	38.7%	39.7%	33.2%	66.8%
Patient discomfort	29.4%	31.4%	35.8%	32.2%	67.8%
Physical breast exam is sufficient†	15.4%	32.7%	36.1%	28.6%	71.4%
Too much time and effort in discussing screening mammography with patients	15.2%	21.9%	23.1%	20.6%	79.4%

* Chi-square test significant at $p < 0.001$, df = 2.

† Chi-square test significant at $p < 0.01$, df = 2.

clinical breast examinations on all their asymptomatic female patients aged 50–75, versus 46% of FPs and 48% of IMs (Table II). The performance of clinical breast examination with annual frequency was also significantly greater for Ob/Gyns at 96% compared to 84% for FPs and 73% for IMs (Table III).

Physician Concerns About Mammography Referral. Table IV ranks responses to items of concern that might influence physician referral for mammography. While not shown in the table, responses to these items tended to cluster on the low end of the scale (ie, little or no concern). Therefore, the scale has been collapsed into "some concern" versus "no concern" and shows that the majority of the physicians had no concern whatsoever about most of the items. The top concerns, mentioned by more than half of the physicians, related to cost—inadequate health insurance coverage and high price of mammograms. Physicians were aware of the cost of mammography because 85% correctly estimated the local prevailing costs at the time to range from $75 to $150. Fifty-three percent of physicians mentioned "equivocal radiology reports," suggesting possible medicolegal concerns.

Ob/Gyns were significantly less likely than FPs or IMs to be concerned about ordering a mammogram because of cost factors or feelings that it is not cost effective or that a physical examination is sufficient (Table IV). The three specialties were closest to one another in the proportions reporting the following concerns about ordering mammography: radiation exposure, patient discomfort, possibility of unnecessary biopsies, equivocal radiology reports, and time and effort in discussing mammography with the patient.

Physician Concerns About Clinical Breast Examination. All items relating to performance of the clinical breast examination were of relatively low concern among the respondents, with patient embarrassment and reluctance being cited most frequently (38%) (Table V). Of interest is that 22% indicated that they lacked confidence in their own technique, and this may be an underestimate due to reluctance to report such insecurity. Ob/Gyns were less likely (than IMs and FPs) to be concerned about performing a physical breast examination because of lack of confidence in the technique or in their own breast examination skills, patient embarrassment, time and effort involved, or other health problems to be checked (Table V).

Ninety-four percent of all physician respondents indicated that they recommended breast self-examination (BSE), and 80% provide BSE instruction in the office. Ob/Gyns were more likely than FPs and IMs to provide BSE instruction in the office (97% Ob/Gyn; 84% FP; 64% IM); to have reminder systems for patient recall for mammography (53% Ob/Gyn; 28% FP; 29% IM) and reminders for breast examination (63% Ob/Gyn; 44% FP; 46% IM); and to know that the lifetime risk of breast cancer is one in ten (68% Ob/Gyn; 56% FP; 47% IM).

Breast Cancer Screening Practices of Women. The postal survey of women in the target age group was conducted in the same townships prior to the planned public education intervention. The average age of the respondents was 61 years (SD = 6.53 yr). Twenty-eight percent had annual incomes under $15,000; 50.4% were high school graduates, and 26% had completed a college education; 69% were currently married; and 92% were white.[7] As shown in Figure 1, about 48% of respondents indicated that they had ever had a mammogram, and about half of those who had ever had a mammogram had had only one. Twenty-seven percent had had a mammogram (including both diagnostic and screening) in the last year, and 19% had had a screening mammogram in the past year.

Women who reported having had a mammogram were significantly more likely to be younger (50–64 yr vs 65–75 yr), to have incomes of $15,000 or more, and to be college educated.[7] By far the most frequently reported reason for never having had a mammogram (44% of women), was that their "doctor never recommended it." The proportion of women having annual mammograms in-

TABLE V. Physician Level of Concern About Items Affecting a Clinical Breast Exam by Specialty

	Level of Concern				
	Obstetrics/Gynecology (N = 95)	Family/General Practice (N = 108)	Internal Medicine (N = 120)	Total (N = 323)	
Items	Some	Some	Some	Some	None
Patient embarrassment and reluctance*	9.8%	55.1%	43.0%	37.5%	62.5%
Too many health problems to screen for first*	5.4%	33.6%	39.2%	27.5%	72.5%
Lack of confidence in effectiveness of the technology†	11.8%	31.1%	30.3%	25.4%	74.6%
Lack of confidence in my own technique*	7.6%	21.5%	32.2%	21.8%	78.2%
Time and effort in doing examination*	2.2%	20.8%	25.0%	16.5%	83.5%

* Chi-square test significant at $p < 0.001$, df = 2.

† Chi-square test significant at $p < 0.01$, df = 2.

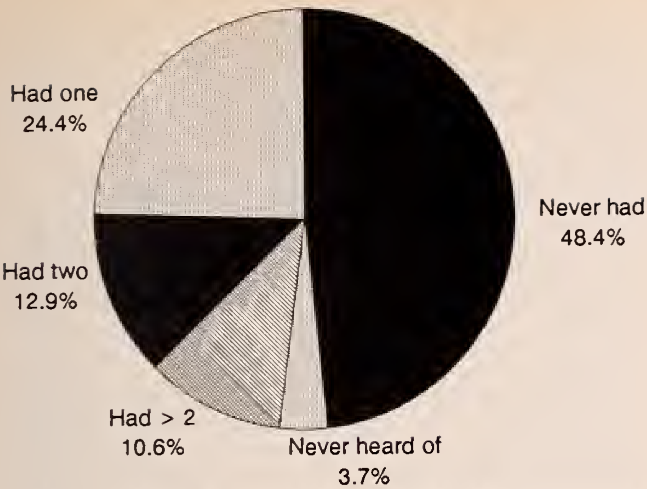

FIGURE 1. Mammography experience among surveyed women (N = 1,440).

creased with the frequency of regular doctor visits (Fig 2). Those who visited the doctor yearly versus less frequently, however, were still significantly more likely to have as a reason for never having a mammogram that the doctor did not recommend it ($\chi^2 = 9.934$, df = 1, $p = 0.002$). The other reasons for not having had a mammogram in descending order of importance to women were as follows: no breast problems; never thought of it; cost; radiation; and fear that the examination may be painful. Among all survey respondents, whether or not they had had a prior mammogram, the most commonly reported concern (62% of all women) was fear of the results of the examination, while concern about radiation (54%) was also prominent.

DISCUSSION

When compared to data from the 1984 national ACS physician survey and other surveys, including one of New York City physicians,[8] the findings from this Long Island physician study suggest a trend over time toward increased physician referral for screening mammography of women in this age group. Similarly, the women's reports of mammography utilization in this study were higher than those reported in the 1983 Gallup Survey,[2] and slightly higher than results shown in the US Health Interview Survey of 1987.[9] The progress in physician acceptance of the value of mammography, however, is not uniform or complete.

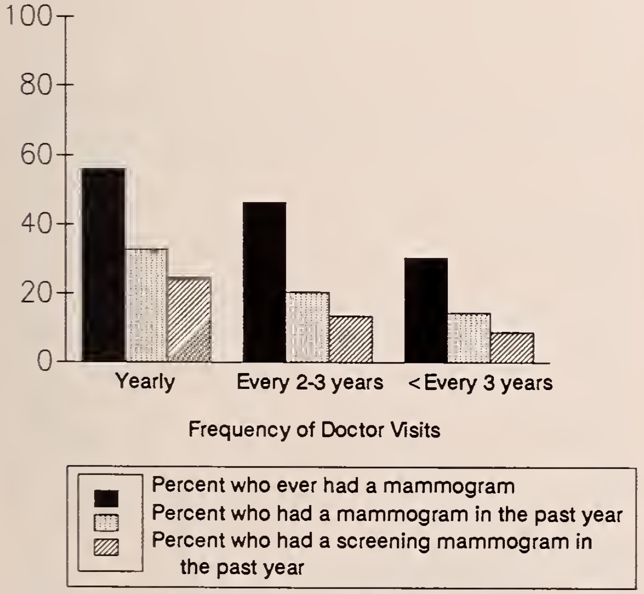

FIGURE 2. Screening behavior of women age 50–75 years (N = 1,440) by frequency of regular physician visits.

Obstetrician/gynecologists far exceeded their general internist and family practitioner colleagues in following the guidelines for breast cancer screening. These specialty differences were also found in earlier studies.[1] This is of particular concern since it has been shown that women over 50, who are at higher risk for breast cancer by age, are much more likely to be seeing a family practitioner or internist than a gynecologist for regular health care.[10] This suggests that continuing medical education (CME) in breast cancer screening should be specifically addressed to family practitioners and internists.

In the 1983 American Cancer Society Survey of primary care physicians, 80% reported that they followed the ACS guidelines for annual breast examination of all asymptomatic women, including women 50 years and older.[1] Only 63% of Long Island physicians reported that they performed regular breast examination on all their asymptomatic female patients in this age group. Although different phrasing of the questions in the two surveys precludes a direct comparison of data, the difference in findings prompts our speculation that there may be increasing reliance on mammography alone for breast cancer screening, despite the fact that mammography misses some 10% to 20% of the lesions detected by physical examination.[11,12]

Physician reporting of lack of confidence in breast palpation skills as a deterrent to the performance of breast examination points to the need to give greater emphasis to breast physical examination skills during medical school and residency training, as well as to provide CME opportunities for such clinical skills training. The recently issued consensus guidelines reiterate the importance of performing *both* physical examination and mammography in breast cancer screening. Hopefully, this will be brought to the attention of physicians who only order mammography, as well as those who do not order mammography because they believe that breast physical examination is sufficient.

Physician concern about the lack of insurance coverage for mammography may in part be addressed by legislation passed in 1989 in New York State which requires health insurance carriers in the state to provide some coverage for screening mammography, and by proposed legislation to provide coverage through Medicare. Cost concerns may also be reduced by a trend toward reduced charges for screening mammography advocated by nationally recognized radiologists and other groups.[13]

Self-reported screening behavior by physicians may be biased by overestimation.[14] This may help to explain the contrasting data from the physician and women's surveys, which indicated that even among women who regularly visit their physician for check-ups, lack of physician recommendation was the primary reason given for never having had a mammogram. It is clear that even if the physician believes in the value of periodic mammography screening, this is frequently not being adequately communicated to the patient. Physician training in patient education and communication is also needed to address the causes of patient noncompliance with mammography recommendation.

While radiation exposure from mammography was a minor concern among physicians in this study, it was a prominent concern of women. This was similarly found to be an important deterrent to the use of mammography by women in an earlier study in one of these communities performed by one of us (DL).[3] The current findings suggest that fear of radiation should be explored during the office visit in order to enhance compliance with mammography referral. Other patient education needs are indicated by the findings that "fear of the results of the exam" was cited most frequently as a concern among all women, whether or not they had had a mammogram, and that "no breast problem" or "not necessary" was cited frequently by women as the reason for never having had a mammogram. These point to the need to emphasize that the purpose of screening is to detect disease before there are symptoms, and that breast cancer is highly curable and treatable with lesser surgery when detected in the early stages.

The second phase of this project has involved the development of CME programs and materials to address the above educational needs. While it is beyond the scope of this paper to discuss these initiatives, such materials can be obtained from the authors on request. The yet-to-be conducted post-intervention surveys of physicians will provide information on the efficacy of this intervention approach, particularly when compared to a control group of physicians, in order to correct for the secular trends that we have observed in the past.

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Senior women physicians: The question of retirement

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ABSTRACT. Little is known about women physicians in their senior years. This paper describes a cross-sectional survey of 21 women physicians ranging in age from 59 to 95 years in eight different medical fields. The survey showed that 14 (66.7%) worked after age 65, including four who worked into their 70s and four who worked into their 80s. Although reasons for retirement varied, most retired due to personal illness or the wish to pursue other interests. Most respondents (76.2%) were married or had been married; 80% of these had children. This survey also explored initial practice styles and changes over time, and retirement planning and financing. Respondents also were asked to identify role models and to share their own mentoring activities. Traditionally, women professionals often have been thought to have shorter careers than men due to family constraints. Yet most of the women in this sample have had long-lived careers with simultaneous marital and family responsibilities.

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What happens to women doctors when they reach the traditional transitional ages of 62-65? Do they retire or do they continue working? Generally, little is known about women physicians in their senior years. The most apparent explanation for this lack of information is that significant numbers of older women physicians have only become a definable population in the last 20 to 30 years.

The increase in the numbers of older women physicians stems directly from the increase in the numbers of women entering medical careers. In 1905 there were 219 women graduates of American medical schools, only 4% of the total graduating that year. By 1988, there were 5,215 women graduates, representing 32.8% of the total graduates, more than an eight-fold increase.[1] As a result, the number of women physicians over 65 years of age has

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more than doubled in less than 20 years, from 2,736 in 1967 to 5,822 in 1985.[2] As this sector of the medical community grows, so will the interest in the issues that it faces.

METHODS

To learn more about the retirement practices of women physicians, a survey was compiled eliciting such demographic information as age, year of graduation from medical school, number of women students in the graduating class, marital status, number of children, field of medicine, and type of practice. Questions on retirement included retirement status, age at retirement, reasons for retirement, what retirement planning was done, and how retirement was financed. In addition, most of these women doctors were asked if they had treated primarily women patients. How did their practice change over time? What role models and mentors had been available to these doctors? Did they serve as mentors to others?

The sample was composed from several sources, including members of the American Medical Women's Association Seniors, personal acquaintances, and members of the local county medical society. Surveys were sent to 20 physicians across the country. Sixteen responded, representing an 80% return rate. Additionally, five local women were interviewed by telephone. Although 21 women constitutes a small sample, the responses represent a variety of attitudes about retirement. Clearly, there is a broad spectrum of outlooks based on personal preferences and priorities.

RESULTS

The ages of the respondents ranged from 59 to 95 years. The respondents had graduated from medical school between 1921 and 1955. Four women graduated in the 1920s (19.0%), five in the 1930s (23.8%), nine in the 1940s (42.9%), and three in the 1950s (14.3%). The percentage of women in co-educational medical school graduating classes varied from a low of two women in 250 students (0.08%) in 1938 to a high of 10 in 38 (26%) in 1950. One doctor trained in Yugoslavia where 50% of her class was female in 1955. Two respondents attended women's medical colleges.

Most respondents were married or had been married. Two of the four women graduating in the 1920s were married (50%), four of five graduating in the 1930s (80%), six of nine graduating in the 1940s (66%), and all three of those graduating in the 1950s (100%). Generally, 52.3% of the entire sample was married, 14.3% widowed, 4.7% divorced, and 23.8% single. Of those who were married, widowed, or divorced, 80% had children.

A wide variety of specialties were represented among these 21 physicians including dermatology, obstetrics and gynecology, psychiatry, family medicine, general practice, pediatrics, pathology, and internal medicine. Despite this large distribution of fields in this small sample, it is interesting that seven women (33%) identified themselves as family physicians or general practitioners, which reflects the less specialized nature of medicine prior to the 1960s.

These women opted for one of several practice styles. Private practice was the most common choice (62%). Of the 13 in private practice, nine were solo practitioners, two worked in partnerships with their husbands, one worked with her son, and one was in a group practice. Two women chose academic careers, two entered the Veteran's Administration, one worked in a state hospital, and one was employed by the armed services. Solo practice was a much more common choice among these women than it is among women trained in the last 10–15 years.

Of the 21 women responding, 15 (71%) had retired at ages ranging between 51 and 88 years, with 10 (48%) retiring in their 60s. Of the six who were not retired, two (ages 73 and 83) planned retirement within the next year. Another woman, age 68, planned retirement but was not sure when. The remaining three physicians could not imagine retiring from direct patient care. One rural general practitioner, age 89, stated that she would only retire if absolutely necessary due to health constraints. A dermatologist, age 71, admitted that she had not thought of retiring at all. Another woman, age 63, said that she could not conceive of leaving her busy family practice, and would only retire if it was absolutely impossible for her to work at all.

Four women retired due to personal illness at ages 51, 68, 73, and 88. The youngest retiree left her family practice at age 51 on medical disability due to increasing angina. She is now 66 years old and enjoying a multiplicity of hobbies and volunteer activities. A dermatologist retired at age 68 after suffering a fractured ankle and transient ischemic attack. Another woman retired from pediatrics at age 73 due to breast cancer requiring bilateral mastectomies. She maintains her license, however, and attends continuing medical education courses whenever possible. Finally, one obstetrician/gynecologist retired at age 88 due to cardiac problems but did deliveries up until age 86.

The remainder of those retired chose to leave medicine in order to pursue other interests and priorities, including grandchildren, cultural activities, community organizations, religious groups, gardening, writing, and peace activism.

Many wished to travel while they and their spouses were still healthy. One internist, age 59, cited increasing medical regulations and bureaucracy as her reason to retire from practice. She stated that although she loved to care for patients, she felt suffocated by the burgeoning rules and restrictions dictating her clinical decisions. She plans to retire at age 60 and travel with her husband.

DISCUSSION

How do these women prepare their practices and themselves for retirement? Several women indicated that they changed their form of practice in anticipation of retirement. One pediatrician left private office practice and became a physician in the student health service of her local college. (When she decided to retire several years later, she was not burdened by patient referrals, as the college simply hired another physician to replace her.) Another woman closed her private office and limited her practice to hospital consultations in infectious disease. When she retired, she simply stopped consulting. Others remained in private practice until retirement, closing their practices in the traditional way.

Most of the 21 respondents financed their retirement from a variety of sources. Nineteen (90.5%) relied on personal savings and investments including rental income, annuities, and insurance dividends. Fifteen (71.4%) had pension plans providing part of their retirement income. Seven (33.3%) had social security income in these years. Three women (14.3%) found it necessary to do some part-time work to supplement their retirement income. One physician depended on her medical disability payments as a main source of income. One woman bred and sold horses. Another lived off the income from the sale of her practice.

The survey results also indicated that a majority (57%) of respondents treated mostly women patients. Two of these 12 women were obstetrician/gynecologists. Four physicians noted that when they first opened their practices (dermatology, general practice, internal medicine, and psychiatry), most of their patients were women but as they became more experienced and more accepted in their communities, more male patients came to them for care.

When asked how their practice changed as they matured, several women stated that they had become more specialized within their field. Two women noted that their patients grew older with them. One family physician gave up pediatrics; another gave up obstetrics. One respondent commented on her increasing confidence and humane involvement with patients as she grew older. Several women decreased their working hours by changing from private practice to salaried positions or consulting. One internist stated that she moved from an 18-hour work day as a full-time faculty member, to a 13-hour work day as a private practitioner, to a five-hour work day as an inpatient consultant over the course of her career.

When asked if they saw themselves as mentors to young women and men in medicine, 17 of 21 answered yes. Two said no and two said they were not sure if they had been mentors. One stated that she felt medicine had changed too much for her to serve as a mentor. Of those who did identify themselves as mentors, most had offered encouragement and advice to young women (and men) students and trainees. Four spoke of participating in activities within their local branch of the American Medical Women's Association to help younger women as role models. One woman had two daughters who became physicians. Another served as the first woman president of her state's Academy of Family Physicians. Several served as faculty advisors. Another invited college students to live and work with her during the summer months so they could learn about medical practice.

Most of the respondents said that they had a role model or mentor who influenced their decision to become a doctor. Sixteen (76%) identified specific individuals who were important in this role including their family physician, physician brother, physician husband, upper-class college friend, certain professors, older women physicians, and historical figures such as Madame Curie. One woman wrote that she had been a social work student in college when she developed a cold that necessitated a visit to the student health service. She was so impressed that the doctor was a woman that she changed her major to premedicine.

Of those five who denied having any mentors, one reported, "I just decided to become a physician and then became one." Another could not identify any mentors but received a scholarship to Yale Medical School during World War II when there were no men available for the award. Finally, a 1926 graduate said there were no mentors available to her as she established her medical career.

Although this survey represents a small cross-sectional sample, it begins to identify questions for further research. Fourteen (66%) of the 21 women surveyed have worked after age 65, including four who worked into their 70s and four who worked into their 80s. Would a larger sample show a similar number of women working after the traditional retirement age of 65%? How does this compare with the retirement practices of their male counterparts? Do these women retire at a later age than men doctors? Traditionally, women professionals often have been thought to have shorter careers than men due to family constraints, yet in this sample most women have had long-lived careers with simultaneous marital and family responsibilities.

How have attitudes on retirement changed between generational cohorts of women physicians? How has the changing societal work ethic affected this population? Do the women who trained in the 1920s work longer than those who trained in the 1950s? Do attitudes regarding retirement change as these individuals grow older? Although this sample includes women graduating in the 1920s, 1930s, 1940s, and 1950s, the numbers are too small to identify general trends. Further research with larger populations of senior women will be necessary to address these questions.

Although there is no formal organization of older women physicians, the American Medical Women's Association numbers 254 retired doctors among its 11,000 members. In 1987, several of these women began a subgroup of the organization known as the AMWA Seniors. They publish a newsletter two to three times each year and present workshops on issues of interest to older women at the annual AMWA National Conference. In 1987, they held a very successful workshop entitled "Retirement Issues for AMWA Seniors."

During this meeting, six women spoke of the transitions from practice to retirement, each describing their individual experiences. One woman became a medical consultant to pharmaceutical firms using her experience to begin this new and exciting independent business. Another moved into a retirement community offering intellectual stimulation, opportunities to make new friends, and health assistance if illness should occur. A third senior suggested gradually reducing professional work hours and increasing hours in another setting. She tapered off her private practice and became a full-time cattle rancher. A fourth participant transferred from a demanding practice to a less stressful level of medical care doing locum tenens work. Another physician retired completely and began all over again as a full-time student in the entirely unrelated fields of philosophy and creative writing, in a new place with new friends of many age groups. Finally, a sixth woman described becoming involved in medical ethics after leaving patient care. She educated herself in this expanding field and found herself in great demand but with less physical stresses than her practice had entailed.[3]

The only organization specifically designed for retiring medical doctors is the Association of Senior Physicians based in Chicago which offers memberships to any physician over 55 years of age. Its purpose is to educate physicians regarding professional, emotional, social, and financial aspects of retirement. It provides seminars on retirement planning, replacement insurance policies, and a bimonthly newsletter. This organization has women members and seminar attendees, but as yet it has not sought to define or address the specific needs of the retiring female physician population.

The diversity and rich heritage of women in medicine is alive in senior women physicians. Like their male counterparts, women doctors have individual solutions to the challenges of aging. Each woman responding to this survey has chosen her own path to fulfill personal needs in later years. Some are happy to continue 80-hour work weeks until they are physically unable regardless of age. Others will formally retire in their 60s to enable themselves to develop other interests. As the face of medical practice continues to change, women physicians will continue to find new approaches to their senior years.

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Primary female syndromes: An update

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Articles discussing menstrual cramps, premenstrual syndrome, endometriosis, or some similar health concern frequently appear in women's popular literature. One might think that the American woman has an unhealthy preoccupation with her health and internal organs, an idea more than once communicated to the medical student and resident. But a more accurate assessment would probably be that many women no longer want to live in discomfort and ignorance with regard to their physical well-being. They have higher expectations of medical science and are better educated than ever before. If not actively seeking information, they are gaining knowledge while at leisure, reading a magazine, or watching television. These women then turn to their physicians for confirmation and treatment.

Women's complaints have long been both baffling and frustrating for the medical profession. Most physicians, being male, have no personal experience from which to draw. Past research has produced little hard data on pathophysiology; and even current technology has only started to explain the physical and emotional changes associated with basically normal hormonal function. Common is the situation in which concerned patients, harboring the misconception that all doctors are comfortable and conversant with such entities as premenstrual syndrome (PMS), confront physicians whose levels of expertise or tolerance for these topics may vary widely.

Physicians who are willing to address these complaints, rather than dismiss them as insignificant since they are not life-threatening, must address the following question: Is there true dysfunction or is this another case of the gynecologic "worried well"?

Consider the experience of dysmenorrhea. For centuries, women were told that their complaint of menstrual cramps was psychosomatic. Then, with the advent of the nonsteroidal anti-inflammatory drugs (NSAID) for relief of arthritis, a serendipitous treatment for dysmenorrhea was found. Now, primary dysmenorrhea is recognized and treated, without questioning the emotional stability of the patient.

With time and technical advances, more understanding of the presently puzzling complaints of women should be forthcoming. Meanwhile, women with such complaints as PMS and chronic pelvic pain have very real problems and deserve the attention and support of their physicians as well as an explanation of current options for treatment. This article reviews the advances in diagnosis and treatment of some of these disorders.

DYSMENORRHEA

Difficult or painful menstruation without evidence of anatomic or infectious cause was long thought to be a functional disorder of women. Physiologic dysmenorrhea, however, has since been found to occur as a result of excessive prostaglandin production by the endometrium. This excess is believed to be responsible for hypercontractility of the uterus and its ensuing pain. Budoff[1] proposed the comprehensive concept of "dysmenorrhea syndrome" to include the associated exogenous symptoms of headache, nausea, vomiting, diarrhea, and syncope. This constellation of discomforts results in the greatest single cause of lost school and work days among women.[2] Secondary dysmenorrhea develops from identifiable pathology, which is presented in Table I.

Diagnosis. The diagnosis of primary, or physiologic, dysmenorrhea remains a diagnosis of exclusion of secondary dysmenorrhea. The former is associated with a normal physical examination and occurrence with ovulation and menses. The latter is associated with organic disease and atypical pain with the menstrual cycle.

Laboratory tests may show anemia and an elevated white blood cell count and erythrocyte sedimentation rate (with pelvic inflammatory disease, for example) but are not specifically diagnostic. Radiography can provide some information, such as that offered by a hysterosalpingo-

TABLE I. Differential Diagnosis of Dysmenorrhea

Non-progressive	Physiologic dysmenorrhea
Progressive/ recurrent	Endometriosis—endometrioma
	History of pelvic inflammatory disease
	History of intrauterine device
	Ovarian cyst—follicle cyst, corpus luteum cyst
	Adenomyosis
	Neoplasm
	Abdominopelvic adhesions
	Anatomical anomaly
	Chronic salpingitis
	Uterine polyps
Acute	Pelvic inflammatory disease
	Ruptured ovarian cyst
	Ectopic pregnancy/miscarriage
Non-gynecologic	Pyelonephritis
	Musculoskeletal pain
	Appendicitis
	Irritable bowel syndrome

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gram, but the diagnostic potential is sufficiently limited to force one to question the risk of radiation to the reproductive organs and the adverse effect of this procedure on pelvic inflammatory disease (PID). A computed tomographic (CT) scan also carries the same questionable risk-benefit ratio. Ultrasound is noninvasive and safe, but often aids with diagnosis only in extremely severe disease. Laparoscopy provides the best opportunity to investigate the possibility of organic disease, as well as a chance to institute surgical therapy. However, it is invasive, expensive, and not always enlightening.

Treatment. Primary dysmenorrhea is usually responsive to prostaglandin antagonists or oral contraceptives. However, up to 20–30% of patients do not respond to these agents. (The treatment of secondary dysmenorrhea depends on the specific pathologic entity.)

Several NSAIDs are currently approved by the Food and Drug Administration (FDA) for the treatment of dysmenorrhea. These include ibuprofen, mefenamic acid, naproxen, and ketoprofen. Other NSAIDs reportedly of benefit but not FDA approved are pirixicam and meclofenamate.

Oral contraceptives have a high rate of effectiveness against cramping, but must always be prescribed with the usual cautions, particularly regarding the risk of fibroids or neoplasm. These agents also help with secondary dysmenorrhea in the suppression of functional ovarian cysts and mittelschmerz.

In general, intrauterine devices (IUDs) are contraindicated in women with moderate to severe dysmenorrhea. The exception to this is the Progestasert™ IUD, which has been shown to decrease pain and heavy bleeding.

Transdermal electric nerve stimulation (TENS) as a treatment modality for chronic pain syndromes has received only lukewarm response from practitioners and patients alike. A unit has been developed by 3M Med-Surgical Division with special electrodes for placement over the pelvic organs for relief of dysmenorrhea (Optenz Feminine Pain Relief System). The unit is available in Europe now, but extensive safety and efficacy studies mandated by the FDA has delayed its release in the United States.

Laparoscopic uterine nerve ablation (LUNA) destroys the sympathetic nerve fibers to the uterus via the uterosacral ligaments. One study found a 60% effectiveness rate.[3] Extreme measures such as total abdominal hysterectomy with bilateral salpingo-oophorectomy were attempted in the past, with an estimated 130,000 women having had this procedure solely for relief of dysmenorrhea.[4]

PREMENSTRUAL SYNDROME

Premenstrual syndrome is a cluster of physical and emotional changes that affects almost every woman on a cyclic, premenstrual basis. Perceptions, complaints, disability, and morbidity vary considerably. So many women consider it "normal" to have their monthly complaints that it is rarely volunteered as a part of the medical history. It is even less frequently inquired about by physicians. As a result, the data on prevalence of PMS are limited, with estimates ranging from 20% to 95% of women having mild to moderate symptoms, and 1% to 40% having symptoms severe enough to disrupt life style.

Frank[5] first described the syndrome in 1931, making the current status one of more than 50 years of limited advancement. Since there is no definitive test for the condition, the practitioner must first rule out conditions such as those listed in Table II.

TABLE II. Differential Diagnosis of Premenstrual Syndrome

- Endometriosis
- Pelvic inflammatory disease
- Ovarian cysts
- Thyroid disorder
- Hyperprolactinemia
- Nutritional deficiency
- Psychiatric illness

Some practitioners have found it helpful to use a diary/questionnaire to improve their rates of detection of PMS. Physicians who inquire directly about premenstrual symptoms can find a remarkable consistency among the more common complaints.[6,7] The physical changes include breast tenderness; bloating; fluid retention; craving for sweet or salty foods; acne; and disturbance of sleep, bladder, and bowel function. Psychological changes reflect a spectrum of exaggerated moods including depression, lethargy, anxiety, irritability, and aggressiveness. Symptoms generally occur seven to ten days prior to menstruation (although up to 14 days has been reported), and then, once the menstrual flow begins, disappear as mysteriously as they came. Dysmenorrhea is often found in association with PMS but is considered to be a separate entity.

Subsyndromes of PMS have long been appreciated and can be useful in understanding and treating this disorder. Abraham's[8] classification is described in Table III, which also reports some of the current estimates of relative frequency of the subtypes.

Etiology. Many theories have proliferated as to the pathophysiology of PMS.[8-10] Since the symptoms only present in women with functioning ovaries and are chronologically tied into the menses, it has been logically assumed that there must be some hormonal shift causing the phenomena. If this is the case, the shift must be quite subtle, for it has eluded researchers who have hospitalized women with severe PMS during the luteal phase, taking serum hormone levels around the clock, without demonstrating any consistent, significant alteration of normal circulating values.

TABLE III. Classification of Premenstrual Syndrome[8]

Class	Symptoms	Frequency
PMT-A	Anxiety, irritability, mood swings, tension	70–90% (Most common)
PMT-C	Cravings for sweets, hypoglycemic-like symptoms (fatigue, dizziness, palpitations, headache, increased appetite)	30–40%
PMT-D	Depression, crying, withdrawal, forgetfulness, confusion, insomnia	1–20%
PMT-H	Breast tenderness, fluid retention, weight gain, bloating	60%

Ongoing research continues to concentrate on the luteal phase of the cycle, with attempts to ascertain any hormonal imbalance, such as decreased or unopposed estrogen, decreased progesterone, or an altered estrogen-progesterone ratio.[11,12] Thyroid disorder is another hormonal possibility being investigated. Roy-Byrne et al[13] found an increased incidence of subclinical hypothyroidism and blunted thyroid releasing hormone (TRH) stimulation among PMS subjects. Other investigators suspect a disorder of androgen or melatonin (alpha melanocyte stimulating hormone) metabolism.[7]

Endomorphin levels fluctuate with the menstrual cycle. Chuong et al[14] have found premenstrual decreases in beta endorphins. There is also a possible link between the effects of endorphins on prolactin (hyperprolactinemia), vasopressin, and prostaglandin E (PgE) and the symptoms of fluid retention and breast tenderness.[15] The latter symptoms are thought by others to result from defects in aldosterone and/or renin-angiotensin mechanisms.[17]

Nutritional deficiency has been found to be associated with PMS but not causal in nature.[16] There is ongoing research focusing on the roles of vitamins B_6 and E, as well as magnesium.[17] Hypoglycemia and altered carbohydrate metabolism are the main features of a subtype of PMS (PMT-C). With the discovery of insulin receptors in the ovary, more research may explain their role in normal hormone function. DePirro[18] demonstrated marked variation in insulin receptor concentration in monocytes of normal women. Insulin cell binding fractions were almost twice as high during the follicular phase as in the luteal phase. In spite of this, plasma insulin levels remained stable throughout the menstrual cycle. DePirro makes the statement that these variations suggest that women should not participate in carbohydrate metabolism studies unless this phenomenon is considered.

Other avenues of investigation are the effects of aging on PMS, with symptoms generally worsening until the advent of menopause. The lack of biological support for PMS has been cited as evidence of the psychosocial nature of the complaint. However, attempts to link PMS with psychiatric disorders seem appropriate only for certain subtypes of PMS, falling short of a universal explanation of the syndrome.

In the analysis of psychosocial factors, reports have been dichotomous to the extreme. One study found increased rates of PMS in women who were typified as dependent, unassertive, and submissive to the traditional female role, while another stated that it is the type A female—the assertive, career-oriented "superwoman"—who is the usual PMS patient. Increased frequency of PMS has been noted among members of some religious groups.[19] Friedman and Jaffe[20] found only the factors of education and profession significant. Another study reported that an absolute absence of PMS was related to severe personality disturbance.[19]

A reasonable hypothesis could be that neuro-endocrinologic disease somehow facilitates the expression of an underlying mood disorder. This has already been seen in the association of major affective disorders and such conditions as hypothyroidism, Addison's disease, pheochromocytoma, etc.

Stress is generally believed to worsen the symptoms of PMS. This premise is supported by the reported alleviation of PMS through stress reduction techniques. This may indicate only that PMS can be added to the list of chronic conditions that are exacerbated by stress, such as asthma, seizures, allergies, migraine, thyroid dysfunction, and dermatitis (urticaria, pruritus, acne).

Current DSM-III-R texts list "late luteal phase dysphoric disorder" as the appropriate diagnostic appellation for the psychological manifestations of PMS. Some practitioners feel that PMS is a subclinical form of manic-depressive illness, with the patient aware of the symptoms only during the luteal phase. Another view is that PMS resembles a rapidly cycling manic-depressive disorder exacerbated by hormonal fluctuations. The similarities to manic-depressive disorder are consistent with the observation that premenstrual dysphoria is often followed by a euphoria with the onset of menses.

Increased psychiatric hospitalization and criminal activity have been documented during the paramenstruum (the four days preceding and the first four days of menstruation). Keye et al[21] reported increased rates of major psychologic, medical, and marital problems in women who believed themselves to have PMS and were seeking treatment for it. A history of substance, child, and spouse abuse, as well as increased suicidal potential, have also been correlated with PMS.[21]

Rubinow and Schmidt[22] addressed mood disorders and the menstrual cycle in 1986. They proposed four possible interactions of psychiatric disorders and the menses. These included disturbed menstruation resulting in psychiatric morbidity, psychiatric illness disrupting menstrual function, menstruation exacerbating pre-existing psychopathology, and menstrual events causing de novo mood and behavior disorders.

The above covers "the chicken versus the egg" dilemma regarding a psychiatric etiology of PMS, but it is also interesting to note at this point the findings of a study by DeJong et al.[23] They analyzed the data from a study that indicated a high rate of psychiatric illness among patients who believed they suffered from PMS. With prospective, longitudinal daily ratings, they found a high rate of misdiagnosis of PMS, with 88% psychiatric illness in PMS-negative subjects and only 45% in PMS-positive subjects after adjustment for accurate diagnosis. Their research also points out the need for personality testing during both the follicular and luteal phases.

Diagnosis. More than 150 symptoms associated with PMS that reflect changes in mood, fluid metabolism, and physical discomfort have been listed in the literature. Accurate diagnosis hinges on a careful history of the timing and variety of symptoms. Sources of stress, education, and profession are particularly pertinent, as is any history of chronic medical or psychiatric problems. It is also appropriate to inquire as to physical or sexual abuse. The physical examination is usually normal, but the practitioner should be attentive to any sign of disease as listed in the differential diagnosis (PMS, Table II).

Laboratory testing appropriate for all patients would include a complete blood count, sedimentation rate, chemistry panel, and thyroid function tests. Of a more discretionary nature would be hormonal testing of serum progesterone, prolactin, follicle stimulating hormone

(FSH)/luteinizing hormone (LH), and estradiol. These studies should be obtained during the luteal phase if they are to be of any benefit. A very thorough evaluation would include personality testing by a qualified psychologist. The Minnesota Multiphasic Personality Test, for example, is often helpful.

Very recently, a pharmaceutical firm in Wisconsin has begun marketing the Portman PMS Serum test. It claims that measurement of LH antibody reveals significant correlation between the symptoms of PMS and low serum LH antibody levels.[24] Validity of this method has yet to be established based on reproducibility by independent laboratories.

Once the initial evalution of a patient is completed, it can be helpful to define recognized subtypes of PMS in each patient, such as the previously discussed classification by Abraham.[8] Other considerations would include periodicity:

- *Interval.* Some patients will report regular monthly episodes, while others have alternating "good" and "bad" months.
- *Duration.* The classic presentation of symptoms seven to ten days prior to the period may be seen, as well as bimonthly exacerbations (at ovulation and seven to ten days prior to the period). Fewer patients complain of continuous symptoms from ovulation through the onset of menstrual flow.
- *Peak Incidence.* Two recognized peaks of PMS in a woman's lifetime are found during the mid to late 30s, and for a few years prior to menopause.

Treatment. Until the pathophysiology of a condition is identified, it is difficult to assess therapeutic success. Since this continues to be the case with PMS, most practitioners are hesitant to recommend more than a "natural" approach to PMS. Shangold[25] has presented an approach to therapy based on the severity of symptoms and mental health of the patient. In the normal female with mild PMS, no medications are needed. Treatment should consist of a program of support, and life style and diet changes. In the normal female with incapacitating PMS, treatment should be specific to the dominating symptom or symptoms. In the female with PMS that exacerbates psychopathology, support should be enhanced with psychotherapy. Women with psychopathology not related to the menses should get the help they need from psychiatric consultation.[25]

Most women are reassured once an educational and supportive dialogue is initiated. Some changes in diet and exercise do ameliorate the symptoms. These general recommendations include elimination of caffeine, alcohol, and other stimulants or mood altering substances; avoidance of simple carbohydrates; increased intake of protein and complex carbohydrates; limited salt intake; vitamin supplementation; exercise 2–3 times per week; and stress reduction.

The nutritional hypothesis has been supported by studies finding higher rates of vitamin deficiencies and altered carbohydrate metabolism among women with PMS. Vitamin B6, when taken as a supplement, can relieve some symptoms, especially headache and breast tenderness.[26] The dosage of B6 can range from 50 mg/day to 800 mg/day, but since there is a possibility of toxicity with doses beyond 100 mg, higher dose regimens should include regular checks for signs of toxicity. It has been hypothesized that vitamin B6 is effective as a result of its action to increase hepatic clearance of estrogen. It may also have a direct action on the brain, being a coenzyme in dopamine/serotonin biosynthesis. There can be a decrease in prolactin inhibiting factor with relative B6 deficiency, resulting in hyperprolactinemia. London et al[27] found that symptoms of anxiety, headache, cravings for sweets, and depression improved on 300 IU of vitamin E per day. Other researchers have found similar results with a therapeutic dose range of 200–600 IU/day. Other vitamins, micronutrients, and trace elements under study are magnesium, zinc, and iron. Evening primrose oil (Etanol) has some anecdotal supporters. However, as with any herbal remedy, it is very difficult to ascertain the active substance responsible for its success. The recommended dosage of evening primrose oil is usually 0.5 grams twice a day from ovulation to onset of menses.

Since the symptoms of PMS are worsened with stress, stress reduction is encouraged through a variety of methods, such as relaxation exercises, meditation, biofeedback, yoga, guided imagery, creative visualization, and psychotherapy. Exercise goes hand in hand with stress reduction. Ongoing, well-designed, prospective studies on the effects of exercise on PMS may add to the increasing knowledge about menstrual disorders and exercise (anovulation, dysmenorrhea, amenorrhea, oligomenorrhea, luteal phase shortening, etc).[28]

Most patients respond to the general recommendations listed above. For the minority of patients with severe symptoms, treatment with pharmacologic agents may be considered. In 1931, Frank[5] recommended the use of cathartics to eliminate excess hormones through the bowel. Lithium has been tried but with no greater benefit than placebo. Some physicians have gone so far as performing total abdominal hysterectomy with bilateral salpingo-oophorectomy, followed with hormone replacement therapy. Even with these extremes of treatment, generally considered inappropriate, there remains a long list of other approaches being reviewed. These are described in Table IV.[29–45] The diversity of pharmacologic modalities attests to the lack of any single reliable medication. Most of these therapies, at best, provide some relief of symptoms.

ENDOMETRIOSIS

Endometriosis, an often overlooked diagnosis, should always be considered in the patient with dysmenorrhea. Its manifestations are extensive, the most frequent being pelvic pain and infertility. Severity of symptoms does not always correlate with the severity of disease. Further confounding the diagnosis is that definitive diagnosis can only be made by direct examination of tissues seen or biopsied at the time of an invasive procedure, usually laparoscopy.

Pathophysiology. The reason deposits of functioning endometrium develop outside the uterus in some women has yet to be determined. It is estimated that 10–15% of all premenopausal women have some degree of endometriosis. Sampson's[46] theory of retrograde menstruation is the most widely held.

TABLE IV. Therapies in Use or in Research for Premenstrual Syndrome

Category	Medication	Dosage	Target Symptoms
Hormonal	Progesterone	Suppositories, 50–400 mg/four times a day or twice a day for 5–14 days ptp* (max, 2,000 mg)[29] IM natural progesterone (progesterone in oil) 50–200 mg given for 7–10 days ptp*[30] Oral micronized progesterone, 10–200 mg daily or twice a day for 5–14 days ptp*[31] Sublingual 50–100 mg/day for 5–14 days ptp*[32]	Mood changes,† fluid retention
	Oral contraceptives	Better response with high progestin type for PMT-A; estrogen dominated oral contraceptive for PMT-D[8,33]	Mood changes†
	Estradiol	Subcutaneous implants with cyclic progestin orally[34]	
Antihormonal	GnRH agonist LHRH agonist	Experimental[35]	All PMS symptoms
	Danazol	50–200 mg/day to three times a day[36]	Fluid retention,‡ mood changes,† appetite changes, breast tenderness
Diuretics	Metolazone	5 mg/day orally for 7 days ptp*[37]	Fluid retention,‡ mood changes†
	Spironolactone	50–150 mg/day for 5–10 days ptp*[37]	
	Thiazides	Per product recommended dose	
	Pamabrom	In over the counter medications	
Antihistamine	Pyrilamine	In over the counter medications	Mood changes†
Antiprostaglandins	Naproxen	500 mg orally twice a day for 10 days ptp*	Fluid retention,‡ mood changes,† pain
	Mefenamic acid	250 mg orally, every 6 hr for maximum 7 days[39]	
	Ibuprofen	200–800 mg orally, every 6 hr for 10–14 days	
Dopamine antagonist	Bromocriptine	1.25–2.5 mg twice a day w/meals for 7 days ptp* (max 7.5 mg/day)[15]	Breast tenderness, mood changes†
Opiate antagonist	Naltrexone HCL	25 mg twice a day, orally[14]	Mood changes,† concentration
Antidepressants	Tricyclics Monoamine oxidase inhibitors	Experimental[40]	Mood changes†
Tranquilizers	Alprazolam	0.25–3.0 mg/day as needed ptp*[41]	Mood changes,† insomnia
Anxiolytics	Buspirone	5–10 mg three times a day; effective at 2–4 wks of prescription[42]	Mood changes†
Alpha adrenergic stimulant	Clonidine oral	0.1–0.3 mg/day for 7–10 days ptp*[43]	Abdominal cramps, concentration, mood changes†
	Transdermal	0.1 mg/day for 7–10 days ptp*	
Central nervous system stimulant	Dextroamphetamine	Experimental 5–30 mg/d for 7 days ptp*	Depression, fatigue
Alpha adrenergic antagonist	Ergotamine	2 mg orally to start, w/1 mg orally as needed (max, 10 mg/wk)	Migraine headache
Combination medication	Bellergal	One tablet orally twice a day as needed	Mood changes,† headache
Beta blocker	Propranolol	10–80 mg orally 1–4 times a day as needed 80–120 mg (long acting) orally, daily, as needed	Headache
Angiotensin-converting enzyme inhibitor	Verapamil	80 mg orally, every 6–8 hr to 120 mg (sustained release)[44]	Mood changes†
Antibiotics	Doxycycline	100 mg twice a day, orally for one entire menstrual cycle[45]	Mood changes,† fluid retention
Thyroid supplement	Levothyroxine	Low dose-adjusted dose orally every day	Fatigue, depression

* ptp = prior to period.

† Mood changes = anxiety, irritability, tension, depression.

‡ Fluid retention = swelling, weight gain, abdominal bloating, breast tenderness.

Endometrial implants are most commonly found along the uterosacral ligaments, the cul-de-sac, and the ovaries. Commonly enough, these implants not only persist, but continue to function on a cyclic basis. Dissemination to distant sites, such as the lung, is also yet to be explained, but hematogenous or lymphatic spread has been proposed. Perhaps the answers relate to excessive prostaglandin production, an immune disorder, or hormonal abnormality.

Symptoms. The typical complaint of those suffering from endometriosis is a deep, constant ache that often precedes, accompanies, and outlasts the menses. A survey by the Endometriosis Association reported 57% of endometriosis sufferers having continual pain.[47] Other pain complaints include dyspareunia, dysuria, painful defecation, and low backache. These are believed to be caused by inflammation, adhesions, scarring, and loss of normal structural elasticity.

Diagnosis. There is often a history of severe dysmenorrhea, infertility, miscarriage, ectopic pregnancy, and use of an intra-uterine device. Up to 10% of women with endometriosis have a mother or sister with the same problem.

Endometriosis is most common in childless women in their 30s to 40s. The Endometriosis Association has noted in surveys that 41% of patients with endometriosis had some symptoms before age 20.[47] There is also a possible association with previous abdominal surgery, especially with laparotomy, laparoscopy, episiotomy, amniocentesis, and cervical cryosurgery or conization.

On physical examination, there is an increased frequency of retroversion (up to 42% of cases) of the uterus. There can also be decreased uterine mobility with tenderness, nodularity, or thickening of the pelvic organs, particularly the uterine ligaments and cul-de-sac. But physical examination is far from diagnostic, in that severe cases of endometriosis have been found with almost no abnormalities noted on manual examination.

Laboratory findings are not conclusive. There can be an elevated white blood cell count and sedimentation rate, but this can occur with almost any inflammatory disorder. Some patients present with fatigue as their prime complaint, with negative studies for other causes of fatigue such as anemia, diabetes, or thyroid disorder. Radiography can at times point toward a diagnosis of endometriosis, but not without radiation of the ovaries (ie, CT scan) or an invasive procedure (hysterosalpingogram). Ultrasound can show distortion of normal anatomy and some endometriomas, which give clues to the diagnosis.

The only truly diagnostic procedure available now is exploratory surgery. The culdoscope has been used in the past to diagnose endometriosis, but its limited field makes laparoscopy or laparotomy the better choices. Laparotomy, while affording the surgeon a larger field for examination, entails more surgical and recovery time. Laparoscopy has become the more common approach, especially with the development of the videolaseroscope that not only permits diagnosis and documentation of the extent of the disease, but also allows for the vaporization of endometrial implants and the lysis of adhesions.

Treatment. As with any illness, education and support are the foundation of the treatment. Patients with severe cases must participate in difficult treatment decisions, as well as adopt major life style changes. Anti-inflammatory medications can be used for initial relief of discomfort. Patients can also be counseled to not delay pregnancies and to breastfeed if successful. Cramer et al[48] have documented decreased symptoms with increased exercise and heavy smoking. While the former can be a routine recommendation, of course the latter can never be encouraged.

Many different treatment regimens are described in the literature, such as low-dose testosterone, depot progesterone, and continuous oral contraceptives. Antihormonal treatment is another approach to therapy, with danazol the most commonly used medication. Its therapeutic use is enhanced as an adjunct to laser vaporization. As discussed previously, laser laparascopy provides treatment with reasonably successful pregnancy rates after surgery.

Hormonal treatment is based on the rationale that the disorder is a result of endometrial stimulation, so that by preventing the LH surge at midcycle, ovulation is inhibited, and stimulation of the endometrium is decreased. Lemay[49] used the luteinizing hormone-releasing factor (LHRH) agonist, buserelin, to produce a reversible hypogonadotropic hypogonadism, thus decreasing symptoms and decreasing the size of endometrial implants after six to nine months of use. Then the temporary menopause was discontinued, with all subjects resuming normal menses within six months of medication cessation. Buserelin is destroyed by digestive enzymes and must be administered either subcutaneously or intranasally. Studies are underway at Johns Hopkins using leuprolide, a synthetic gonadotropin-releasing hormone (GnRH) agonist, as a nonsurgical approach to treatment of endometriosis.[49] Leuprolide may suppress ovarian function as well as, if not better than, danazol, with fewer side effects. Similar to leuprolide, nafarelin may be another GnRH agonist effecting nonsurgical treatment of endometriosis, but its use has yet to be approved by the FDA.[50,51]

ADENOMYOSIS

Even less well known than endometriosis, adenomyosis is a condition in which endometrial tissue invades the myometrium. Its symptoms mimic endometriosis and uterine leiomyomata, with pelvic pain and menometrorrhagia. Of help in diagnosis is the fact that this abnormality can at times be seen with ultrasonic examination of the uterus. Initial treatment usually consists of symptomatic relief. NSAIDS are helpful, particularly mefenamic acid and naproxen. These also have the occasional benefit of decreasing menstrual flow and frequency of passed clots. Cyclic progestins can be used to improve the dysfunctional flow. Some cases ultimately become so severe as to require hysterectomy.

CHRONIC PELVIC PAIN

Important causes of pelvic pain include endometriosis, chronic pelvic inflammatory disease, and pelvic adhesions. A few women will have chronic pelvic pain for which no cause can be found. Chronic as opposed to acute pain in general is more difficult to diagnose and document, much less to treat medically. Complaints can be vague, intermittent, and, at times, contradictory to scientific knowledge. The condition is usually not life-threatening. But chronic pain is just that, a prolonged course of noxious and worrisome stimuli often never remedied, resulting in mutual patient and physician dissatisfaction.

With chronic pelvic pain, symptoms may change with the menses; even if some pathologic process is found, the extent of the pathology does not always account for the degree and variety of symptoms. More attention is paid to patients with some identifiable pathologic process, and less to patients lacking obvious disease, but both suffer similarly.

There is also the problem of differentiating nongynecologic from gynecologic disorders, the former including degenerative joint disease, cystitis, urethral syndrome, diverticulitis, irritable bowel, and Crohn's disease.

Many patients find it difficult to accept the physician's reassurance that the pain is not a harbinger of serious illness. They are frustrated by the continuing pain and by what they perceive as lack of concern for their dilemma. The effects of prolonged, chronic, unexplained pain on the psyche are depression, withdrawal, guilt, and loss of self-esteem. For many women, this brings the first realization of human frailty, that illness does not always happen to someone else, that fertility is not everyone's gift, and that being female has not been cursed by some women without reason.

The depression reinforces the pain, and the pain reinforces the depression. Even with the removal of the stressors and the pain that may have led to this state, mental health professionals will admit that this alone will not cure depression. Once depression is established, it can have, in a sense, a life of its own. Practitioners who treat women with chronic pelvic pain should recognize that almost all patients will go through an episode of depression as the natural course of the ailment. Factors affecting the length and severity of depression include the mental state of the patient, what pain means to that individual, and other social constructs and learned behavior that would contribute to depression.

Treatment. When the underlying cause has been addressed, education and support start the therapeutic process. It is important to realize that alleviation of symptoms, not total cure, is the standard of success. Explanations should be offered regarding the source of the pain, and options for treatment proposed. This enables the patient to develop insight and gain some feeling of control. Besides medications and procedures, a comprehensive regimen would also provide attention to depression, psychiatric disorders (ie, manic-depressive illness), drug abuse, and history of sexual abuse, with referral to mental health specialists as needed.

CONCLUSIONS

This review focuses on a number of gynecologic complaints that share more than just similarities of symptomatology. While rarely life threatening, they can severely diminish a woman's quality of life. The alleviation of dysmenorrhea through anti-inflammatory drugs and oral contraceptives has been a welcome advance in well-being for thousands, if not millions, of women. It may also be a reason why many women expect there to be just as simple a remedy for premenstrual syndrome, endometriosis, and other complaints. In the meantime, practitioners of women's health care can provide more education and support as a routine of medical care. Treatment options can be discussed and individualized. Learning to accept, cope, and go on with illness is not an innate skill for all, but certainly can be attained with short term counselling for most patients. Until cures are available, patients need support and guidance not just for the illness but for the experience and impact of it on their lives.

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Women in medicine since Flexner

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For many, the Flexner report[1] remains a major explanation for the decrease in the numbers of women doctors in the early decades of the 20th century. For example, Kenneth Ludmerer's[2] highly acclaimed history of American medical education includes this comment: "Of all the oppressed groups, women suffered the most ironic setback. In the wake of the Flexner report, all but one women's medical college closed." Since a number of other scholars have also viewed the Flexner report as a crucial point in the history of women physicians, it seems useful to ask why so many writers have accepted the Flexner myth.[3-7]

In reality, the evidence is quite clear: the Flexner report did not itself cause the women's medical colleges to close nor did it cause a decline in the number of women physicians. Both changes were well underway before Flexner began his study.* In 1909, for example, the year before the publication of the Flexner report, 14 of the 17 women's medical colleges had already closed and the total number of women medical students, including those attending coeducational schools, had dropped to 921 from a total of 1,419 15 years earlier.[8,9]

Perhaps this misconception exists because scholars are unable to find another explanation for what happened in the early 20th century to turn back the tide for women physicians. As documented elsewhere, the late 19th century in America was a relative golden age for women physicians. Compared to some other countries, relatively large numbers of women gained access to medical schools in this time period, making the United States a leader in advancing the cause of women doctors. As it turned out, in spite of these initial gains, institutional barriers remained remarkably resilient in the first half of the 20th century and they became the central force in limiting the number of women who entered medicine. As a result, there were fewer women physicians in Boston in 1950 than there had been in 1890 and this pattern was repeated in cities across the country.

Sexism is not a simple phenomenon, however, and researchers have found it extremely difficult to tease out the causes as well as the solutions to the problems affecting women in American medicine. Some scholars still ignore the problems of sexism and minimize its importance, somehow assuming that because they do not acknowledge it, it is not an important issue. William Rothstein,[10] for example, has only two brief references to women in his 1987 history of American medical schools and the practice of medicine. Rothstein claims that medical education for women developed slowly in the 19th century and that there was an absence of overt sex discrimination in the period from 1950 to 1970, a remarkable assertion which is inconsistent with what we have learned about the topic in recent years.[10]

Unlike the period of the early 1970s, a growing literature on sex discrimination law now exists. Social and behavioral scientists have also demonstrated the extent to which sex bias determines both our perceptions and our behavior. The complex connection between patterns of sex discrimination and the determination of social policy is highlighted in a recent paper by Faye Crosby[11] entitled "Sex discrimination: How can we correct it if we can't see it? And how can we see it if we're not prepared to correct it?"

EARLY BARRIERS TO WOMEN IN MEDICINE

The evidence gathered by this writer on sex discrimination in the medical profession, from a variety of sources and spanning more than 150 years of history, suggests that from the very beginning, the unwillingness to correct patterns of sex discrimination prevented some of the gate-

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* Evidence for historical arguments in this paper, unless noted otherwise, is presented in detail in the text and footnotes in Walsh.[8,9]

keepers in medicine from seeing it in the first place. Certainly this was not true of all physicians and the problem was not limited to medicine or to men in American society. On the other hand, medical men were in a position to open the gates to women and their obduracy became the first barrier. Originally many men could not even conceive of women as physicians. Walter Channing, a professor of obstetrics at Harvard Medical School writing in the 1820s, claimed that it would be impossible for women to submit to the rigors of medical training. He reasoned that some of man's delicate feelings and much of a man's "refined sensibility must be subdued" in the practice of medicine but the female psyche could never stand up to such a demanding career. Womanliness would be completely destroyed. As Channing and most male physicians saw it, the female personality was entirely distinct from that of the male and this rhetoric reflects what historians have referred to as the "cult of true womanhood."

But the subject to which every medical opponent of women was irresistibly drawn was the subject of the female reproductive system. "It was," one physician exclaimed, "as if the Almighty, in creating the female sex, had taken the uterus and built up a woman around it." Horatio Storer, a gynecologist on the faculty of the Harvard Medical School in the 1860s, saw himself in direct competition with women surgeons. To Storer, a woman was defined "in health, in character, in her charms, ...mind and soul because of her womb alone." Who could trust the great questions of life or death to one whose equilibrium varied from "month to month and week to week...up and down"? Storer claimed that menstruation led to "periodical infirmity...mental influences...temporary insanity" and it was a force to be reckoned with. In his opinion, women were crippled because of their biology, certainly more in need of medical aid than able to furnish it. Still, despite this opposition, women made slow but steady progress as the 19th century progressed. They also began to invade the specialties of surgery and obstetrics.

One of the outstanding obstetrician-gynecologists of this early period was Dr Marie Zakrzewska, who founded the New England Hospital for Women and Children in 1862. After observing the statues of famous men in Westminster Abbey while on a trip to Europe in 1881, she wrote:

> Will there ever be a monument to the first woman physician?...We need such landmarks of civilization...because the now-living, as well as those who will live long afterward, need encouragement...The person who is covered by a monument is of no consequence, but the fact that a woman can work and make an impression upon civilization needs to be known to be remembered.[12]

When I began my research on women physicians 18 years ago, I found that Zakrzewska's hoped-for monuments had not materialized. Even worse, much of the early history on women physicians had been distorted. Instead of descriptions of the incredible barriers confronting women physicians, a "blaming the victim" approach was used and women were depicted as being unable to meet the increased professional requirements for training emphasized in the Flexner report.[1] In fact, as I have argued in my book[8] and elsewhere, women physicians welcomed the professionalization process with its carefully delineated medical prerequisites which spelled out in detail the requirements for being a doctor.

Harriot Hunt, the first woman physician to practice in the United States, illustrates this point very poignantly. Hunt and her sister, Sarah, completed their medical apprenticeship with a husband and wife medical team and then began their practice as "female physicians" in Boston in 1835. Sarah Hunt practiced only a few years. Harriot described in her autobiography the problems which women physicians encountered when they were shut out of medical schools and deprived of access to formal training. The fact that Elizabeth Blackwell is usually credited with being America's first woman doctor reflects a historical double standard. Blackwell's status results from having been the first woman to have received a medical degree, in 1848, a standard which, if applied to her male colleagues, would have sharply reduced the number of male doctors in the country.

In New England, for example, in the period 1790 to 1840, in one of the few counties where we have demographic data on physicians, most male practitioners obtained their training by apprenticeship. In Worcester County, 73% of the physicians had not graduated from college and still, as men, they could set up a medical practice. Women who might have been interested in medicine in this preprofessional period were subject to numerous unwritten laws and customs which limited their visibility and obviously their practice of medicine. Even the apprenticeship experience was not equally open to women applicants, depending as it did on informal arrangements, usually with a male physician. Besides Harriot and Sarah Hunt, there is little evidence that other women were able to become physicians through the apprenticeship route, though this was the norm for male physicians.

THE BENEFITS OF PROFESSIONALIZATION

At this point, it is important to ask the questions: Did professionalization offer women their first real opportunity in medicine? The answer to this seems to be yes. If a medical degree were necessary, interested women could apply to medical colleges and if rebuffed, they could then establish their own. If medical society membership or a license were necessary, women could rise to meet the requirements and, if rejected, could raise the cry of injustice to gain support. In other words, it was easier to overcome known obstacles than to tilt at shadowy specters.

On such known obstacle was the medical licensing examination. Women physicians regularly boasted how well they did on these tests. Official examination results categorized by sex do not exist, but women physicians, particularly those who attended women's medical colleges, were vigilant in this matter and occasionally compiled their own statistics. One study, published in the *Transactions of the Woman's Medical College of Pennsylvania*, shows that only 4.9% of the graduates of the allopathic Pennsylvania and Baltimore schools and New York (homeopathic) school, which were the three remaining women's medical colleges, failed between 1901 and 1903. During the same period, 16.3% of the total graduates of the other medical colleges in the country failed the examinations.

Women also made great strides in gaining access to established male medical schools in the closing decades of the 19th century. By the early 1890s, some 40 medical colleges across the country, almost half of which were regular medical schools, had female enrollments of 10% or more. In a few cases, women accounted for more than half of the graduating classes. The long struggle to gain access to the leading medical schools seemed assured when an enterprising group of women in Baltimore organized a campaign to endow Johns Hopkins University's long delayed medical school on the condition that women be admitted on the same terms as men. Funds were raised in several cities and with the offer of a $500,000 endowment, the financially beleaguered Johns Hopkins surrendered to the women's terms.

THE EFFECTS OF THE DISAPPEARANCE OF WOMEN'S MEDICAL COLLEGES

Successes such as this contributed to the disappearance of the women's medical colleges because they encouraged the belief that separate islands of feminism were no longer needed. As Emily Blackwell noted when the New York Infirmary Medical School was absorbed by Cornell University Medical College in 1899: "It has now fulfilled its purpose and medical education may hereafter be obtained by women in New York in the same classes, under the same faculty, with the same clinical opportunities as men."

In some ways, events at the Cornell Medical College were a microcosm of what was happening to women physicians in the years surrounding the publication of the Flexner report.[1] Seventy women from the New York Infirmary entered Cornell in 1900 after it had absorbed the women's college. By 1903, the number of women students at Cornell had been reduced to ten. One way Cornell managed to cut the numbers of women was to create new obstacles for them. For example, the women students, unlike the men, were required to take their first two years of medical education at the Ithaca campus in upstate New York, some 250 miles from New York City. In response to a 1917 survey of medical education, the dean of Cornell's medical school admitted that the policy was deliberately adopted at the time to reduce the number of women medical students.[13]

Equally important, the absorption of the New York Infirmary cut short the teaching careers of a number of able young women faculty. Twelve years after the disappearance of the woman's medical school, Cornell had yet to appoint a single woman to its major teaching staff.

We know relatively little about the teaching faculty, either men or women, of most medical schools except in those few cases where individual biographies have been written. Recent accounts of medical education, for example, provide only the sketchiest of details about faculty characteristics. The reasons that research related to women faculty has been neglected are complex. Sexism and prejudice often made women invisible, and their teaching appointments were apt to be informal. Even a prominent woman like Florence Sabin, a well-known professor at Johns Hopkins Medical School in 1911, was not eligible to vote on policy at the medical school. When women were employed in off-track appointments, in "acting" positions, and as associates or lecturers, instead of in regular faculty slots, they were even less likely to appear in catalogues and medical school brochures because schools might not want to establish a formal precedent by making this information public.

Most 19th century medical women and their friends viewed separate women's schools as a temporary expedient while they worked toward the long term goal of gaining access to the coeducational medical schools. Nevertheless, the disappearance of women's medical schools has to be considered in any discussion of why the numbers of women doctors declined in the early decades of this century. To take just one individual case, the Woman's Medical College of Pennsylvania graduated fewer than 50 women doctors each year in the period from 1920 to 1965. Yet this number was four or five times the number graduating from most other medical schools. Even worse, some medical schools graduated no women at all or just one or two for many successive years during these decades.

Moreover, a separate women's medical college was not just a matter of the number of graduates. The psychological significance of these schools is underscored by Dr Alice Weld Tallant in an address in 1912 at the Woman's Medical College of Pennsylvania, the longest surviving female medical school:

> "Until I took my internship," she recalled, "I had never seen a woman operate, and I do not think those of you who have had your training in this [woman's medical] school can realize what it means to have never seen a woman doing that which to you seems second nature from your student days. It must be a great incentive to the student," she continued, "to see what women can do; it is almost inevitable, if you have never seen a woman doing anything, to think she cannot do it quite as well as a man, no matter how strongly you feel in favor of women."[14]

TWENTIETH CENTURY OBSTACLES

Although it is hard to measure, the major problem in this historical period is that relatively few people, except for women physicians themselves, really valued the professional advancement of women in medicine. The first barriers that many medical women faced were within their own families. For example, the mother of Dr Elizabeth Mosher, who attended the University of Michigan in the 1870s, claimed that when her daughter first broached the subject of going to medical school she said she would sooner see her shut up in a lunatic asylum. Dr Dorothy Reed Mendenhall, who entered Johns Hopkins in 1900, later recalled that in all the years she was pursuing her degree in Baltimore, her aunt told her friends that Dorothy had gone South for the winter.[13(p100)]

Within the medical establishment, many opposed the advancement of women, some tolerated it, but few members of the medical establishment advocated the cause with enthusiasm. Most male physicians simply reflected the prejudices of the larger society. Consequently, the examples of the few men who did support women physicians and worked on their behalf stand out in the historical record. During the late 19th century, for example, women physicians were especially fortunate in attracting to their cause three of the most prominent physicians in Boston: Henry I. Bowditch, Samuel Cabot, and James Chadwick.

As Martin Duberman[15] has noted, we know far too little about why people do anything, let alone why they do something as specific as joining a reform movement. These three men differed in their areas of medical specialization and their ages, ranging from Bowditch, who was 71, to Chadwick, who was thirty-five years younger. The one thing that all three men seem to have shared was a combination of personal and public success which enabled them to bear the tension connected with supporting a highly unpopular cause. Each man appears to fit Gordon Allport's definition of the mature personality: the ability to get outside one's self-preoccupation and involve oneself in abstract ideals. For Bowditch, who championed the first female applicant to the Massachusetts Medical Society, the women's struggle was an extension of the principles of the abolitionist movement which he had joined in its infancy. Samuel Cabot had longstanding friendships with a number of women doctors and actively supported the New England Hospital for Women and Children. James Chadwick took the women's cause as his own and he not only carefully kept a scrapbook of clippings and letters to document their progress but he actively worked to gain allies for them.

It is clear that medical leaders, except for a few exceptions such as these, failed to supply what one would hope from them on this issue—that is, leadership. Though one can argue that they were merely men of their times, they clearly lacked the ability to appreciate the difficulties that women faced. Even the medical reformers were blind to the problems of women in medicine. For example, Abraham Flexner, in 1910, casually dismissed the difficulties of women by noting that their choices in medical education were "free and varied."[1(pp178–179)] Flexner's visits to 155 medical schools in the United States and Canada, completed in a little more than a year, would challenge even a modern day traveler who had access to planes and automobiles. In the first decade of this century, with poor roads and difficult travel conditions, Flexner must have had very fleeting site visits at any one school. Considering how difficult it has been in the recent decades to investigate sex discrimination and its impact on women's opportunities in professional education, it is understandable why he could not tap this phenomenon in the schools that he visited. The information that he gathered on women's medical opportunities were most likely based on anecdotes and comments from male physicians who were not particularly well informed about the realities women were experiencing. The aggregate statistics we have on medical school enrollments indicate that women were not as freely admitted to schools as Flexner argued. We also have considerable archival information on the obstacles women encountered once they did matriculate as students.

When Flexner published his report in 1910, for example, more than half of the medical schools in the country did not accept women students. Although some schools did open their doors to women for the first time in the years immediately following the Flexner report, those decisions did not arise from a real commitment to the equal education of women. Rather, they often reflected some combination of local events that tipped the balance. For example, Yale's decision to admit women for the first time in 1916 seems to have been stimulated by Henry Farnam, a Yale economics professor who had a daughter who wanted to enter medical school. When Farnam found out that the major obstacle to the admission of women was the $1,000 necessary to construct a separate women's bathroom, he offered to pay the bill himself. Yale accepted, the bathroom was built; and Louise Farnam went to medical school. It was a room that women students in the 1920s referred to as "the Louise Farnam memorial." The Farnam family got off cheaply. As previously noted, it cost women $500,000 in the 1890s to convince Johns Hopkins to open its doors to women.[16]

It should also be pointed out that those schools that did open their doors to women did not open them very wide. Northwestern University Medical School admitted women in 1926 as a result of Mrs Montgomery Ward's casual inquiry, after her gift to Northwestern's endowment fund, as to whether the school admitted women. Unwilling to take any chances on Mrs Ward's possible feminist sympathies, the university quickly decided to admit women and was rewarded handsomely when she doubled her original gift. But the school limited the number of women to four in each class. The university justified the number, which remained in effect with few exceptions until the 1960s, with the explanation that four was the number necessary for a complete dissecting team.

Those interested in the medical education of women faced a new challenge in the 20th century: the growing emphasis on postdoctoral medical training. But it was not these rising standards, per se, that threatened women. The problem lay in the fact that there were so few internships open to women. According to the 1921 *AMA Directory*, only 40 of the 482 general hospitals approved for internships in the country admitted women interns. In other words, 92% of the available hospitals did not train women, however excellent a woman's medical school record. As late as the mid-1930s, 12 states had no hospitals available for women interns. In 12 other states, there was only one hospital where women medical school graduates could go for training.

All of this meant that women had to limit their internship applications to a handful of hospitals. In 1925, for example, 50% of all women interns trained in nine widely separated hospitals, most of which were owned and operated by other women. One of these was located 5,000 miles away, in the Philippines! The result of this limitation was that women were confronted with a "systems effect." Women's inability to gain internships could be (and often was) used as a reason for not admitting them to medical schools. Opponents of the women argued: what profit was there in educating women doctors if they could not complete their critical postgraduate training?

The existence of quota systems in many medical schools also had a chilling effect on women's applications. Before the 1970s, most medical school officials publicly denied the existence of discrimination but when guaranteed anonymity, they occasionally confirmed that female quotas existed. In 1946, the dean of one large eastern medical school acknowledged limiting the enrollment of women to 6%. As recently as 1961, another dean boasted of a similar practice: "Hell, yes, we have a quota," he said. "Yes, it's a small one. We do keep women out when we can. We don't want them here—and they don't want them elsewhere, either, whether or not they'll admit it." Another medical school spokesman remarked proudly, "Yes, indeed, we take women, and we do not want the one woman we take to be lonesome, so we take two per class."[17]

In fact, published information put out by the medical schools themselves is quite explicit about the existence of overt sex discrimination. For example, from 1959 to 1971 the Association of American Medical Colleges (AAMC) handbooks on medical school admissions requirements included a separate information section for each school entitled: "Medical School Preferences," which was designed to guide the prospective student. Each school's policy regarding its preferences for applicants in terms of the sex, race, residence, and age of applicants was described. Of the 88 coeducational medical schools listed in 1959, 28% stated that they preferred male students and they stated this fact in print. During the same period, excluding Howard and Meharry medical schools, only 10.5% of the schools admitted a racial preference. By 1963, 15% still expressed some bias against women applicants as opposed to 4.5% that admitted to a racial preference.[18] This does not mean that those schools that did not express sexual and racial preferences did not have them. It does demonstrate, however, that a significant number of medical schools during this period saw nothing wrong with expressing a preference for male candidates.

RECENT REFORMS

All this began to change in the 1970s with the rise of the feminist movement and the pressure of affirmative action. As a result, medical schools began to revise their discriminatory admissions policies, which led to an increase in the number of applications by women. Expectations of being accepted help to determine how many applicants apply to a particular school. Earlier, medical schools had helped discourage potential female applicants through their acceptance policies and subtle clues in recruitment and application procedures. When women perceived an improvement in their chances of being accepted, they increased their applications. This, in combination with federal guidelines requiring equal treatment, led to the dramatic increases of women students in the 1970s.

As a result, the blatant discrimination in recruitment and admissions procedures dramatically changed in the next few years. Schools began to openly welcome women. A University of Hawaii Medical School poster, for example, portrayed native Hawaiian, Asian, black and white women under the caption: "We're Going to be Doctors." The poster stated that "medicine is not for men only" and that the school "actively encourages women to apply."

Other examples include a special recruitment publication entitled "Women in Medicine," published by Stanford University Medical School depicting a pregnant woman medical student ministering to a patient. The brochure contained this paragraph: "We're not being excluded now, and we're not dropping out. But far fewer women than men have been encouraged to apply to medical schools, including Stanford. And this is what we hope to change. We can soon expect about 25 percent female M.D.'s but we need and want more."

Aside from admissions changes, a great deal of the effort to combat sexism in medical education has been conducted on an ad hoc basis. Harvard Medical School, for example, published a student task force report in 1974 which identified a long string of discrimination practices at the school such as role stereotyping, sexual innuendo, and ignoring female presence in a group situation.

Attacks were also launched against medical textbook authors who used pictures of *Playboy* bunnies and other titillating female models to liven up their presentations of scientific information. For example, Dr Estelle Ramey led a successful battle to force Williams and Wilkins to withdraw its textbook *The Anatomical Basis of Medical Practice*[20] from the market in 1972 because it contained pictures of nude females in seductive poses and statements such as: "We are sorry that we cannot make available the addresses of the young ladies who grace our pages. Our wives burned our little address books at our last barbecue get-together" and "If you think that once you have seen the backside of one female, you have seen them all, then you haven't sat in a sidewalk cafe in Italy where girl watching is a cultivated art. Your authors, whose zeal in this regard never flags, refer you to [the following illustrations] as proof that female backs can keep an interest in anatomy alive."[19,20]

PROBLEMS STILL REMAINING

It is now almost 20 years since the number of women admitted to medical schools began to rise rapidly. The sharpest increase took place in the 1970s. What has changed and what problems persist for the current crop of women medical students and physicians?

It is clear that the climate for women physicians has improved significantly in the past decade, but the reality is that women will remain a minority within the profession for a long time to come. If the current percentage of women medical students levels off at about 35%, it will take until the second decade of the 21st century for women to constitute 25% of practicing physicians. Still, women are no longer the anomalies in medicine they once were.

This is not to say that reservations about women in medicine have disappeared. In reviewing the recent literature I was surprised to see traces, albeit expressed in a somewhat more sophisticated and subtle way, of some of the issues that had "dogged the footsteps" of earlier medical women. One that I thought had been buried was the question of whether women were constitutionally capable of pursuing a medical career. Admittedly, I found no echo of Dr Horatio Storer's characterization in 1866 of menstruation as "temporary insanity." Nevertheless, I did find a surprising number of articles that raised questions about the mental and physical capabilities of women physicians. This is not the place to do an exegesis of the literature, but I would like to cite just a few examples of the phenomenon. In an article entitled "Suicide among U.S. women physicians," the authors analyzed factors causing a higher suicide rate among the women. On the basis of these statistics, they calculated that "about 65 percent of American women physicians have primary affective disorders." Dismissing "role strain" as a possible cause of suicide, they concluded that there was a connection between affective disorders and the decision of women to pursue a medical career.[21]

Although there is not space here to analyze this article carefully, interested readers are referred to the letters to the editor in the December 1979 issue of the *American*

Journal of Psychiatry.[22] I would like to point out, however, that when I looked at the original data, I found that there are almost no gender differences in the suicide rates, which were 40.7 per 100,000 for female physicians and 38.1 for male physicians. Perhaps like beauty, some gender differences are in the eye of the beholder.

Another of the old arguments against women surgeons was that they lacked the strength to do the job. The more sophisticated modern version of this point of view can be found in a recent issue of the journal *Surgery*. After noting that right-handed residents scored higher on a number of tests, the authors traced the superior performance of male surgery residents at Loyola University to spatial and motor proficiency which localized in the right hemisphere of the brain.[23] Happily, the paper elicited the following response from a surgeon who had several of the residents from Loyola under his supervision: "the best one we have seen yet is a woman who is right-handed [and] we have one left-handed woman for whom I predict stardom in academic surgery."[24] It is not clear if this qualifies as a more "even-handed" evaluation of women's surgical skills. What is encouraging is that articles like this have resulted in criticism from both male and female respondents. Women are either no longer alone in their criticisms of these kinds of studies or editors feel compelled to publish more diverse letters of response.

There are, however, any number of unresolved problems related to the increasing number of women entering medical schools and the practice of medicine. If one had to choose one issue that constitutes a major dilemma for the woman physician and at the same time symbolizes medicine's difficulty in coping with women-related issues, it would be the problems surrounding pregnancy and parenting. In 1894, Dr Gertrude Baille wrote in the *Women's Medical Journal* that the reason why many women physicians did not marry was because they know "No woman can serve two masters." She argued that when professional women had a family, there was inevitable conflict between the two roles, and "Either her work or her family will feel the neglect."[25]

Today, women physicians do marry. Moreover, about two thirds of practicing women physicians have children. Almost one half of these have their first child during their training period, a time which usually coincides with what many view as the best physiological age for women to bear children, their 20s and early 30s. Nevertheless, a recent study found that four fifths of the residency programs surveyed had no policy on medical leave and most continue to treat pregnancy as a crisis rather than a predictable life event.[26]

Worse, some administrators seem to equate pregnancy during the training years with treason. For example, a recent article in the *Chronicle of Higher Education* reported that the chair of a gynecology department at a major medical school wrote a memorandum describing a resident's pregnancy as a "disservice" to herself and to her colleagues. His memo declared that her decision to have a baby before completing the three-year residency program raised the issue of women as "unreliable colleagues" and further challenged the idea that women should be admitted to residency programs in the first place. Indicating that we have made some progress, the dean of the medical school accepted the chair's resignation and likened his comments to those of "Attila the Hun."[27]

A more typical problem arising out of the failure to plan for resident pregnancies is the concern of other residents who may have to take on extra work to cover the leave time that might be required during a pregnancy. Aware of this, many pregnant residents feel pressured to work as long as possible despite dangers to their own health and the fetus. Many hospital program directors still seem dedicated to treating internships and residency training as an endurance test. A case in point is a recent episode at Harvard. Dr Maureen Sayres, director of the Harvard Medical School Office for Parenting, received a call from one of the directors about a resident whom he described as suffering from postpartum depression. When Sayres investigated the case, she found that this woman resident's weekly work schedule had increased from the usual 100 hours to 118 hours per week to compensate for the fact that another resident had abruptly dropped out of the program.

The woman resident complied with the new schedule but found that she was constantly fatigued and saw nothing of her newborn son. After two months, she approached her training director for a reduction in hours and was told she was expected to carry her weight. Dr Sayre's examination of the woman indicated that there was no postpartum depression, just "a normal physical and psychological response" to a work overload. What struck Sayres was that the structure of the residency training program transformed a normal response of exhaustion into a "pathological and psychiatric condition."[28]

The fact that Harvard Medical School has established an official Office for Parenting indicates that some programs are beginning to recognize the special problems which many women experience. Efforts to take pregnancy into consideration are not without their problems, however. The issue of protective labor legislation for women has divided feminists since the passage of the 19th amendment. Even legal scholars are in deep disagreement on this issue. In the book *Gender Justice*, three legal policy experts dismiss maternity benefits as "the latest version of paternalism."[29] Nevertheless, on balance, training programs that deal with pregnancy as part of a general parenting package would meet a real need. It is interesting to note that the day care center in the Harvard Medical complex is five times more difficult to get into than the medical school itself.

This is one example of how the special problems of women raise issues that have relevance for all medical trainees. If the increased numbers of women in medicine do nothing more than force medical schools and hospitals to rethink the structure of their training programs, women will have made a major contribution to medicine.

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FROM THE LIBRARY

WOMEN IN MEDICINE

TO THE EDITOR: I have read with very great interest your editorial on Women in Medicine, which appeared in the issue of the *New York Medical Journal* for November 21, 1923.

You were kind enough to say that "on the whole women who have chosen the medical profession appear to have justified the selection of a career." You say, also, that "preventive medicine offers better facilities for women than ordinary practice."

You give to women the credit for being intuitive and admit "the great importance of this quality in diagnosis and prognosis when reinforced by training." You deserve credit for these laudatory statements but, do you not stultify them by subsequent statements not based on up to date knowledge of facts?

You say, "there are few women in general practice." The fact is, that the census of 1918 gives the name and address of more than seven thousand medical women in the United States, and the number in England is even larger, I believe, in relation to the small area covered.

You further limit them by saying "they appear *only* to be adapted for certain kinds of medical practice," and this lack of adaptability was shown during the war more with women than men. The facts are that a large number of medical women filled the places left vacant by medical men who served in military hospitals in this country, in England and in France. One woman physician, whom I know personally, carried on her own large general practice and that of her husband during the entire war, and this was done without either *mental* or physical breakdown. . . .

Farther on in your article you say, rather apologetically, "In the factors which make for success in *any sphere* of life it may be that women are not as well equipped by heredity as men." If this were true, one would be tempted to ask, do women inherit nothing from their fathers?

Sir Humphrey Rolleston, from whose address at the Royal Free Hospital in London some of these statements in your editorial are borrowed, advises "women students to remember that they are more liable to mental breakdowns than men." In forty years of acquaintance with women medical students, I fail to recall one mental breakdown among them, and it has long been known that women continue the practice of medicine to a greater age, as a rule, than do men.

The last bomb thrown at medical women in your editorial, whether manufactured across the Atlantic or in America, is, to say the least, filled with a poison gas, unworthy of its source. May I repeat it? "It would appear that there is a *definite place* for women in medicine *but* that at any rate in existing circumstances they are not nearly so well fitted as men to practice medicine. In the course of time a type of woman may be evolved who will equal man as a practitioner of medicine, but that time has not come yet."

I leave it your readers to decide whether, as a matter of fact, this "is very laudatory" to women physicians, as you suggest in your letter to me.

ELIZA M. MOSHER
(*NY Med J Rec* 1923; 118:784)

Deep vein thrombosis in pregnancy

CASE PRESENTATION

Aruna Pulijaal, MD (Medical Intern): A 37-year-old woman was admitted to the New York Infirmary Beekman Downtown Hospital with left-sided chest pain and shortness of breath of two days' duration.

Three years and one year prior to admission, the patient had had two incomplete abortions, both of which required dilation and curettage. She was currently 13 weeks pregnant. Ten days prior to admission, she noticed the onset of vaginal spotting; complete bed rest to forestall threatened abortion was advised by her obstetrician. Two days prior to admission, she noted mild pain and a sense of fullness in the left calf; almost simultaneously she noted the onset of sharp stabbing pain in the left side of her chest. The pain was nonradiating but worsened with respiration, and was associated with shortness of breath and occasional palpitations. She denied cough, sputum production, hemoptysis, sore throat, nasal symptoms, fever, chills, substernal chest pain, nausea, vomiting, or diaphoresis.

The patient denied past use of contraceptive medications and was taking no other medications. The patient's medical history was not otherwise remarkable. Her menarche occurred at 13 years of age; menstrual periods were regular but brief.

A review of systems was entirely negative. The family history was noncontributory. The patient works as a seamstress; she denied cigarette smoking, excessive use of alcohol, or drug abuse.

Physical examination disclosed a well-nourished woman in mild respiratory distress. Her blood pressure was 108/70 mm Hg; pulse rate, 104/min with regular rhythm; respiratory rate, 24/min; temperature, 99.8°F. The skin was warm, dry, and free of lesions. There was injection of the right conjunctiva with a small amount of purulent discharge. Examination of the ears, nose, and throat was unremarkable. The lungs showed dullness on percussion over the left lung base posteriorly, with decreased breath sounds over both lung bases, especially on the left. There were no audible wheezes, rales, or pleural rubs. The heart sounds were normal without murmurs. The abdomen was soft, with normal bowel sounds; no masses or enlarged organs were palpable. The rectal examination was normal; Guaiac test for blood in the stool was negative. The cervix uteri appeared nulliparous; the external os was closed with dark blood in the vaginal vault; the uterus was palpable at about ten weeks' size. There was mild tenderness of the left calf, which was approximately one inch greater in girth than the right; there was no local redness or increased warmth. Homan's sign was negative; no thrombosed vessels were palpable. No edema was present in the lower extremities. The neurologic examination was entirely normal.

Urinalysis was negative, including microscopic examination. The hemoglobin was 12.2 g/dL; hematocrit, 35.3%; white blood cell count, 11,000/μL, with 85% mature polymorphonuclear leukocytes, 2% immature forms, 8% lymphocytes, 4% monocytes, and 1% eosinophils. The platelet count was 169,000/μL; prothrombin time (PT) and partial thromboplastin time (PTT) were at control levels. Blood urea nitrogen, serum creatinine, blood glucose, and serum electrolyte levels were all within normal limits.

Arterial blood gas determinations, with the patient breathing room air, were as follows: pH 7.46; $P\text{CO}_2$, 35 mm Hg; $P\text{O}_2$, 72.4 mm Hg; bicarbonate, 25 mEq/L; base excess, 1.5 mEq/L; oxygen saturation, 97.1%. The alveolar-arterial oxygen gradient was 33 mm Hg. With the patient breathing 2 L/min of oxygen by nasal cannula, the arterial pH was 7.46; $P\text{CO}_2$, 35 mm Hg; $P\text{O}_2$, 101 mm Hg; bicarbonate, 24 mEq/L; base excess, 0.8 mEq/L; and oxygen saturation, 98.1%.

The electrocardiogram showed normal sinus rhythm with a rate of 90/min, normal electrical axis, and normal intervals. There were no acute changes of ST segments or T waves.

A pelvic sonogram showed a blood clot in the uterus with retained products of an incomplete abortion. The venogram revealed extensive left leg deep venous thrombosis (Fig 1).

The patient was transferred to the medical service. Pulmonary embolism was strongly suspected, and the patient was immediately given a full course of intravenous heparin, with the aim of keeping the PTT at 2 to 2½ times control levels. Two days following transfer, a ventilation/perfusion lung scan showed a matched defect in the left lower lobe, a mismatched perfusion defect in the right middle lobe, and a questionable defect in the posterior segment of the right upper lobe (Fig 2). The patient's condition improved steadily, with subsidence of the chest pain, shortness of breath, calf swelling, and tenderness.

Nine days after transfer, a dilation and curettage removed the remaining products of conception. Intravenous heparin was temporarily discontinued seven hours prior to the procedure and was restarted five hours afterwards; protamine sulfate was immediately available in the event of significant excess bleeding. On the same day, warfarin anticoagulation was also begun. On the tenth day after transfer, a repeat ventilation/perfusion scan indicated improvement in both the right and left lung field defects.

Based on a clinical conference held at New York Infirmary Beekman Downtown Hospital, New York, NY, on February 3, 1989.

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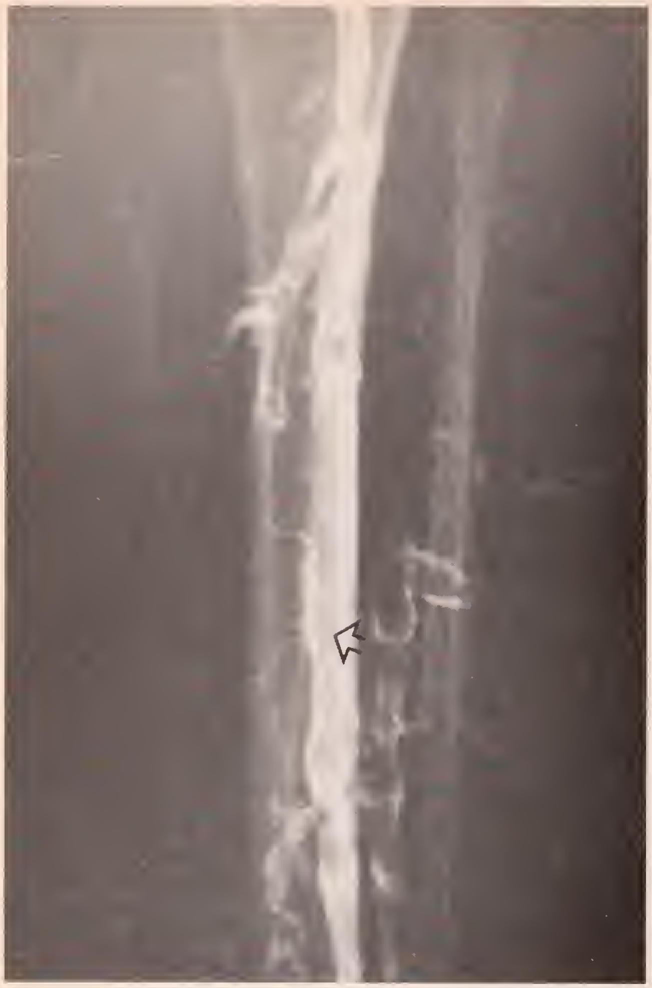

FIGURE 1. Venogram of the left lower leg with elongated venous thrombus (arrow).

Thereafter the patient was closely observed for evidence of bleeding; to date, repeat stool Guaiac tests, urinalyses, and blood counts have given no indication of blood loss. The patient was discharged to her home, and warfarin therapy will be continued.

Irwin Sharkey, MD (Chief of Medicine): I think we should consider how we might have managed this patient if her pregnancy had not terminated in an abortion.

John T. Flynn, MD (Associate Chief of Medicine): What was the result of the pregnancy test?

Dr Pulijaal: It was positive at the time of admission.

Steven Domnitz, MD (Senior Resident, Obstetrical Service): The patient probably had viable placental tissue at that time, but did not have a viable fetus. The pelvic sonogram did not show a blood clot, but rather an empty gestational sac, which strongly suggests the presence of a blighted ovum. A positive pregnancy test only suggests a recently viable pregnancy, it does not confirm present viability. We expected the serum pregnancy test to be positive; at 13 weeks, in the absence of a fetal pole and fetal heart motion, the diagnosis is one of missed abortion or blighted ovum.

Dr Sharkey: What did the pathologic specimen show?

Dr Pulijaal: Only chorionic villi and blood clots.

Radiologic Presentation

George Braff, MD (Chief of Radiology): The first nuclear perfusion scan of the lungs, in the right posterior oblique (RPO) projection, shows a wedge-shaped defect in the lateral segment of the right middle lobe (Fig 2). Diminished activity is present at the left lung base.

With positive findings, it is necessary to determine whether there is a match or a mismatch on the subsequent ventilation scan. Specifically, this means whether the defects—the photon-deficient areas—on both scans are found in the same location in the lung, constituting a match; or whether the defects noted on the two scans occur in different locations, a mismatch. The ventilation scan here did not demonstrate comparable defects on the right, while diminished activity was noted at the left lung base. Therefore, we have a mismatch, suggesting a high probability that the perfusion scan defect in the right middle lobe represents a pulmonary embolus.

The lack of nuclear activity at the base of the left lung on that examination is explained by the chest film, an important complementary study. With a positive chest film finding, a pulmonary embolus in that location is of low probability, but an infarction could present in that fashion.

Consequently, this initial set of scans suggests a pulmonary embolus of the right middle lobe. My confidence in the diagnosis is raised by the finding that on a second set of scans eight days later, and approximately 12 days after the beginning of anticoagulant therapy, the wedge-shaped defect on the perfusion scan has disappeared (Fig 2).

The chest film taken on admission is negative in the region of the right middle lobe. The later chest film shows an elevated hemidiaphragm, an extensive degree of atelectasis on top of the infiltrate on the left, and a second patch at the right lung base (Fig 3). The wedge-shaped defect on the perfusion scan was located more laterally, as seen in the right posterior oblique position, and therefore was independent of these findings. Follow-up chest films demonstrate partial clearing in the vicinity of the right hemidiaphragm, although the density at the left base is still present.

The venogram shows one characteristic of a thrombus, a track—two white parallel lines with a negative filling defect between them (Fig 1). The superficial veins do not

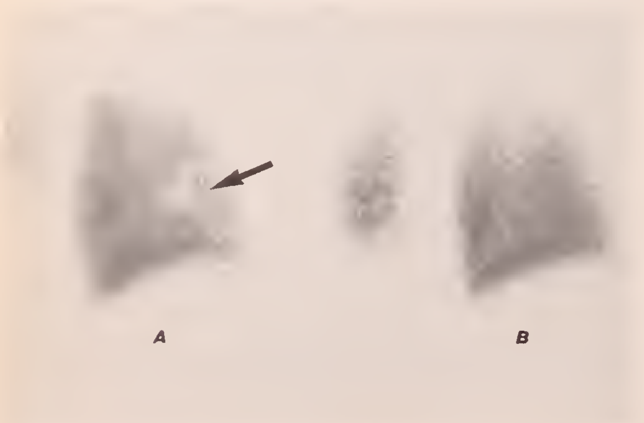

FIGURE 2. A, Initial nuclear perfusion scan of lungs, right posterior oblique view, showing wedge-shaped defect (arrow) in lateral segment of right middle lobe, due to pulmonary embolus. B, Repeat scan eight days later, demonstrating clearing of defect.

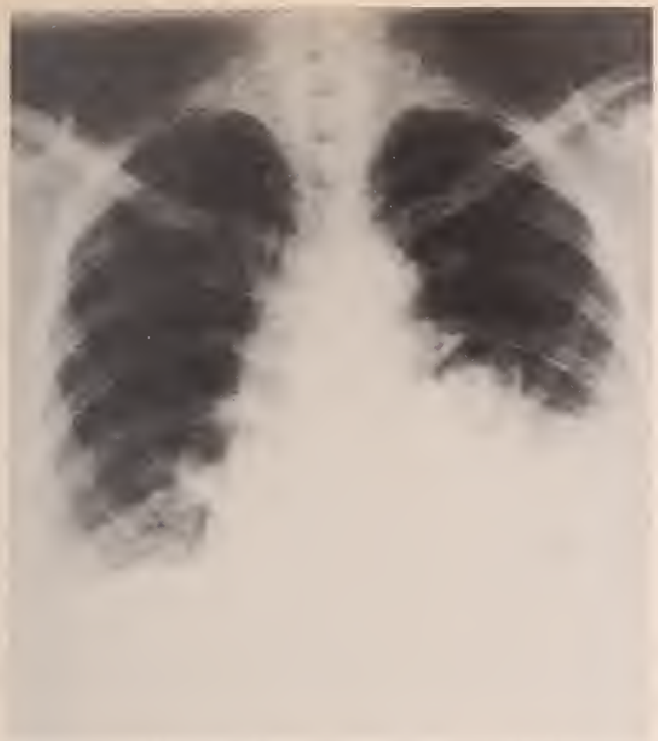

FIGURE 3. Chest film (posterior-anterior view) showing elevation of left diaphragm, with pleural fluid and atelectasis at left base, and patch of atelectasis above right base.

fill out at all. The films are purposely overpenetrated; with underpenetrated films there is the risk of failing to visualize the contrast material. Segments of the deep venous system demonstrate pruning, which is an indirect sign of thrombus formation. The primary finding is a track with a serpiginous filling defect through the deep system, consistent with an extensive thrombus.

Dr Sharkey: Is that degree of loss of volume in the left lung common with pulmonary embolism?

Dr Braff: If you have a volume loss of this magnitude, then you would suspect the possibility of a pulmonary infarction, not merely an embolus. The area that we saw on the RPO projection on the perfusion scan remains relatively clear on the chest films, and that finding is most consistent with a pulmonary embolus.

The radiographic changes in the left lung field are due to either an infiltrate, fluid, infarction, or pleuritis. The nuclear studies show a right-sided pulmonary embolus, but I would not be able to comment on whether there may be a pulmonary infarct at the left base. The left-sided chest pain must be due to something other than the pulmonary embolus on the right.

Dr Sharkey: Any other lesions in the lung could be due to pulmonary emboli, since, based on clinical findings, she had clearly had one such episode.

Mathiah Sukumaran, MD (Attending Pulmonologist): I felt that she had a pulmonary infarction on the left side as well. The pain subsided after four or five days, and the chest film also improved after that, though it did not return to normal.

Kok-Tong Ling, MD (Medical Resident): I wish to discuss the diagnosis of deep vein thrombosis (DVT), with attention to the etiology and pathogenesis. The German pathologist Virchow first introduced the term thrombosis and postulated three possible mechanisms: stasis, endothelial damage, and hypercoagulability.[1] Stasis is present in patients on prolonged bed rest, after trauma, or with congestive heart failure, infection, shock, obesity, malignancy, or pregnancy. Endothelial damage can arise from hypoxemia, trauma, collapsed vessels, indwelling catheters, injection of irritating substances, and sepsis. Hypercoagulability may develop in patients with a strong family history of DVT, malignant tumors, use of contraceptives, blood dyscrasias, and deficiencies of antithrombin III, protein C, or protein S. These three main causes may be intermingled. The most common transient hypercoagulable states are associated with recent trauma, major surgical procedures, and sepsis.[1]

In the pathogenesis of DVT, most venous thrombi begin as a platelet nidus on the valve cusps of the deep calf veins. Tissue thromboplastin is released, forming thrombin and fibrin which trap red blood cells and propagate proximally as a red thrombus. Such a propagating thrombus may become attached to the opposing walls of the blood vessel, causing interruption of blood flow, retrograde thrombosis, and signs of venous stasis. Many times the thrombus propagates without interrupting the venous flow and develops a long floating tail which is liable to break loose, causing pulmonary embolism without premonitory signs or symptoms.[1]

The clinical diagnosis of DVT is difficult. Only about 40% of patients with DVT have any clinical findings. Pathologic studies and studies using iodine 125-labelled fibrinogen have shown that at least 50% of deep vein thromboses are silent.[2] Moreover, symptoms and signs, when they are present, tend to occur late in the course of the disorder. Pain, swelling, discoloration, distended veins, and blanching are the more obvious clues to DVT ("milk leg"), though these are not frequent. Homan's sign is an unreliable indicator of DVT.[1] Clinical signs that may falsely suggest DVT can arise from other conditions, such as rupture of calf muscles, nonspecific trauma, muscle tear, and popliteal cyst rupture. Recently, painful and tender swelling of the lower extremity associated with overlying cutaneous erythema in patients with the acquired immunodeficiency syndrome (AIDS) can suggest DVT falsely.[3]

On the other hand, over-diagnosis is common. False positive diagnosis of DVT with a rate of 45–50% is noted using physical diagnosis alone, as compared with objective tests such as venography.[4] There are, however, a number of other diagnostic tests that are fairly reliable, noninvasive, and generally available for the early detection and follow-up of deep vein thrombosis.[5] These include impedance plethysmography, Doppler ultrasonography or duplex studies, and radiofibrinogen leg scanning using iodine-125 labelled fibrinogen. Impedance plethysmography measures the volume of the extremities with temporary occlusion of the venous system. A pressure cuff is applied to the upper thigh and is inflated to 50 mm Hg or until maximum filling has occurred. The cuff is then rapidly deflated, allowing the rapid outflow and reduction of the volume in a normal limb. Prolongation of the outflow wave suggests a major venous thrombosis with 95% accuracy—much more reliable than any other voluntary technique of venous occlusion.[1]

Impedance plethysmography (IPG) is highly sensitive to the presence of above-the-knee thrombosis,[5] but it fails to detect many below-the-knee thrombi. It is useful for prospective studies, because when thrombi begin in the calf veins, a subsequent series of sequential IPG studies can detect the extension of such thrombi into the deep venous system of the thigh.[3] Through serial examinations, the diagnostic accuracy of this procedure has increased and is comparable to that of venography. Plethysmography is noninvasive and can be done in the outpatient setting.

Radiofibrinogen leg scanning involves injection of radiolabelled fibrinogen into the veins of the foot, after iodine blockade of the thyroid gland, which takes about 24–36 hours. Readings over both the calf and the thigh are recorded as a percentage of surface radioactivity obtained over the heart. Venous thrombosis can be suspected if there is an increase in the ratemeter reading of greater than 20% at any point on the leg when compared with readings over adjacent parts of the same leg, or over the same point on a previous day, or over corresponding points on the opposite leg.[2] Venous thrombosis is diagnosed if the scan remains abnormal at a second examination, and if the abnormality persists for more than 24 hours. Radiofibrinogen leg scanning is very sensitive to thrombus formation in the calf veins and lower thigh veins, but is not sensitive to thrombi that have formed in the upper thigh or above.[1]

A study published in 1985[6] outlined the long term follow-up of 645 patients with clinically suspected venous thrombosis but with normal IPG findings. During periods of initial surveillance, DVT was identified in six (1.9%) of 311 patients tested by serial IPG alone, and in 30 (9.2%) of 323 patients tested by serial IPG plus leg scanning with radiolabelled fibrinogen (most with calf-vein thrombosis). On long term follow-up, six (1.9%) of patients studied by serial IPG alone developed DVT, as compared with seven patients (2.2%) studied by the two tests. Five of the six patients studied by serial impedance plethysmography alone developed venous thromboses between the fourth and the 11th week of follow-up; among the seven studied by the combined approach, four developed venous thromboses between the third and the 11th week, and the remaining three between the fourth and the eighth month. Thus, in suspected cases of venous thromboembolism, the detection of DVT is about as effective with serial impedance plethysmography alone as with the combination of serial IPG and radiolabelled fibrinogen. Doppler ultrasonography is highly dependent on the operator's experience.[5]

The most reliable technique of confirming the diagnosis of deep vein thrombosis and ascending involvement from calf veins is ascending venography. The procedure can also be performed with isotope injection using a gamma scan counter to record the uptake of the isotope. Delayed imaging of persistent "hot spots" may also reflect isotope retention at the site of thrombus formation. A perfusion lung scan can also be obtained for baseline comparison and for detection of silent embolism.[1]

Venography is the most useful diagnostic criterion, with demonstration of a persistent filling defect, venous obstruction, and evidence of collateral circulation. But venography does not always fill all of the veins of the lower extremity; interpretation of the films requires familiarity with the varieties of venous architecture.[4] In the absence of a filling defect, one cannot detect a thrombus. Venous inflammation may be induced by the dye in fewer than 5% of patients. This may be avoided by emptying the vein with heparinized saline solution at the end of the study.

In pregnancy, there is danger of exposure of the fetus to radiation during the venography. In addition, the diagnosis of deep vein thrombosis is even more difficult in pregnant than in nonpregnant patients, because leg pain and swelling are more nonspecific in pregnant patients. The diagnosis of deep vein thrombosis during pregnancy may be complicated by the fact that standard venography exposes the patient to radiation, while impedance plethysmography has the potential to be falsely positive in the latter part of the third trimester, because of the pressure of the markedly enlarged uterus on the common iliac veins.[7]

In clinically suspected deep vein thrombosis during pregnancy, impedance plethysmography should be done initially; if this is negative, then sequential IPG studies can follow on days three, five, seven, and ten. If the IPG is positive and if the patient is in the first trimester or the second trimester of pregnancy, then the diagnosis of proximal deep vein thrombosis is made, and the patient is treated with heparin.[7]

If the patient is in the third trimester, there are two possibilities. The test may be a true positive, or a false positive test caused by the enlarged uterus and compression of the common iliac veins. Consequently, the physician has two options: either to treat the patient with heparin if there is a likelihood that the test is a true positive, or to proceed with limited venography, using a lead-lined apron to cover the patient's abdomen and reduce the fetal exposure to radiation. However, this protective procedure limits the value of the test, because the apron over the abdomen may prevent adequate visualization of thrombi in the common iliac and femoral veins.

If the venogram is positive, then the patient is treated with heparin. If the limited venogram is normal but the IPG is positive, we have not excluded deep vein thrombosis and consequently may have to risk performing the complete venogram, which includes radiation to the fetus. If the complete venogram is normal, then deep vein thrombosis is excluded; if it is positive, then the patient is treated with heparin.

Cathy Schwartz, MD (Medical Resident): Heparin is the therapy of first choice in pulmonary embolism. If this condition is strongly suspected, heparin therapy should be started immediately, without waiting for diagnostic confirmation, unless the initial dose of heparin places the patient at great risk for a serious complication of anticoagulant therapy, which usually involves bleeding.[5]

The overall aims of treatment in deep vein thrombosis and pulmonary embolism are immediate cessation of the growth of thromboemboli; promotion of the resolution of the underlying thromboembolic condition; and prevention of recurrent thrombosis and future embolism. Heparin can achieve the first goal and promotes the second by allowing the normal processes of fibrinolysis to proceed unopposed by thrombus growth. Heparin assists but does not ensure prevention of recurrent thrombosis. In addition, heparin inhibits platelet aggregration at the site of the embolism. Its anticoagulant actions can be promptly reversed, if necessary, by injections of protamine.

Anticoagulation therapy of thromboembolic disease has not yet attained a uniform consensus of clinical opinion. Certain aspects remain uncertain, such as the safest and most effective dosage regimen, the most informative coagulation profiles to monitor during therapy, if at all, the long term duration of the anticoagulant treatment, and the selection of those patients for whom thrombolytic therapy should precede antithrombotic therapy.

Three methods of heparin administration have been advocated in deep vein thrombosis and pulmonary embolism: continued intravenous administration of heparin, commonly administered at a rate of about 1,000 units per hour; intermittent intravenous administration, approximately 5,000 units every four hours, or 7,500 units every six hours; and subcutaneous administration of heparin at 5,000 units every four hours, or 10,000 units every eight hours, or 20,000 units every 12 hours.[5]

At present, the continuous intravenous regimen delivered by an infusion pump is the most popular method. Many practitioners advocate an initial intravenous bolus dose of at least 10,000 units of heparin before beginning one of the standard regimens.

The value of the activated partial thromboplastin time (aPTT) or of other measurements of coagulation activity during heparin therapy is not clearly established, since the risk of bleeding from heparin therapy is not clearly correlated with such coagulation profiles. That risk appears to be related to the coexistence of other conditions contributory to bleeding, such as peptic ulcer, coagulopathies, uremia, and increasing age. Animal studies indicate that maintaining the aPTT greater than one and a half times its normal control level prevents the formation of venous thrombi. Such data are not available from human studies; however, many physicians continue to measure the aPTT and adjust the heparin dosage so that the aPTT is prolonged at slightly more than one and a half times the patient's baseline value. If that baseline value lies above the normal control range, the baseline should be assumed to be in the middle of the normal control range. If the patient's baseline value in seconds is less than the average normal control range, then the aPTT can be prolonged to at least twice the patient's baseline control value.

In deep vein thrombosis and pulmonary embolism, a full-dose course of heparin is usually maintained for seven to ten days—the period of time usually required for the dissolution and/or organization of the thrombus. Beyond this acute phase, the clinician can either continue with intermittent heparin given subcutaneously or can change to oral coumadin therapy. With intermittent subcutaneous heparin, a reasonable dose is one sufficient to prolong the PTT to at least one and a half times the baseline value at the halfway points between injections.[5] A dose of 7,500 to 10,000 units given subcutaneously every 12 hours is considered to be adequate for prophylaxis, is generally safe, and may not need to be monitored with coagulation profiles.

When oral anticoagulation with coumadin is used for longer term anticoagulation, a dose is selected that prolongs the prothrombin time (PT) to 1.3–1.5 times the patient's baseline control value. The test for the PT, however, must utilize rabbit brain thromboplastin, the constituent used in most commercial laboratories in the United States. No additional benefit has been demonstrated by further prolongation of the PT or PTT beyond 1.5 to 2 times the control values, but a higher incidence of bleeding complications has been noted.

When the decision is made to change from parenteral heparin therapy to oral anticoagulation with coumadin, treatment with both drugs should overlap for four to five days. Coumadin should not be given to pregnant women during the first trimester, since it is fetopathic and teratogenic. Instead, adjusted doses of subcutaneous heparin can continue to be administered intermittently for longer term coagulation in pregnant women with deep vein thrombosis and pulmonary embolism, after completion of the initial full-dose heparinization.

The duration of anticoagulation treatment for deep vein thrombophlebitis remains controversial. However, most experts agree that three months of such therapy is adequate for patients who give no evidence of contributing risk factors. Longer therapy may be necessary for patients with tumors and for those who are not physically mobile. When venous thromboembolic disease recurs after anticoagulation treatment has been discontinued, such treatment should be reinstituted for at least one year. Persons with permanent thrombotic tendencies related to deficiencies of antithrombin III or protein C should probably receive anticoagulant therapy indefinitely. When thromboembolism recurs during oral anticoagulation with coumadin, the patient should again be given heparin for seven to ten days, and thereafter should continue with oral anticoagulation for at least one year.

Whether patients with calf vein thrombosis, in contrast to deep vein thrombosis in the thigh, should receive anticoagulation remains controversial. It is believed that calf vein thrombi can extend proximally to larger and deeper veins and can contribute to pulmonary embolism. Consequently, patients with calf vein involvement should probably receive anticoagulation therapy for three months.

The thrombolytic agents streptokinase and urokinase are usually reserved for treatment of massive pulmonary embolism associated with hemodynamic deterioration in the patient. An additional small embolus that develops during the subsequent 24 hours in such a patient may be lethal. A thrombolytic agent assists in stabilizing the cardiovascular functions and blood gas values more quickly than conventional therapy. However, there is as yet no proof that thrombolytic therapy is life saving in such patients. The major complication of thrombolytic agents is bleeding; this can be minimized by selecting patients without other evident risk factors for bleeding. Tissue plasminogen activator is currently under study as a thrombolytic agent.

Venocaval interruption is indicated when a life threatening pulmonary embolus develops at least 24 hours after anticoagulation therapy with heparin has been deemed adequate, or in a patient for whom anticoagulation is contraindicated. Venocaval interruption is usually performed concurrently with pulmonary embolectomy or endarterectomy.

In summary, despite the many unsettled questions in the treatment of deep vein thrombosis and pulmonary embolism, certain useful trends have emerged to assist the physician in administering treatment.

Peter M. Polos, MD (Chief Medical Resident): While there should be an overlap in time during which both anticoagulant medications should be administered, the key is that there should be a therapeutic elevation of the PT due to the coumadin before the heparin is discontinued.

Dr Sharkey: I would still like to hear a clear reason for the suggested overlap of four to five days that Dr Schwartz referred to. An overlap of only two to three days could be explained by the need to wait for the coumadin to have a therapeutic effect on the prothrombin time.

Dr Polos: It is generally accepted that overlap of two to five days is reasonable to insure that therapeutic elevations of the PT are achieved. It is clear from clinical experience that some patients require longer overlap periods than others.

Calf vein thrombosis may extend to proximal veins in about 20% of cases, leading to pulmonary embolism in less than 10% of cases. While the venogram is the gold standard for the study of venous thromboembolism in the extremity, impedance plethysmography is becoming the preferred method of making the diagnosis. If the patient is strongly suspected of having deep vein thrombosis because of risk factors or because of the clinical picture, but the impedance study is negative, then repetition of the test three or four or five times over a span of ten days can be done in the physician's office. As Dr Ling indicated, this serial method of follow-up might be considered in the hospital setting also, if indicated, before proceeding to other diagnostic efforts. Negative results on serial IPGs will exclude clinically significant deep vein thrombosis.[6,8]

Dr Sharkey: Are you saying that we should have followed that diagnostic course rather than the venogram in this patient under discussion?

Dr Polos: We could have done serial plethysmography instead. The role of lung scans in the diagnosis of pulmonary embolism has been the subject of controversy. The traditional categories of lung scans are identified as low probability and high probability for pulmonary embolism. High probability scans suggest an incidence of 80–90%.[9] However, in many instances of so-called low probability scans, there is now found to be a risk of pulmonary embolism as high as 40%. Labelling a ventilation-perfusion (V/Q) scan as one of so-called low probability may tend to deter the clinician from further pursuing the diagnosis and, therefore, missing a significant number of pulmonary emboli. There are efforts now to amend this classification, so that the terminology indicates scans of high probability and those of non-high probability.

With a normal ventilation-perfusion lung scan, the clinical management of the patient is obvious; pulmonary embolism has been ruled out and there is a 1% chance of a subsequent pulmonary embolus developing immediately. With a high-probability lung scan, you know that anticoagulation therapy must be carried out. But when there remains a gray zone of the non-high probability ventilation-perfusion scan, it is advisable to resort to impedance plethysmography. If a positive impedance result combines with a non-high probability ventilation-perfusion scan, you should treat with anticoagulation.

The gray zone involves a non-high probability scan and negative impedance plethysmography, in which case an angiogram is indicated. Studies have shown that patients with a non-high probability V/Q lung scan and a negative plethysmography, but with good cardiac and pulmonary reserve, need not be subjected to anticoagulation therapy, since this group has only a 2% incidence of pulmonary embolism within three months after such testing.[9] However, if the patient has evident cardiopulmonary disease and does not have good reserve, even in the face of a non-high V/Q lung scan and a negative IPG, you may then opt to proceed with anticoagulation therapy, since these patients are at a somewhat higher risk, 8% to 10%, for pulmonary embolism.

Pulmonary angiograms in patients with non-high probability lung scans and a negative IPG may be negative; nevertheless, the incidence of pulmonary embolism in such patients is still about 2%. Thus a number of pulmonary emboli will be missed.

Once heparin treatment is initiated with the appropriate indications, a certain amount of guesswork is involved. Traditionally, a three- to five-day overlap of heparin with coumadin has been adopted, but the reasons are not entirely clear, as has been discussed earlier.

Regimens for anticoagulation are quite varied, as I will indicate, and despite the variety have been reported to have good results. Heparinization over a ten-day period can be coordinated with coumadin treatment beginning on the seventh day, building up to therapeutic levels of the prothrombin time rapidly for at least a day or two before stopping the heparin and discharging the patient on coumadin alone. An ultrashort regimen, with a short course of heparin for four days and initiating the coumadin therapy at the end of the first day, gives three days of overlap of the two-drug effects. The results of this regimen have yet to be tested fully.

Another regimen involves starting the heparin on admission and coumadin on day two, and continuing the combined therapy over three days prior to cessation of heparin. In the case of women in the first trimester of pregnancy, when the adverse effects of coumadin should be avoided, or when the patient's situation prevents regular follow-up of the prothrombin times, adjusted dose subcutaneous heparin is the long term regimen of choice.

Thus, future medical therapy may be pointing toward shorter heparinization periods with overlap with coumadin for at least two to three days; the patient is then discharged on coumadin. Studies of this mode of therapy are underway and have not yet yielded conclusive results.

Dr Sharkey: Do you know the incidence of pulmonary thromboembolic disease in the presence of a negative V/Q lung scan?

Dr Polos: That would be less than 1%.

Dr Sharkey: The incidence of documented pulmonary embolism, when the perfusion lung scan is negative, is essentially zero. For all practical purposes, a negative perfusion scan excludes the diagnosis of pulmonary embolism.

Lester Blair, MD (Director of Pulmonary/Critical Care Medicine): A normal perfusion lung scan, done with all six views, for all practical purposes rules out the presence of pulmonary embolism.

In connection with the duration of overlap therapy, the rationale for heparin includes not only the benefit of anticoagulation but also the benefits of its anti-inflammatory effects, with the probability that it reduces the likelihood of a recurrence of embolic disease in the future and reduces the incidence of post-phlebitic syndrome in the involved calf veins.

Dr Sharkey: It has been known for several years that there are significant coagulation factors that have an effect on thromboembolic disease; these factors are reduced promptly by heparin but much more slowly by coumadin, requiring up to seven to ten days. The current recommendation in most cases is treatment with heparin for five to ten days, overlapped with coumadin for four to five days. Current studies are attempting to refine these recommendations further.[10]

If you do not treat a patient with thromboembolic disease at all, the risk to that patient of having another thromboembolic episode in the near future is perhaps between 30% and 40%. According to older data, if you treat the patients with coumadin, you reduce the risk of recurrence to about 10%–12%. If you treat with subcutaneous heparin, you reduce the risk to under 5%; with intravenous heparin, the risk is less than 3%.[10] These are strongly supported data from large numbers of patients, indicating that heparin is the better drug in reducing the incidence of recurrent thromboembolic phenomena.

There are no hard data about the effect on recurrence of thromboembolism by shifting from heparin to coumadin. We are talking about a difference between a 10% recurrence rate for coumadin versus a 5% recurrence rate for heparin. This would be a difficult study to perform, because of the large numbers of patients who would have to be treated with long term heparin.

Dr Blair: I think that the choice of regimen is not as important as the ability to maintain a therapeutic PT and PTT. Some studies suggest that patients did not do well on a particular regimen because of a specific drug or route of administration; but, in reviewing the data, it appears that the major determinant of success or failure was the PT and PTT. If you can achieve a therapeutic PT and PTT the results are good, no matter which regimen you use.[11,12]

This question is still being studied, with regimens that compare early initiation of coumadin, that is, on day three of heparin therapy, versus starting it later, say on day seven; to date, no difference in outcome has been seen. Perhaps the studies will be further extended, for example, starting coumadin together with the initiation of heparin on day one, and noting any differences. But I do not think such a study will be made.

I would like to comment on a point made by Dr Schwartz related to the difference in methods by which the PT is measured. In Europe, human brain thromboplastin is used in the measurement, and with this test, the PT level should be kept elevated to a measured level of 1.5 to 2.0 times the control. In the United States, rabbit thromboplastin is used, so that the PT standard level that is therapeutic is lower than that in Europe.

Dr Sharkey: What about the role, if any, of thrombolytic agents in the management of thromboembolism? I don't think that we should be using them routinely. There are several studies underway, but the role of thrombolytic agents is still uncertain and must be individualized. A patient with a pulmonary embolus that results in shock has an extremely high risk of mortality, and there are no data that show that you can bring down that mortality figure by using thrombolytic agents. Previous studies of urokinase really did not show any beneficial effect on the mortality associated with acute pulmonary embolism, even though there was more rapid clot lysis.

The question was raised as to how long you should treat thromboembolic disease with anticoagulants. Basically, there are no good answers, because most people continue the medications for a varying period of time, usually for at least three months, after the risk factors for the thromboembolism have disappeared. If the risk factors continue, then the treatment should continue.[10]

Then we have the difficult questions related to the use of anticoagulants during pregnancy.[13] Can coumadin be given in the second and third trimester?

Dr Domnitz: Again the risk factors involved in pregnancy are due to venostasis, with the weight of the gravid uterus causing vena caval compression. In addition to venous stasis, hypercoagulability in pregnancy is associated with decreased fibrinolytic activity and increased coagulation factors. Approximately 75% of the thrombotic and thromboembolic events in pregnancy occur after delivery, especially in the first three days following delivery.[14] With cesarean section, the risk of thromboembolic disease rises fourfold. Thromboembolic disease accounts for almost half of all obstetric morbidity and mortality.[15]

Dr Sharkey: But that means that a patient who has a thromboembolic episode in the first trimester is really at risk for some time afterwards.

Dr Domnitz: That is correct.

Dr Sukumaran: The risk of thromboembolic events in pregnancy is estimated to be more than five times that in a person who is not pregnant.

Dr Sharkey: Let's say that this was the first trimester of pregnancy. A venous thromboembolus reaches the lungs, the patient must be given anticoagulation therapy with heparin. How long are you going to keep such a patient on heparin? At what point in the later trimesters of pregnancy would you then be willing to risk the institution of coumadin therapy? When you introduce coumadin in the second or third trimester, how long do you continue it? Up to, through, and after delivery? What is the risk of bleeding during delivery if the patient is on coumadin?

Dr Domnitz: There is still no standard of care in this regard. It is clear that coumadin is embryopathic in the first trimester of pregnancy, resulting in a variety of well-known facial congenital anomalies.[16] In the past, heparin was continued throughout the first trimester; in the second trimester, the patient was then switched to coumadin. Then at about 34 to 36 weeks of gestation, the patient was switched back to heparin and maintained on that anticoagulant until the onset of labor.

Once the patient was in labor, heparin was stopped until about six hours after delivery, at which time it was restarted. Heparin or coumadin was then continued for six weeks postpartum. If the patient is still receiving therapeutic anticoagulation when she arrives in labor, protamine sulfate can be used to reverse heparin effect; alternatively, fresh frozen plasma will counteract the effect of coumadin. If the patient delivers while still on anticoagulation medication, bleeding should not be a problem, since hemostasis following delivery is accomplished by tetanic contractions of the myometrium, which occludes vessels, in addition to involvement of the usual coagulation pathways. Most studies have shown that fetal hemorrhagic problems were associated with the trauma to the infant passing through the birth canal at the time of delivery.[17,18]

So clinical decision making has swung around toward the idea of not giving coumadin in the third trimester. There are still some reports, however, which suggest that coumadin may cause fetal hemorrhage in utero not associated with trauma. Many obstetricians are now switching back to using heparin for the entire course of the pregnancy.

Dr Sharkey: If this patient's pregnancy had continued, what would you have done? Would you have continued heparin for the entire duration of pregnancy by means of self administration?

Dr Domnitz: Yes.

Dr Sharkey: I have the same impression. I would be hesitant to use coumadin in any phase of pregnancy.

Dr Domnitz: The other important fact to be mentioned, not only for the pregnant patient but for any female in the reproductive years who is receiving therapeutic anticoagulation therapy, is the need for effective birth-control measures, so that pregnancy does not begin while the woman is taking coumadin. Also, you should try to prevent ovulation if at all possible, because with every ovulation, there is a potential for bleeding from the ovary. Progestational birth control, if at all possible, is recommended for any woman on anticoagulant therapy with coumadin.

Dr Sharkey: You want to eliminate ovulation rather than the coumadin. The other point that I wish to raise is if this patient still had a viable fetus, would we have done a venogram? Would we have done ventilation and perfusion scans? We would probably have to limit our diagnostic approach if we strongly suspect thromboembolism in a pregnant patient.

Dr Domnitz: You can do a venogram if you suspect that the venous obstruction is not high in the pelvis. You have to shield the abdomen in the venogram; and, as was mentioned, if you suspect the venous obstruction is high in the thigh or even in the pelvis, you will no doubt shield the pelvis and run the risk of not detecting the venous obstruction. I believe that impedance plethysmography and Doppler studies are indicated for diagnostic evaluation in such a situation.

Dr Sharkey: You would not use isotope studies.

Dr Domnitz: Radioisotopes are contraindicated because the radio-iodine would concentrate in the fetal thyroid.[19,20]

Dr Sharkey: The need to consider diagnostic pulmonary angiography should be rare.

Dr Domnitz: If you deal with cases of pulmonary embolism in pregnancy, the mother's life comes first, since maternal mortality is so high. Sometimes we must place the fetus at risk in order to make a diagnosis of pulmonary thromboembolism.

It was mentioned that venacaval obstruction by the uterus in the third trimester may cause false positive impedance plethysmography. But the progression toward that false-positive status is really a continuum; it can occur anywhere from the second trimester on. But it certainly becomes more and more prominent as the pregnancy progresses.[19]

Dr Sharkey: I think that in a patient like this, with the symptoms and signs that she presented, with the high risk and with the absence of contraindications to anticoagulation, further diagnostic tests would not have been necessary.

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Perspectives on women physicians in New York State: Past, present, and future

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In the past 20 years, the number of women physicians in the United States has quadrupled, approaching 80,000 in 1985. In 1987, one third of all medical school graduates were women. New York State has the largest percentage of women physicians in the country, with approximately 14%.[1]

Women physicians in New York State have a rich historical tradition. It is ripe with women challenging the antiquated system, demanding their right to study and practice medicine. Women physicians were among the first advocates of the poor, of women, and of children. They were the first to organize into professional societies and thus served as role models for women nationwide.

This paper focuses on women physicians in New York State, from their lonely beginnings in the mid-1800s through the 20th century, when they slowly but steadily grew in numbers and influence. Their present status in the medical community is discussed, and an attempt is made to explore and project what lies ahead in the future. One thing is certain: Dr Mary Lobell's statement, "The woman physician has come and she has come to stay," is as true today as it was in 1905 when she wrote it in the *Women's Medical Journal*.[2]

THE PAST

The Origins of Women Physicians in New York State. Prior to the 1850s, women were not a part of the medical establishment. They did, however, practice medicine, as they had since colonial times. They were lay practitioners who provided most of the medical care given in the home. They were nurses, midwives, dabblers in folk and herbal medicine, and the first health reformers. By the mid-19th century, with the increasing numbers of male physicians and the rise in professionalism, there was a real need for women to have a formal medical education and credentials.[3]

In 1849, Elizabeth Blackwell became the first woman to earn a medical degree from a recognized medical college in the United States. She gained admission to the Geneva College of Medicine, a small school in upstate New York, much to the chagrin of the dean and faculty. She had not chosen an easy road to travel; she was snubbed by the townspeople. She was asked to be absent when her anatomy class studied the male reproductive system, and, when she finally graduated at the top of her class, the Medical Society of the State of New York censured the school. In 1850, Harriot Hunt, after gaining entrance to the Harvard Medical School, was rejected and forced to leave the class after a threatened boycott by the students. She succeeded in obtaining a medical degree from Syracuse University as a homeopathic physician and later went on to become a professor of midwifery and diseases of women and children in Rochester.[4(p569)]

Women's Medical Schools. Although those early women physicians proved that they were not only physically but intellectually capable of pursuing a career in medicine, the American medical schools were resistant to giving them places in entering classes. Because of this, it became increasingly clear to those pioneering women physicians that it was necessary to open schools of medicine exclusively for women students. The first of these schools, founded in 1850 by a group of Quaker gentlemen who believed that women should be given the opportunity to study medicine, was the Female Medical College of Pennsylvania (later, the Woman's Medical College of Pennsylvania and now the Medical College of Pennsylvania). Very early on, however, New York became the center for the education of the woman physician.

The first of the New York medical schools, The New York Medical College and Hospital for Women, was founded in 1863 by Clemence Lozier, who received her medical degree from the Eclectic Medical College of Syracuse. In 1868, Elizabeth Blackwell, in conjunction with her sister Emily and Marie Zakrzewska ("Dr Zak"), founded the Woman's Medical College, which was attached to the New York Infirmary. This school later merged in 1900 with Cornell University Medical College. However, women students were only allowed to attend classes at the upstate Ithaca campus. The Hygeio-Therapeutic Institute was founded by Mary Gove Nichols, a noted lecturer and health reformer in the mid-1800s.

The women's medical schools served several important functions. First, they offered a genteel, nonprejudical atmosphere in which women students were able to study and learn the art of medicine. They also offered teaching positions for outstanding women physicians, enabling several women faculty members to become known and respected members of the medical community. The hospitals attached to the medical schools offered clinical experience and opportunities for research unavailable elsewhere.

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These institutions also served as springboards for many of the health and welfare reform policies that women physicians were dominant in bringing to the attention of the medical community. Finally, the practice of "networking" had its origins in the women's schools and hospitals.[5(p159)]

Women's medical schools, especially in New York State, were genuinely supported by several prominent male physicians. The New York County Medical Society served on the Board of Examiners of the Women's Medical College, with several of its most prestigious members also serving as consultants and teachers. By the 1880s, the women's medical colleges offered an education as good as any of the "regular" medical schools. The university medical schools finally began to accept women at the end of the 19th century. By 1890, 35 schools allowed women in their classes, although there were quotas.

Women's Medical Societies. The women doctors of New York State were in the forefront of originating all kinds of organizations of female physicians. In 1873, the Alumnae Association of the New York Infirmary Medical College for Women established the Association for the Advancement of Medical Education for Women. This was most likely the first formal association of women physicians nationwide. The association was renamed the Women's Medical Association of New York City in 1899. On May 2, 1908, the women physicians of upstate New York incorporated their association in Rochester and named it the Women's Medical Society of New York State, thus becoming the oldest legally incorporated women's medical organization in the United States. Soon after, in 1909, the Women's Medical Association of New York City was incorporated in upstate Albany.[6]

Unfortunately, national, state, and county medical societies refused to allow women to join their ranks. Many practitioners believed, without validity, that women physicians were too closely associated with the medical counterculture of the eclectics (botanic doctors) and the homeopaths. One of the more progressive medical societies, the Medical Society of the County of Kings, accepted its first woman member in 1877. Phoebe J. Ferris was a graduate of the Woman's Medical College-New York Infirmary. Shortly after, in 1879, three more women physicians became members. Between 1877 and 1914, 66 women were admitted to membership in the Medical Society of the County of Kings.[7]

In 1915, women were given full membership in the American Medical Association (AMA). That same year, the Medical Women's National Association was formed. The American Medical Women's Association (AMWA) was organized later.[4(p571)]

Pioneering and Prominent Women Physicians of New York State. The following physicians are outstanding examples of women who have practiced and worked in New York State. This list is incomplete and provides only a flavor of the accomplishments and breadth of influence of these women doctors.

- *Elizabeth Blackwell*—the first woman graduate of a recognized medical school in the United States (1850) and founder of the New York Infirmary and Women's Medical College.
- *Mary Putnam Jacobi*—founder and first president of the Women's Medical Association of New York City; first woman admitted to the New York Academy of Medicine and chair of the section of neurology.
- *Emily Dunning Barringer*—first woman ambulance surgeon in World War II; she was instrumental, with Congressman E. Celler and the Medical Society of the State of New York, in enabling women to obtain commissions in the United States armed forces.
- *S. Josephine Baker*—a prominent health reformer who structured the model for the Children's Bureau in the New York City Department of Health, which championed health care for children; the first chief of the Division of Child Hygiene in 1908, the first in the United States.
- *Mathilda Wallen*—one of the first to advocate physical therapy in heart conditions in 1912.
- *Elise Strang L'Esperance*—researcher in the prevention and cure of cancer; founder of the Strang Tumor Clinic in 1932.
- *Connie Guion*—distinguished teacher and clinician at Cornell University Medical College, for whom the Outpatient Clinic Building was named.
- *Ida Scudder*—graduate of the first class at the Cornell Union Medical College and founder of the Hospital and Medical School for Women in India.
- *Esther Pohl Lovejoy*—founder of the American Women's Hospitals (AWH), an agency that provided medical aid to Europe and Asia Minor during and after World War I.
- *Katherine Dodge*—Lieutenant-colonel who served with the United Nations Relief and Rehabilitation Administration; chief medical officer in refugee camps in Palestine and Greece.
- *Rosa Lee Nemir*—first woman to obtain full professorship in the Department of Pediatrics at the New York University Medical School.
- *Adelaide Romaine*—first woman officer of the New York County Medical Society.
- *Janet Travell*—a pioneer in the field of pain relief; appointed White House Physician for President John F. Kennedy.
- *Leona Baumgartner*—the first woman Commissioner of Health of New York City.
- *Nancy E. Gary*—Dean of the Albany Medical College.
- *Jo Ivey Boufford*—immediate past-president of the New York City Health and Hospitals Corporation.
- *Joyce M. McChesney*—first woman to be elected as an officer of the Medical Society of the State of New York.
- *Lila A. Wallis*—current president of American Medical Women's Association; originator of the earliest women physician's leadership conference.
- *Mary Ann Payne*—first woman president of the New York Academy of Medicine.

The Impact of Women Physicians in New York State. One of the greatest areas of influence of women physicians has been public health. Women physicians in the early days dominated this field. In the 1890s, the *Women's Medical Journal* made social medicine its focus.

The journal appealed to the AMA in 1905 to support programs in public health. Prominent was Rosalie Slaughter Morton, a New York State surgeon, who in 1909 came before the AMA and highlighted the need for that body to take an active role in the promotion of preventive medicine, prenatal care for mothers, and the education of the public in child health and welfare. The Women's Medical Association of the State of New York supported a standing committee on social hygiene in 1907. They also sponsored symposia for the lay public and medical professionals on public health related topics.

As a result of the caucus activity by women physicians at the turn of the century, the New York Academy of Medicine, in conjunction with the Medical Society of the State of New York, set up a series of lectures on a wide range of public health topics.[5(pp283–286)] A similar program was inaugurated in Brooklyn by the Medical Society of the County of Kings. Many of the speakers on the program were women. In 1887, Dr Lucy Hall, a prison physician and member of the Medical Society of the County of Kings, presented her paper, "Inebriety in Women, Its Causes and Results," which was a compassionate, realistic discussion of the problem entirely consonant with current thinking on the subject today.[7]

The women physicians of New York State have had an impact in other areas of medicine; however, they were truly pioneers in public health. As a tribute to the importance of women in this field, Dr S. Josephine Baker of New York City was recruited to teach at the New York University School of Medicine in 1915. Dr Baker taught at New York University for 15 years. It was through her efforts that New York University began to offer a degree of Doctor of Public Health.[8]

The Present

New York State has the largest number of women physicians (10,000) of any state in the Union. This number represents approximately 14% of the total number of women physicians in the United States. In New York State, approximately 20% of all physicians are women. This number is rapidly changing, with the graduation of larger numbers of women medical students.

Women and Medical Society Membership. Only 22% of New York State's 10,000 women physicians are members of county medical societies and the Medical Society of the State of New York (MSSNY). In an effort to bring more women into the society, the subcommittee on Women Physician Membership was formed in 1986. (This subcommittee is part of the MSSNY Committee on Membership currently chaired by Dr Randall D. Bloomfield.) The purpose of the subcommittee is to focus on increasing the membership of women physicians. Its mission is to structure a plan to attract more women into the medical society, to identify those issues of concern to women physicians, and to encourage women to take a more active role within the county as well as the state society.

Traditionally, women have tended not to join organized medicine. Nationally, only 29% of women belong to state societies, compared with 49% of male physicians. The reasons most frequently cited for this are lack of time and interest, dues believed to be too high for benefits received, the lack of representation of the views of women physicians by the medical society, inadequate opportunities for positions of responsibility and leadership, the tendency for women to be salaried and/or employed part time, etc.

The county medical societies and the MSSNY are in active competition with other organized medical groups for membership of women physicians. Women, like their male counterparts, are more apt to join specialty societies than the county and state organization. Some women are more comfortable joining AMWA, where they feel there is more support and a greater opportunity for networking. There is also competition with the AMA, which many women have joined in lieu of local groups. This has been especially true when money for society membership is limited, and the benefit package offered by the AMA had been judged equal or superior.

Presently, women are under-represented in the MSSNY, in general membership and in positions of responsibility and leadership. In the past, this was due in part to the fact that their membership was not actively sought. Prior to 1989, there had never been a woman officer or board of trustee member at the MSSNY. In 1989, Joyce McChesney, an anesthesiologist from Rochester, was nominated and elected Assistant Secretary of the Medical Society at the Annual House of Delegates meeting. Since the inception of the Subcommittee on Women Physicians and the increasing awareness that the Committee on Membership has focused on the women physicians within the MSSNY's ranks, the number of women physician MSSNY delegates to the AMA has increased to four, and the number of women delegates to the MSSNY annual convention has risen to more than 20.

At the county society level, women are frequently given opportunities for leadership, serving not only as board of trustee members, but as officers, including presidents and chairs of standing committees. Some women physicians who have distinguished themselves in the county medical societies in recent years include the following: Juana Toporovsky—Bronx, President; Nancy Nielsen—Erie, President; Ann Cea—Westchester, President; Donna O'Hare—New York, President; Elise Korman—Suffolk, President; Geraldine Keyes—Ulster, President; Coleen Livingston—St Lawrence, President; Joyce McChesney—Monroe, President; Patricia Randall—Onondaga, President; Hilda Ratner—Queens, President; Nicetas Kuo—Queens, Board of Trustees; Veronica Santilli, Kings, President-Elect.

Many women have also served as delegates and alternate delegates to the AMA and the MSSNY. There are many women physicians who have served as committee chairs and in other leadership positions. Women are also members of every standing committee at the MSSNY, the Governing Council of MSSNY Hospital Medical Staff Section (Vickie Seltzer), the AMA Council on Scientific Affairs (Patricia Numann), the Associate Editorial Board (Florence Kavaler) and the staff (Naomi Bluestone) of the *New York State Journal of Medicine*, and the MSSNY Council (Kathleen Squires, past resident representative).

In June 1987, the MSSNY council approved Resolution 87-49 (HOD) on "Women Physicians" which called for the society to set up a leadership conference to identify those women physicians who would be willing to assume a more active role within the MSSNY and their county societies. In addition, the Committee on Membership was charged with the task of continuing to focus on strategies to attract more women into the society. Thus, MSSNY mobilized its forces in an attempt to bring women into the mainstream of the society.

The Subcommittee on Women Physicians formulated a strategic marketing plan for the purpose of implementing the directives of the council concerning women physicians. The stated objectives of this plan are to increase the number of women physicians in the MSSNY and the county societies, identify women willing to take a more active role within the society, identify issues of concern to women physicians, analyze the MSSNY benefit package vis-a-vis women members, focus on women members via workshops, newsletters, etc, and open dialogue with nonmember women physician groups.

Each one of the above objectives has been addressed within the past few years. There have been membership drives targeting women physicians. Surveys have been completed identifying the concerns and life styles of women doctors, and the MSSNY benefit package has been reevaluated. Women physician delegates have met to discuss resolutions and other issues that concern them. A well-attended workshop held at the Warwick Hotel in New York City in November 1987 focused on leadership and life style skills for women physicians. (Another such symposium was held in May 1990.) The MSSNY has cosponsored with the AMWA two dinners following the 1988 and 1989 MSSNY CME Conventions in New York City. The county medical societies have been urged to sponsor local seminars and meetings for member and nonmember women. They have also been encouraged to set up task forces and committees on women physicians, similar to those in Westchester and New York County. The author serves as a liaison for the MSSNY with the Women's Medical Society of New York City.

The plan of the MSSNY to increase women membership within the society has been set in motion. As Albert S. Lyons, MD, wrote in his chapter on women in medicine, "Today, the road is open and the future journey seems clear. The first woman to become president of the state medical society may attract attention, but before long others will follow and, like their male counterparts, hardly be noticed."[4(p571)]

Women Physicians in Medical Academia. Possibly the greatest advancements for the women doctors of New York State have been in the field of medical academia. In a survey taken by the AMA in 1982,[9] major problems facing women who chose an academic career were reported. These problems included lack of academic advancement and tenure, the perception that women were less confident than their male faculty colleagues, and some negative feelings on the part of the male establishment.

In New York State, women have played a role as medical school faculty members and administrators. Nancy E. Gary, MD, who was appointed in 1989 as the 14th dean of the Albany Medical College, is the first woman dean at a coeducational medical school in New York State and the fourth nationally. There is also good representation of women as associate deans in the various medical schools in New York State. Women have also distinguished themselves as full and assistant professors at the medical schools. Cornell University recently established the Doctor Lila Wallis Visiting Professorship in Women's Health.

Although advancements in academic appointments have been made, salary structure has not kept pace. Donoghue[10] studied salary inequities for women and minorities in medical academia. She found that although women have generally received salaries that equal those of male faculty colleagues, women full professors with many years of rank tended to have significant discrepancies in salary. There are now women who hold the title of full professor and who head major departments at medical schools and hospitals. They have taken on much of the responsibility for the teaching and guidance of medical students and house staff. But there is still a discrepancy in their numbers when compared to male faculty and administrators.

Women House Staff in New York State. Data from 1987 indicated that there were approximately 3,000 women in residency training in New York State. Women, unlike their counterparts 20 years ago, can be found in every type of residency program. Although pediatrics, internal medicine, and general practice account for the vast majority of specialty areas, approximately 7% of women are in surgery and the surgical subspecialties.

Women in residency training require more from their program than their predecessors. Changes deemed necessary include a flexible work and on-call schedule enabling a responsible marital and family life, shared residencies enabling time off for pregnancies and motherhood in the early childbearing years, and quality refresher courses for women who have interrupted their training.[11]

Program directors will soon have to define their maternity leave policies for women residents and come into compliance with the 1978 Pregnancy Discrimination Law of the Federal Government. These policies will be written as part of the residents' contracts and will be structured in accord with the AMA "Maternity Leave for Residents" study and recommendations.[12]

THE FUTURE

The number of women physicians in the United States is growing. It has been estimated that by the year 2000 it will quadruple. It has been further estimated that by the early 1990s, women will make up approximately 35% of the medical school graduates. With these increasing numbers will come new practice patterns and life styles. The surge in the female physician population means that women will have a new visibility in the health care system. Since New York has the largest number of women physicians nationwide, this state will be the first to see the impact of women in large numbers on the practice of medicine.

Dr Clair Callan, past president of AMWA and a member of the Advisory Panel of the AMA Women in Medicine Project, sees two challenges for those who champion an increased role of women in organized medicine.[13] First, all physicians must be educated as to the importance of joining medical societies if they are to have a voice in the practice of medicine. Second, those women who join organized medical societies must be mainstreamed into committee appointments and encouraged to accept leadership roles.

The real challenge to the MSSNY and the county societies is to increase female membership. If the medical society fails to attract women members, then the membership will gradually decrease, and the MSSNY will lose credibility as the voice for physicians in New York State.

Judith Lorber, PHD[14] (Professor of Sociology at the City University of New York Graduate Center and Brooklyn College), challenged women physicians not to settle for "unsung, necessary, unglamorous Primary Care." In such a setting she sees women as part timers, group practitioners, and competitive with nurse practitioners. They would have no impact on medical practice, no voice in health care policy decisions, and would simply form the bland backdrop of medicine. Instead, she sees the role of women in the future as a "different voice," which can bring to medical practice changes and new directions in many areas that need critical thought, as well as a new ethic of responsibility. These areas include the care of the dying, organ transplantation, genetic counseling, and new reproductive technology.

Historian and philosopher Regina Morantz-Sanchez, PHD, addressed the American Academy of Pediatrics "Woman Leadership in Pediatrics" conference (Chicago, June 1988) and asked the question, "Will the increasing population of women in the medical field change the way medicine is practiced?"[15] She called on women physicians to bring back to the practice of medicine the "humanism" that was lost with the scientific advances of the 20th century. This humanism is what she contends will be the woman physician's greatest impact on the practice of medicine in the future.

In New York State, with its many poor, homeless, drug addicted, and mentally ill patients, and the AIDS crisis, women physicians have a unique opportunity to champion health care reforms that will aid the downtrodden of society. Medicine, influenced by increasing numbers of women, has the potential to be shaped and molded into a more caring profession. In areas of patient care, medical society leadership, legislative reform, and health care policy decisions, the woman physician can bring more empathy, compassion and, most of all, the ability to listen.

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An interview with Nancy E. Gary, MD, a medical school dean

LILLIAN GROSS, MD

Nancy E. Gary, MD, was named dean of the Albany Medical College in 1988. In October 1989, she was interviewed for the New York State Journal of Medicine's *special issue, "Women as Physicians—Women as Patients."*

Lillian Gross (LG): How does it feel to be the first woman dean of a co-educational medical school?

Nancy Gary (NG): To begin, I am not the first woman dean. The first woman dean of a co-educational medical school was Leah Lowenstein, who interestingly enough was also a nephrologist. She was dean at Thomas Jefferson University Medical School a few years ago, in 1983 I believe. She passed away quite tragically a few years ago. There are now two other women who are deans of American medical schools. One is Nydia deJesus at the University of Puerto Rico, and the other is B. Lyn Behrens at the Loma Linda Medical School in California. So I am the third sitting woman dean.

LG: It is still quite impressive. How did you get there?

NG: A lot of hard work.

LG: I believe it.

NG: I followed a traditional academic career. Much of my early professional activity was in teaching, research, and clinical practice. My research led to original contributions to the literature which enabled me to be promoted to the rank of full professor. It was then that I decided I was ready to make a change. I considered medical school administration as an alternative, thinking it would be interesting, and decided to give it a try. I found that I had some natural talent for it, and I liked it. So I have stayed with it since 1981.

LG: Is the field ready for more women to assume positions of power in medical academia?

NG: I think so. Many women have the background,

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Nancy E. Gary, MD, dean of the Albany Medical College.

experience, and skills to assume leadership roles in our medical institutions.

LG: What would you consider to be the major obstacles to the advancement of women in medical academia?

NG: I think that for whatever the reasons, there are certain areas that offer greater opportunities for women than others. You know, most women don't rise to senior academic ranks, *or* to leadership positions in medical school administration. I think some women become disenchanted with the lock-step progression this usually entails. They want to take time off to have a family and nurture that family, and this runs against a clock that ticks away toward tenure. So they decide to leave. Or they find that an academic career is more demanding than they thought it would be, and they can have a better life style in some sort of part- or full-time private practice arrangement, where they have more control over their lives than they do in a medical school position. I think some of this represents a misconception about an academic career.

LG: A misconception?

NG: Yes. I think there are a variety of opportunities within the medical school environment that permit a stimulating and rewarding career. Part-time arrangements or clinician educator tracks are options that might provide a better life style. However, I also think medical schools must review their tenure policies to permit faculty, regardless of gender, to stop the clock to raise a family or pursue some unique experience on a short term basis such as the Peace Corps or public service. I also believe that in some institutions women have got to really be outstanding in order to attain promotion and tenure and thus rise to the senior ranks.

LG: More outstanding than the men?

NG: I think so, in some schools.

LG: I thought those days were fading fast. What makes you say so?

NG: In my opinion women physicians remain under careful scrutiny, probably because we are still relatively few in number. Remember it was only in the mid-1970s that women were accepted to medical school in significant numbers, and the "pipeline" is at least seven years long.

LG: What advice would you give those following in your footsteps?

NG: One way you achieve goals is through hard work, but hard work isn't peculiar to being a physician, and it isn't gender related. To be a concert pianist you have to practice long hours and make personal sacrifices. The same is true if you're a tennis player or a competitive swimmer or anything else. If you want a career in medicine you have to be motivated to achieve it and have a desire to make that kind of contribution to society. In order to rise in an academic institution, you have to know the rules of the organization in which you are working, and you have to continue to make progress towards your goal.

LG: I sense you are talking about gamesmanship.

NG: Gamesmanship or organizational policies. You must know the rules and the players to understand how to be successful in an organization.

LG: Did *you* have to make a lot of personal sacrifices to be able to do it?

NG: I think everyone does, men and women alike.

LG: Did you have to *personally*?

NG: Did I? Yes, I suppose I have. I have been extremely focused. I don't particularly want to go into my personal life, but I think, yes, I *have* had to make sacrifices over the years.

LG: Would you feel comfortable sharing with us some examples of what you remember?

NG: I'm really a very private person and would prefer not to cite examples.

LG: Why do you think you made it when others didn't?

NG: It's a matter of discipline, of wanting to achieve. One fundamental thing is that I really like to work. I derive a lot of enjoyment from work, and so it is pleasurable and satisfying for me. This makes me keep wanting to take the next step, to be challenged by the next opportunity, and see what things I could be successful at. I'm fulfilled by these accomplishments.

LG: Did you have any mentors at all, and do you believe in a mentor system?

NG: I certainly believe in mentors. After all, Socrates taught Plato, Plato taught Aristotle, Aristotle taught Alexander the Great, and he conquered the world, at least as it was known in 330 BC! Mentoring, however, is one area where women may be at a disadvantage.

LG: Who were your mentors?

NG: They were all men. I was very fortunate in that I had some outstanding ones early in my career, when I was starting out in academic medicine as a nephrologist. I had some wonderful people as mentors. In my latest career, Dick Reynolds, who was the dean at the Robert Wood Johnson Medical School, truly wore the mantle of mentor for me and nurtured and shaped my career. I feel as though I am his legacy to academic medicine. Such relationships are very important and I'm not sure why women are not as successful as men at mentoring. It seems that when women come into an organization they often don't seek mentors out. Consequently they are not always told what the rules are and are not guided along the way as most men are.

LG: You have made a number of references to "rules" and knowing the system. Have you any concrete suggestions for how to pick up the rules in any given organization?

NG: Begin by reviewing the bylaws; they are the rules of governance for the institution. Next, attend departmental and faculty meetings so you can learn how business is conducted and who the players are. Soon the establishment's power brokers will become known. Volunteer for committees, because such service will help you to learn the political structure of the institution. Learn to debate and understand the process of compromise. Observe and counsel with those who are responsible for political events after you have learned the issues. Select as mentors individuals with both administrative and political skills—they're inseparable.

LG: I have seen a lot of women myself who just don't know the rules, and get into trouble.

NG: That's right. They don't take the time to learn and observe the organization or they don't seek the help they need. There may not be a person senior enough who is willing to say, "Look, this is what you have to do." In some cases, despite your attempts to advise them, they don't want to know. They don't want to hear.

LG: Are you saying women are stubborn, poor learners, or prideful?

NG: I don't know. Maybe all three! Seriously, most successful women I know have spent so much of their time "going it alone" without help or support, and I think they are just unaccustomed to seeking out a mentoring relationship.

LG: Have you had any really "hairy" experiences in going up the ladder?

NG: Not really "hairy" experiences but rather a constant feeling that I have walked on the edge of a cliff for 25 years. As a woman trying to achieve within the male dominated academic environment, I have had to be careful. If I supported too strongly some issues which might be judged to be feminist issues, I might have fallen off the cliff. If, on the other hand, I *didn't* support some of the issues that it would have been appropriate to support, I could still have fallen off the cliff, because I might have been perceived as not possessing an understanding about women's issues, positions, or whatever. So it is really difficult. I'm not sure if I am being very clear about what I trying to say.

LG: I think I understand. But can we have some examples, please? Not that I'm trying to turn you into a mentor yourself!

NG: A woman faculty member comes to you with reasons to suspect a salary inequity. She has unsuccessfully discussed the matter with her immediate supervisor. She wants you to go over the head of her boss. What do you do? Risk your own position or disappoint her?

LG: I see what you mean. Tell me about grantsmanship. One of the people working with us on this special issue of the *New York State Journal of Medicine* said that she gave up on academia when it was a matter of getting herself a grant.

NG: I'm not sure what she means. Did she lack the creativity to ask the research question? Did she lack the technical "know how" to write a grant? Or did she not want to get her own salary through grants? Some women aren't very well trained in how to write grants unless they have a good mentor. I never perceived that there was any bias there. I may be mistaken, I am just saying that I personally didn't perceive it. But I think that some women are not getting the kind of coaching, experience, and guidance they need to obtain grants. There are, of course, some women who are enormously successful.

LG: Are you planning a course in "grantsmanship" at your medical school?

NG: Yes, and admission won't be based on gender! It will be open enrollment!

LG: Where did you learn the business and managerial skills you need to run a medical school?

NG: On the job!

LG: All on the job?

NG: Yes, I took two brief management courses from the Association of American Medical Colleges, but I think I learned most of it on the job.

LG: The professional managers and deans of programs offering masters degrees in administration might disagree with you, but I agree, some people are just born with it.

NG: Yes, I may be one of those. Perhaps, more important than management training, deans need to be leaders. This is a subject that I do read about. John MacGregor Burns' book, *Leadership*, is a particularly excellent treatise on the subject.

LG: Next question. What changes in the traditional medical curriculum do you plan to implement, if any?

NG: I am very interested in developing a medical curriculum that is more in keeping with contemporary medicine. This would emphasize several things. One is teaching how to care for patients in an ambulatory setting. Most students who graduate don't end up practicing in a hospital, much less in a tertiary care hospital. Most of them practice in communities, and in medical schools we give them inadequate experience in seeing what the practice environment is like. I also think it is important to teach students to practice high quality medicine in an efficient and cost effective manner. This includes how to practice in a health care environment where there is tight management of the health care dollar. These are two important areas that we need to address to make education more in keeping with what the physician actually does professionally.

LG: That makes a lot of sense. Logistically and practically, what plans have you for accomplishing this?

NG: At Albany Medical College we are in the midst of a feasibility study to determine how best we can enhance the ambulatory experiences we already offer students. We are also conferring with representatives of two other schools to develop a special primary care track, perhaps with emphasis on the managed care environment.

LG: Tell me, where do you plan to go from this job? Or haven't you given it any thought yet?

NG: I have no idea. I'm convinced that medical school deans should have a finite tenure. I am not sure I know what that should be. My own life cycle has been to change every seven years. So I think I will be doing this job somewhere between five and seven years, and then there will be at least one more for me. Don't ask me where! I might be interested in going back into the public sector. I spent last year working in Congress, and a public service opportunity might prove interesting. I might also be interested in another deanship, or doing something in foundation work, or some charitable experience.

LG: What were you doing in Congress? What's going on there that women physicians should stay alert to?

NG: I was fortunate to receive one of the Robert Wood Johnson Foundation Health Policy Fellowships. The program is run by the Institute of Medicine of the National Academy of Sciences. After a two-month intensive orientation the fellows work in Congress or the executive branch of government. I worked on the Energy and Commerce Committee, Subcommittee on Health and the Environment in the US House of Representatives. Access to health care, expenditure targets, and the resource based relative value scale are topical and deserve all physicians' attention.

LG: Tell me, do you think things would have been a little bit different for you professionally if you had been saddled with a family and all of that?

NG: Very much so.

LG: Do you feel that women who are leading a traditional, married life style have a more difficult time, or a different kind of time, in academia?

NG: Yes, I think there are women who can achieve as I have achieved. Lyn Behrens and Nydia deJesus I believe are both married. Leah Lowenstein's children happened to have been pretty grown up by the time she took the position. So there are women who have become medical school deans while concurrently raising a family. I don't think I would have been as successful had I had a family.

LG: I know I gave up the academic route when I had children.

NG: A lot of women do.

LG: I just found I couldn't do it. I had my two children in the bathtub one day, and my husband was out on house calls, and they called me from an emergency room with a convulsing child. I couldn't be on call 24 hours a day. No maid, two screaming children, it was too hard.

NG: I think there are some women who can achieve it but I think . . .

LG: They need a super support system.

NG: Absolutely. I am fortunate in that I've had supportive family and friends.

LG: Most of the women I have spoken to find that their careers are really handicapped when they have small children. The housekeepers quit, helpful people aren't around, and it makes a big difference.

NG: Sure. You can't be on call and have small children at home. It's not possible. You can't do it, unless you have somebody else.

LG: And are they hard to find! Before we let you get back to work, any other thoughts you'd care to share with us? Any words to house staff, would-be doctors, or soon-to-be retirees?

NG: A last thought to share? Perhaps a quote from Thoreau's *Walden* would be fitting: "If one advances confidently in the direction of his dreams and endeavors to live the life he has imagined, he will meet with a success unexpected in common hours . . . If you have built castles in the air, your work need not be lost; that is where they should be. Now put the foundation under them." Medicine continues to be a wonderful profession with unlimited opportunities to have a variety of professional experiences within one career. It offers great freedom and that is why I think it is a particularly excellent choice for women.

LG: I don't want to intrude any further on your time. I just want to thank you very, very much.

NG: It's been my pleasure.

History of the formation of the Committee on Women of the Westchester County Medical Society

Toni G. Novick, MD

The Committee on Women of the Westchester County Medical Society was granted permanent status by the board of directors in April 1986. The story of this committee had begun more than ten years earlier.

In 1975, a small group of women physicians issued an open invitation to all of the 400 women physicians living in this county immediately north of New York City. (It was recognized that not all physicians who lived in the county worked there, as Westchester is a bedroom community.) The invitation was to attend a meeting to share mutual concerns by virtue of sex and profession.

Approximately 40 women physicians attended this initial meeting and proved eager to share their ideas. They talked informally about problems of social isolation, conflicts between career and family responsibilities, concerns about sexism, needs for continuing medical education (CME), and more participation in professional societies. From this, a steering committee developed, and a telephone survey followed to provide information on the needs and desires of the 360 women who had not attended the first meeting. Of the 125 women we were able to contact, 75% indicated an interest in attending professional meetings, 65% wanted to socialize, and most were interested in both. We also learned that only 40% were members of any

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county medical society, and only 10% were members of the American Medical Women's Association (AMWA).

With such a large number of women physicians indicating a need for professional and social communication, the steering committee decided to petition the board of directors of the Westchester County Medical Society to become designated as a Committee on Women.

The committee would focus on encouraging greater participation by women physicians within organized medicine; the exchange of professional information such as CME programs; the provision of a voice for women physicians to express their needs and concerns to the society; the attempt to dispel stereotypes and misconceptions about women physicians and publicize women physicians' contributions to medicine; and the need to draw attention to medical issues concerning women, such as reproductive rights and rape.

Although the Committee on Women applied for permanent status within the medical society, the best we could achieve was ad hoc status awarded annually. As far as we knew, ours was the first such committee within a medical society in the country.

During the first five years, the Committee on Women had yearly outreach meetings. These took place at the medical society offices, at the home of one of the participating physicians, or at the New York Medical College. Several sessions at the medical college were well attended by large groups of female and male students and their families. Discussions dealt with postgraduate medical life styles, as well as participation in organized medicine. Several panels and speakers on various topics such as financial planning, premenstrual syndrome, new concepts of the psychology of women, and rape were presented.

The committee, unassisted by the program committee of the Westchester County Medical Society and working autonomously, developed its own yearly programs. These addressed midlife issues for women; sexually transmitted diseases; treatment of breast cancer; geriatric medicine focusing on special needs of women; eating disorders such as anorexia, bulimia, and obesity; health problems in adolescent females, including smoking, planned and unplanned pregnancy, and substance abuse; alternatives in conception and contraception; and plastic surgery, including liposuction and breast reconstruction.

Somewhere around the fifth year, as permanent status continued to be denied, the members of the committee anguished over whether or not it should just regroup, perhaps as a branch of the American Medical Women's Association. Although consideration was given to maintaining the committee as a presence in both societies, fear was expressed that this would water down the effectiveness of each group and perhaps decrease the participation of women whose time was already very limited. The dual role was chosen. An AMWA chapter was formed and now has more than 60 enrollees, as well as at least 52 medical student members. During 1982, the two groups jointly ran a very successful workshop on leadership for women in medicine with participation by practitioners, medical students, social workers, administrators, nurses, and professors in medical schools.

At this same period in the committee's history, the county medical society president, while continuing to deny the committee permanent status, stated that he would like to see greater participation of the women physicians on other committees of the society, and he asked the committee's help in encouraging this. In a relatively short time, each committee member also enrolled in a second committee, and we immediately became more visible and vocal. Today, every committee of this county society has one or more women on it, as does the editorial board of the society's journal and the board of directors.

Each year, the committee requested permanent status, and each year the request was denied for a variety of reasons. In one rejection letter, the increasing participation of the women on all of the committees was cited. The belief was expressed that this reflected the interests of women and that there was therefore no need for a special committee. The committee itself strongly disagreed with this thesis. It thought that without this committee highlighting special issues of interest to women, participation by women might no longer continue as actively.

Once the quotas on women in medical school were lifted, women constituted one third of the medical students and novice physicians in a very short time. This did not mean that women physicians would automatically progress along the pipeline and show up in similar percentages as heads of major services, deans of medical schools, or heads of hospitals and large medical centers. They are confronted on the one hand with the need to be recognized as women of achievement, and on the other hand to be recognized as people of achievement. Women who wish to become part of a male inner circle feel the need to overcome men's suspicions of women as women. By creating a committee on women, the women physicians who embarked on that journey hoped to offer personal support through networking and communication, action on health issues—especially those pertaining to women—and information on career options and development.

It took nine months to become an ad hoc committee and ten years to become a permanent committee, albeit a special committee and not a standing one, of the Westchester County Medical Society. Today there are more than 500 women physicians living in the county, and the percentage of women members in the society remains the same. However, they are more active and continue to proselytize for more women members. One former committee member is president-elect of the society this year. There can be no question that without the committee, the society would never have sponsored a program on midlife issues for women, and would never have had as many active women, or a woman as president-elect.

Women physicians need other women to offer a balance to the subtle "put-downs" that men sometimes use to protect the boundaries of their collegial brotherhood. Women physicians need other women to serve as active role models, mentors, and sponsors. Women physicians need other women in crucial gatekeeping positions, where they can search out talented, able women and support their bids for staff positions and heads of committees and services. Women physicians need other women as colleagues and professional friends, just to exchange information about running an office, handling patients, or moving into a new field. Women physicians need the support of other women to make significant changes in medical education, health care delivery, and research priorities.

All physicians realize that the profession is changing. The grimness of malpractice, third party interference, HMOs, PPOs, DRGs, and the rest of the alphabet soup has led to decreased satisfaction with medicine as a career, a diminished entrepreneurial image, and decreased participation in organized medicine. All this portends a radical change in the image of the doctor and the way that medicine is practiced in the United States. This change in medicine coincides with the increased number of women practicing medicine in the United States. Men and women physicians are all in the same boat in this sea of change. The history of the Committee on Women shows that women physicians must act in order to have a substantial impact on the design and the character of that boat and the direction in which it will sail.

Parenthood and practice

ADELAIDE T. SCANLON, MD

My daughter has just delivered her third daughter, Carolyn, my 28th grandchild. How lucky can Tom and I be! I reminisce over the last 44 years. Then I think of the statistics today of women in medicine; 63% of women surgeons are childless. Do they know what joys in life they are missing? I wonder how many remain childless because of the fear of not being able to be a perfect mother because of a career?

Work need not interfere with caring, responsible parenting. Tom Sheehy and I established our priorities early on with the knowledge that there would be times that exceptions would have to be made. Our first priority was to each other, the second to the children, and the third to our careers.

At first we lived in New York City, but soon after our first child was born, we felt suburban living would better suit our needs. We moved to Roslyn, Long Island, and for one year I commuted to my New York practice and clinics in pediatrics while starting a practice on Long Island. This was a very hectic time. Tom was still in the service, stationed at Mitchell Field, and then, on discharge, was starting a solo practice in obstetrics and gynecology. When my son John was born, I decided to give up practice in New York City. Practice increased more rapidly on the Island when people realized I was available all the time.

A child care person was mandatory, and my own was heaven sent. She lived in the Bronx, and when I lived in the city, her daughter "baby-sat" for me during office hours and clinics during the summer of 1949. The mother, Mrs McCormick, provided back-up service and took over when Barbara returned to school. In November, when we moved to Long Island, she said she would commute (one and a half to two hours each way) to help me unpack. "Mac," as the children called her, never finished unpacking. She stayed on to help with my brood of four boys and six girls for 25 years.

Finding a "Mac" is essential for a woman physician's career. She has to be loving, intelligent, and willing to carry out your wishes on child care, and she must be treated with respect and love by you; in turn, your children will respect and love her. That is the magic ingredient to keeping your help. Mac was included in all of the children's activities and treated as a family member. Tom and I took over the care of the children evenings and weekends.

We found it essential to have specific times off, and to find good coverage for our patients. Tom entered into a partnership that lasted many years, and I arranged coverage with other women physicians in the area, all practicing pediatrics in home offices. On a first visit, patients were instructed about our routine of early morning phone hours, vacation time, and coverage. They were told that these physicians were also on call for my own children.

Take vacations and time for family. You can always obtain coverage for patients, but the day you have a heart attack, there is no one to cover for a mother or a wife. Second to child care comes house care. Are you a better wife or mother because you clean house or do the wash? Of course, some of you may enjoy household chores, and some of these can be used to teach children responsibility and independence. My children could all use the washing machine and dryer to follow his or her laundry through at age ten. This made for one unusual plaint: "When is the machine available for me?"!

Human nature being what it is, interesting stories could be told. I remember, for example, when Marie, after a few grinding hours of studying, remembered she needed clean gym uniforms for the next day. At 1:00 AM she came and knocked on our door to ask, "If I put my things in the laundry now would they be ready in the morning?" Of course her father, like a fairy godfather, put them in the machine and one hour later awoke again to put them in the dryer. He also folded them in the morning in time for school. He said, "It's a mighty poor house that can't afford at least one princess." We had many laughs about such incidents. One fall, Jim had faded bleached jeans before they were the fad. I questioned him about it and he said, "You know the Clorox® ad where the cat pushes the plastic bottle of Clorox® off the shelf and it does not break? Well, it does! I experimented and it broke and splattered."

Some household activities can become family affairs,

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such as getting dinner. All the children learned to cook, and cook creatively and well, and I would tell the boys I would give them excellent references that they would be good husbands. (P.S. They are!)

Continuing medical education course were popular with the family, as we would take one child with us. There were guided tours of Boston with Tom while I was at "Advances in Pediatrics." Eileen saw Montreal when Tom took an oncology course. Margaret had an in-depth tour of Washington and Williamsburg during another OB/GYN seminar. Family tours were generally long weekends in New York in spring or Washington at Easter, as our house in Vermont was too muddy for most springtime activities.

In December 1961, we bought the old farm in Vermont. This decision came about after we spent a wonderful Christmas vacation at the Lake Placid Club. The whole family was anxious to return yearly, but we realized when we checked out that what seemed to be a down payment on ownership was only an eight-day bill for 13 people. The next year we found our 14-room house on 75 acres of woods and pasture in Vermont with the mountains and a lake nearby, at a more affordable rate. This has been our vacation spot up to the present, and the grandchildren love it as much as their parents did, in all seasons.

In summer, after eight weeks of local day camp, we would spend two weeks of relaxing, swimming, boating, and a ball game or two. At this time the children could bring a friend and we often had a great time with as many as 30 people up there. (We had no television!) One of my women colleagues, taking a leaf out of our book, said to me this past fall, "We are doing everything with our children and taking them with us. I never realized that it could be so much fun!"

Private practice gave me flexibility. I could pick and choose my time off within reason. If I needed two hours to attend a school play, I could arrange appointments. If I was teaching first aid to the Girl Scout troop, I worked a little later. If dinner was late, the children learned to adjust, and they would start some homework or take a nap. (We rarely had dinner before 7 PM. The boys commuted to high school in New York City and the train arrived in Roslyn at 6:30 PM.) On Fridays we made a family affair in winter and went to the basketball games.

Tom and I have had 44 full years. Now eight of the ten children are happily married, and we still have wonderful times together. They enjoy dinner for all here on Sunday and take vacations together in Vermont. Occasionally, they take tours to Disneyland with two or three families together. Eileen and John are with me in a pediatric practice. Eileen's husband, Carlo Annese, MD, is practicing internal medicine across from our office in a professional building, and one of their classmates, James Walker, MD, a radiologist practicing ten miles away, is married to my daughter Marie. We practice medicine with our priorities in mind. There are no evening hours except "on call nights," every other night and every other weekend. In spring and early summer, Tom took Saturdays off to coach Little League while his partner played golf on Sundays.

Having work, a supportive husband, and a wonderful family has given me a very fulfilling life. I do not play afternoon bridge, play golf, or join clubs, and we did not travel until recently, but I would not have changed a moment of it. Parenting cut into our income but what an exchange! We have had all the more to love.

Jane V. Williams, MD (1924–1989): An appreciation

NAOMI R. BLUESTONE, MD, MPH

I am sitting here looking at Jane's curriculum vitae, and it reads like that of a stranger. It is so impressive, so organically correct, as informative as an official tractate issued by the Mayor's Task Force on the Correctness of Resumes. How methodically it tracks the advancing career of a dedicated physician, records her training and professional positions, reminds the reader of her selfless involvement in community affairs. A good doctor's life in synopsis! But who would have suspected that underneath all that propriety was a Janie?!

Her CV begins at Hunter College High School, that famous breeding ground of bright young things, and a bachelor's degree from the parent institution in 1945. It notes her removal to the nation's capital to obtain her medical degree from Howard University (but makes no note of the fact that the budding physician packed up her dirty laundry every Friday night and mailed it home to be laundered and returned fresh on Monday by the family help!). It duly records her internship and residencies at Sydenham, Lincoln, and the old Willard Parker hospitals. It furthermore says here that she did three years in the private practice of pediatrics in Philadelphia, at which time she also made her first entry into the field of public health. She then returned to New York City to begin a 20-year association with the Bedford-Williamsburg HIP group. Six years later, she came to the City of New York Department of Health, to undertake a second specialty training that would culminate in her master's degree in public health from Columbia University in 1965, and her certification by the American Board of Preventive Medicine in 1968. It is at this time that I entered her curricu-

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lum vitae, although this minor fact, too, was not recorded.

"Let's go have a lunch," she proposed to the new resident, struggling to adapt to the big city, which was unwieldy and tempestuous even then. "You want some lunch? Let's go have a lunch!" A firm hand propelled me by the elbow, and I ordered my first grilled cheese on rye at Squire's, while a gimlet eye and a smooth drawl inquired what I thought of the Health Department. Although the question was thoroughly professional, and characteristic of her direct approach to the satisfaction of her curiosity, I never knew her to draw an artificial line between personal and professional. She made it clear from the outset that this senior colleague of the same sex, with all the instincts of a large, whiskey-bearing dog, considered me, a novice climber in the field of health politics, her personal mission. She was going to be responsible for the education of Naomi Bluestone.

Janie wasted no time. I recall one specific day when we had attended one of those exceptionally dull administrative meetings at some uptown site, emerging at the ambiguous hour of three in the afternoon. Ever the grind, I was prepared to head back to my desk for what was left of the day. Jane was scandalized, absolutely horrified, that anyone could interpret the Puritan ethic of work so literally. "We are going shopping," she announced firmly. "We will not go back to the Health Department. We will go to Bergdorf-Goodman. They are having a sale, and they are open late tonight." Propelling me firmly by an elbow that should have become bursitic by now, she hauled me like a recalcitrant donkey into a perfumed salon whose salesladies made me want to flee in abject terror back into the bas coutured middle class to which intimidated and impoverished medical students naturally gravitate.

"This will do nicely," she said, moving swiftly back and forth from the rack, and pushing the new me in front of the mirror. I looked so good I had to pinch myself. "Try this one, too," she said, looking over her black-rimmed glasses, and tossing a green poie de soie in my direction. Again I thrilled, only to immediately suffer the true obsessive's decisional dilemma. "I don't know which one to buy," I wailed, in genuine anguish. "Take them both," she replied, with the insouciance of the indulged nouveau riche.

Take them both! It was probably the first time in my life that anyone had said, "Take them both." In one bold, and I'm sure calculated stroke, Janie introduced me to the pursuit of hedonism, a worldly grace totally absent from my upbringing. I was changed forever. We left Bergdorf's that day with 11 dresses, a coat (a princess line pink silk with a bow at the neck!), a suit with matching blouse (primrose linen!), and a bill of well over a thousand dollars (in those days!). Janie smiled her bemused, Saint Bernard smile, shook a few drops of whiskey over the fainting climber, and breezily propelled me through the rotating door. "Have them sent to Dr Bluestone when the alterations are completed," she announced to the ecstatic saleswoman, as she hailed a cab (my first). Singlehandedly she had transformed this workaholic drudge into someone who was beginning to understand in some inchoate way the concept of "having everything."

Janie took great pride in her own appearance, and was always well tailored in classic feminine suits cut by a dressmaker to conceal her figure faults. She looked much younger than her "stated age," perhaps because she made time for personal care, a luxury unheard of in medical school. (She did her best to get me from loafers to heels, but never succeeded.)

She wasted as little moral anguish over her work as she did on apparel. She thought it extremely foolish of me to waste too much time studying for my Board examination, when most of it was all that radiology and aerospace nonsense that I would never look at again. Why not get the old tests and just memorize them? After all, those elderly gentlemen who made up the exams were probably too lazy to completely research and rewrite them every year, no? "You will pass, and it doesn't matter by how much," she said. "Now let's go have a lunch." It was my turn to be scandalized but I did as she suggested, and passed as handily as if I had sacrificed sleep on the altar of multiple choice. (I wouldn't have been caught dead doing that in medical school!)

Jane made time for fun. She whisked me away to her oceanside vacation home in New Jersey, introducing me to the sybaritic pleasures of washing sand off with a shower before getting dressed. Although her big and handsome fellow Bill was soon to go his separate way, she encouraged me to spend more time dating. Not that she was a conventional woman herself! When she met Ronnie Williams, the big love of her life, whose death a year ago undoubtedly served to hasten hers, she gleefully reported that there was no need to marry him, since (by sheer coincidence) they both had the same last name anyway. To this day, I don't know whether or not she ever stood up with him in front of a preacher.

When my biological clock started ticking more audibly, an invisible cuckoo popping out periodically to upset me, she reminded me that poo! who needs children anyway. She was ten years my senior and had no intention of staying at home to raise children. I suspect the Saint Bernard was deliberately "putting me on," but it was helpful to meet a woman who could at least joke about it. Today's attitudes about the barrenness of women without children are infinitely more civilized, in part due to the courage of women like Jane.

Although Janie could be a provocateur par excellence, a devil's advocate on any subject from hypertension in young black males to the stagnation of "Central Office," she was above all else kind and motherly. Her bemused, slightly quizzical smile, even when she was good-naturedly teasing, would quickly give way to concern if anyone was in trouble. Her friends reported daily phone calls when their husbands were ill, or their children in some scrape. A colleague of mine, a departmental chair and a full professor, admitted to dumping on the still practicing pediatrician all the details of his malfunctioning sacroiliac. "Did she have good advice for you?" I asked. "Absolutely," he replied cheerfully. "She told me to go out and get myself a copper bracelet!" "And did you?" I asked. He started pawing the floor. Jane was a convincing woman.

I think we could have been forgiven for dismissing Janie initially as a "Black American Princess," since this was a role she deliberately cultivated. But often her brow would furrow and the other Jane would emerge. The other Jane was a card-carrying, board-certified, bona fide idealist, who fought for principles, made her voice heard, offered a presence on countless battle lines, engaged in vociferous argument, and stayed in working harness until virtually the end of her life. It was not until I reviewed her lengthy and solid curriculum vitae recently that I realized how extensive her sphere of influence actually had been, and how thoroughly fraudulent had been her carefully cultivated persona of the idle dabbler in peds and public health.

Jane was a working member of the American Medical Association, the Medical Society of the State of New York, the Kings County Medical Society, the National Medical Association, the American Public Health Association, the Public Health Association of New York City, the American College of Preventive Medicine, the New York Association for Ambulatory Care, the Physician's Forum, and the Provident Clinical Society. She contributed actively to all of them. She served as president of the Public Health Physician's Association of New York City. She was treasurer and trustee of the Welfare Fund, and a member of the Doctor's Council at the Health Department. She was a member of the Fort Greene Community Council and an active member of many community boards, whose nocturnal gatherings she faithfully attended.

All of this was voluntary activity, performed in her "spare" time. There were no breaks in her professional career. Before her appointment as District Health Officer in Fort Greene, she worked at the Health Department's Central Office, and served as Director of Professional Education there for four years. No public health professional who crossed her path escaped without her "shpiel" about the importance of graduate education, the master's degree in public health offered by Columbia. In conjunction with her clinical activities outside the Health Department, Jane served on the staff of a number of Brooklyn hospitals, including Kings County Medical Center, Downstate, the Jewish Hospital of Brooklyn, Unity Hospital, and Brookdale Medical Center.

She also taught throughout her professional career, for 15 years as instructor of pediatrics at the Downstate Medical Center, State University of New York, and also as instructor in the Department of Family Practice. She was appointed Clinical Assistant Professor of Preventive Medicine and Community Health in 1979, and held this post until her death.

As a member of the wealthy and privileged elite, Jane could have lived and worked anywhere she wanted. She chose to live in an apartment on Willoughby Avenue in Fort Greene, just a few blocks from the Health Center whose District Health Officer she eventually became. Although she inevitably dabbled in the Florida sun traffic, she came back home and did not leave again. Although she could have taken more prestigious jobs elsewhere, she knew where she wanted to be. Downtown Brooklyn was her people, her neighborhood, her home. How fortunate for Brooklyn, which lost so much else!

Jane was not well. She had a thyroid that periodically went out of whack and made her eyes pop out and her body bloat up with frightening amounts of fluid. She had high blood pressure, cardiovascular irregularities and progressively worsening scleroderma. She worked through a lifetime of symptoms and treatments that would have halted a lesser person with a vitality which made her death at 64 seem an insult and an ingratitude.

Janie will be remembered as a woman who built friendships, solidarity, networks of people who cared about each other. She was warm, loving, generous, helpfully critical, acerbic when necessary, and productive of challenging ideas that had been purged of all maliciousness. She was an iconoclast who loved to shake up people and their ideas, but left no hurt feelings or cutting edges. She was the kind of woman you could call on a Saturday when you were blue, pulling your earrings off and switching the receiver from one red ear to another until your throat ran dry. She was also the kind of woman you could call late at night when a community board member was threatening mayhem, or your driver failed to show up to take you home.

But most important, I believe that Dr Jane V. Williams was a historical figure. Jane belonged to the transitional generation of women physicians, the living bridge between the young women of today who are thronging into medicine, and the few pioneers who fought their way into a male preserve. The old-timers devoted their lives to the ascetic ideal, service to science and mankind through self-abnegation. They devoted all to medicine, and willingly sacrificed what else life had to offer. Jane's generation tried to provide a more balanced life for themselves, thus enabling larger numbers of women to consider medicine as a career. Many women have become thoroughly competent physicians who would not have been willing to give up what yesteryear demanded. It remains for the young women of today to fight their own modern-day demon, the threat of too great a preoccupation with "having it all."

On balance, my friend Jane made a good thing of her life, and I am grateful to have been a small part of it. So would say her friends Vashti, Georgia, Pearl, Mabel . . . her many colleagues . . . and all those black hypertensive young men who never knew how much of her life she dedicated to them.

Unexpected, unattended deliveries

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JERROLD S. SCHLESSEL, MD

The pregnant woman commonly receives instructions from her obstetrician concerning the need to acquire adequate nutrients,[1] the importance of avoiding drugs and alcohol,[2] and the value of obtaining timely prenatal care.[3-7] However, what to do if the woman is unexpectedly forced to deliver alone or with an untrained assistant is a topic not usually discussed. This is surprising given the fact that approximately 9,000 of the four million deliveries in the United States in 1988 occurred unexpectedly, out of a hospital, and without the assistance of a physician or nurse-midwife.[8] Lack of specific education on unattended deliveries may contribute to central nervous system impairment and/or death of the infant. Burnett et al[9] reported that unplanned home deliveries attended by untrained personnel have ten times the national mortality rate compared to in-hospital deliveries—ie, 120 deaths per 1,000 live births. Schramm et al[10] support this concept, and report mortality for unplanned home births to be 18–20 times the rate for hospital births. Undoubtedly, lack of knowledge of the labor and delivery process, lack of knowledge of what to do in the emergency situation, panic, and inexperience all contribute to poor outcome. The following three cases illustrate such outcomes.

CASE REPORTS

Case 1. A full-term infant was born to a 25-year-old gravida 1 para 0 mother, who had received prenatal care during her pregnancy. The mother was in active labor while in transit to the hospital and reported that the baby was partially born 15 minutes before arrival at the hospital. On inspection in the emergency room, the infant was compressed in the mother's dungarees and underpants. The infant was brought to the Neonatal Intensive Care Unit (NICU), unresponsive, pale, flaccid, cyanotic, with no respirations, and with a heart rate of 60 beats/min. The infant was intubated and resuscitated; however, bradycardia lasted ten minutes, after which the heart rate rose to 140 beats/min. A chest film showed a tension pneumothorax, and a chest tube was inserted. The patient was noted to have had a left-sided focal seizure, and phenobarbital and phenytoin were administered. The initial arterial blood gas findings on 100% FiO_2 were pH, 7.18; PCO_2, 16 mm Hg; PO_2, 187 mm Hg; HCO_3, 6 mEq/L.

Cultures were taken, and the infant was started on ampicillin and gentamicin. Physical examination showed retinal hemorrhages, nonreactive pupils, no spontaneous respirations, flaccidity, no glabellar reflex, and clonus. During the hospital course, the infant required 100% oxygen. Blood gases were monitored, and appropriate respirator adjustments made. Seizure activity appeared to be controlled, but twitching was noted with increased tone in all extremities. The infant's neurologic status remained grossly abnormal. On day four following delivery, the infant became bradycardic, did not respond to resuscitative measures, and died.

Case 2. A full-term infant was born to a 38-year-old gravida 8 para 7 mother who had received no prenatal care. The infant was born in the mother's underpants in the parking lot of an outlying hospital. When the garment was removed, the infant was noted to be covered with maternal stool, meconium, and blood, and to have a weak cry. The cord was cut, and the infant was wrapped in blankets and brought to the emergency room. Shallow respirations were noted with periods of apnea. An endotracheal tube was inserted for a short time; after its removal, the infant was placed in an oxyhood.

The initial laboratory studies were within normal limits. At the infant's first feeding, his suck was poor. Twenty hours after birth, the infant was noted to be cyanotic with curled toes and clenched fists. This episode lasted for 90 seconds and ended in a brief period of leg tremors. The infant had multiple seizure episodes and was treated with phenobarbital. A full sepsis workup was performed, and the infant was transferred to a level III nursery. A head sonogram was obtained and showed a Grade IV intraventricular hemorrhage. Follow-up head sonograms showed dilation of the lateral ventricles and partial liquification of the hemorrhage. Multiple lumbar punctures were performed to decrease intracerebral pressure. The infant's head circumference increased, and a ventriculoperitoneal shunt was placed when the infant was six weeks old. A follow-up CT scan showed a decrease in the degree of dilation of the lateral, third, and fourth ventricles. The infant's neurologic examination showed poor head control and muscle tone, which improved slightly during the infant's hospital stay.

Case 3. A full-term female infant was born to a 23-year-old gravida 1 para 0 mother who had received prenatal care during her pregnancy. In transit to the hospital, the baby's head crowned, and the assistant pushed the baby's head back into the vaginal canal to await arrival at the local hospital to complete the delivery. The infant was examined by a physician, and her condition was stable. Arrangements were made to transport the mother and infant via ambulance. On arrival at the tertiary nursery, the infant was cold, mottled, and gasping. Vital signs were as follows: temperature, 33°C; heart rate, 110–120/min; blood pressure, not recordable. The pulses were not palpable, and heart sounds could not be auscultated. The infant was moved to the NICU, where intubation and ventilation were instituted. Initial arterial blood gas results were pH, 6.99; PCO_2, 19 mm Hg; PO_2, 93 mm Hg; HCO_3, 4.9 mEq/L. Sodium bicarbonate was administered. Repeat arterial blood gas findings were pH, 7.05; PCO_2, 17 mm Hg; PO_2, 75 mm Hg; and HCO_3, 4.8 mEq/L. The infant was given two pushes of 5% salt-poor albumin. The blood pressure was still not recordable, and the infant was started on dopamine and dobutamine, with no response. Epinephrine and atropine were also administered. The infant was not responsive to resuscitative measures and died. An autopsy revealed a contusion of the second cervical vertebra and a subarachnoid hematoma.

DISCUSSION

Emergency delivery while the mother is at home, en route to the hospital, in

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a parking lot, or outside the confines of the obstetrical service is relatively rare. The overwhelming majority of emergency deliveries performed by untrained personnel outside the health care environment result in a healthy mother and infant. Occasionally, however, infants born in emergency situations suffer central nervous system damage and/or death. The cases reported here illustrate that during an emergency delivery, infants may be left within the mother's clothing for a prolonged period of time, or may be impeded during the delivery process.

Women who seek prenatal care, as in cases 1 and 3, have close contact with their obstetricians. Prenatal visits afford excellent opportunities to educate expectant mothers and their labor partners concerning emergency labor and delivery.[3] How to recognize when labor starts, how to time contractions, and what to do if the membranes rupture should be part of the standard prenatal curriculum of the pregnant woman and her significant other. In cases of emergency delivery, women should be specifically counselled to allow labor to proceed naturally, to remove undergarments and all clothing below the waist, to allow the baby to emerge without hindrance or pulling, to allow delivery of the baby to occur on as broad and flat a surface as possible, and to lift the baby from between their legs after delivery to prevent asphyxiating and/or crushing the baby. If the baby does not breathe spontaneously, the mother should be counselled to remove debris from the mouth with a finger-sweep. The baby then can be given mouth-to-mouth resuscitation, if necessary. Such instructions might have been beneficial in cases 1 and 2.

How to educate women who do not receive prenatal care as to what to do during an emergency delivery poses a more difficult problem. Instructions similar to those listed above could be presented in public places, ie, posters in buses, trains and post offices, articles in newspapers and women's magazines, announcements in houses of worship, messages on radio and television, etc. Such information could provide the knowledge required to handle the emergency. Specific step-by-step instruction may be necessary for some women.

Panic may result in actions that seem unreasonable when reviewed at a later date, for example, failure to undress. Simple, easy-to-follow, step-by-step instructions reviewed by the expectant mother and her assistant during pregnancy and prior to the emergency delivery offer the best hope of avoiding a panic reaction.

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Sarcoidosis of the breast coexisting with mammary carcinoma

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Involvement of the breast by sarcoidosis is uncommon. Of the 20 cases that have been reported,[1-15] systemic sarcoidosis preceded or followed the breast mass in 15 cases (Table I). Sarcoidosis of the breast coexisted with mammary carcinoma in three cases.[5,8,9] We report an additional case, the fourth, in a young woman who had been treated for pulmonary sarcoidosis and subsequently presented with a breast mass.

CASE REPORT

A 36-year-old black woman was admitted to the hospital because of a mass in her left breast. A diagnosis of pulmonary sarcoidosis had been documented by lung biopsy three years previously at another hospital. Steroid therapy had been administered for one year but was discontinued because of side effects.

The breast mass, located in the upper inner quadrant, measured 3 × 2 cm. It was irregular, firm, freely movable, and was not tender. There were no palpable lymph nodes. Laboratory values were within normal limits. Pulmonary function tests revealed minimal obstructive and restrictive disease. Radiologic examination of the chest and a computed tomographic scan revealed bilateral lung infiltrates and bilateral lymphadenopathy. A bone scan was negative. An excisional biopsy of the breast mass was followed within two weeks by a left modified radical mastectomy.

Sixteen months later, the patient was readmitted for excision of a nodule in the left chest wall located at some distance from the surgical scar. At the same time, a nontender, irregular mass in the right breast accompanied by right axillary lymphadenopathy was found.

Pathologic Findings. The excisional biopsy of the left breast consisted of a 4 × 3.5 × 1.5-cm hard mass with gritty cut surface and ill-defined borders. Microscopic examination revealed invasive duct carcinoma. Noncaseating epithelioid granulomas were scattered within the tumor and the adjacent fat (Fig 1). The specimen of the left chest wall consisted of an ellipse of skin and subcutis which contained several firm nodules up to 0.4 cm in greatest dimension. Microscopic examination revealed recurrent invasive carcinoma that was similar to the primary tumor. Discrete epithelioid granulomas (Fig 2) were present contiguous to, rather than intermingled with, foci of carcinoma.

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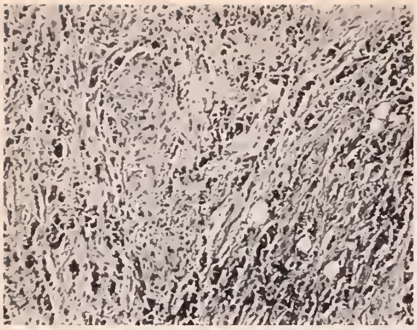

FIGURE 1. Invasive duct carcinoma surrounding three poorly formed epithelioid granulomas (hematoxylin-eosin stain).

The right breast mass (3 × 3 × 1 cm) consisted of two lymph nodes embedded in fatty breast tissue. The right axillary lymph nodes measured up to 3 cm in greatest dimension. The breast parenchyma and the lymph nodes contained numerous epithelioid granulomas. Stains for fungi and acid-fast bacilli were negative. No carcinoma was present.

DISCUSSION

Granulomatous mastitis has been described in a variety of conditions, including tuberculosis, leprosy, fungal diseases, parasitic infestation, malignant lymphoma, and giant cell reaction to foreign bodies. Granulomas may occur in lymph nodes that drain carcinomas and within the tumors themselves, probably representing a reaction to necrosis or tumor products.[16–19] The diagnosis of sarcoidosis remains one that must be based on the exclusion of other conditions.

Because sarcoidosis is usually accompanied by various immunologic disturbances, a high incidence of associated malignant tumors may be expected. For example, in a series of 2,544 patients with pulmonary sarcoidosis, the incidence of carcinoma of the lung was three times higher, and the occurrence of malignant lymphoma was 11 times higher than in the general population. In contrast, the incidence of other malignancies was not increased.[20] The association of breast cancer and sarcoidosis, pulmonary or mammary, is uncommon.

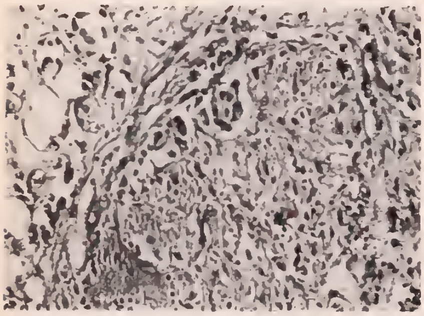

FIGURE 2. Discrete florid granuloma. Note peripheral clusters of tumor cells within the granuloma (hematoxylin-eosin stain).

TABLE I. Mammary Sarcoidosis With or Without Associated Cancer of Breast

Authors	No. of Cases	Age (yr)	Mammary Carcinoma	Systemic Sarcoidosis
Azzopardi[1]	1	Not stated	—	Not stated
Banik et al[2]	1	28	—	Lymph nodes
Bodo et al[3]	1	48	—	Not stated
Fitzgibbons et al[4]	2	65	—	Lymph nodes
		49	—	No
Gansler and Wheeler[5]	2	31	—	Lymph nodes, lungs, eyes
		28	Medullary	Lymph nodes, lungs
Geschickter[6]	2	Not stated	—	Not stated
	2	Not stated	—	Skin
Gupta and Chatterjee[7]	1	42	—	Lungs, lymph nodes, skin
Haagensen[8]	1	53	Type not stated	Lungs, spleen, liver, lymph nodes, tongue
Prior[9]	1	59	Medullary	Lymph nodes, spleen
Raju and McShine[10]	1	60	—	Lungs
Reitz et al[11]	1	39	—	Skin, lymph nodes, liver, lungs
Rigden[12]	1	22	—	Lung
Ross and Merino[13]	1	29	—	Lymph nodes, lungs
Scadding[14]	1	43	—	Lungs, lymph nodes
Scott[15]	1	38	—	Lymph nodes, eyes, lungs
Present case	1	38	Infiltrating duct cell	Lungs, lymph nodes

Mammary sarcoidosis was first alluded to by Strandberg[21] in 1921. The patient described by Strandberg had generalized sarcoidosis and bilateral breast involvement. There are 20 reports in the literature of cases in which sarcoidosis affected the breast (Table I). Sarcoidosis was confined to the breast in only one case,[4] and no reference to systemic involvement was made in four other cases.[1,3,6]

In 1952, Prior[9] reported the first case of sarcoidosis of the breast coexisting with medullary carcinoma. Gansler and Wheeler[5] reported another case of medullary carcinoma of the breast and sarcoidosis in a 28-year-old woman. This patient later had a second mass in the contralateral breast, which proved to be sarcoidosis; no tumor was present. Sarcoidosis of the breast coexisted with mammary carcinoma, type unspecified, in a patient who, at autopsy, was found to have generalized sarcoidosis involving the lungs, lymph nodes, spleen, and tongue. There was no residual tumor.[8]

In the case reported here, sarcoidosis of the left breast developed after pulmonary sarcoidosis had been diagnosed. The left breast also contained an invasive duct carcinoma. Sixteen months later, the patient presented with recurrent carcinoma of the left chest wall, which again was associated with sarcoid granulomas. There was also sarcoidosis of the right breast and right axillary lymph nodes.

When a diagnosis of sarcoidosis of the breast is made by fine-needle aspiration biopsy,[3] special consideration should be given to the possibility of a coexisting carcinoma, which may have been overlooked as a result of a sampling error. In such cases, excisional biopsy is strongly recommended.

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ON BEING PICKED UP

When people ask me how I met my husband, I tell them that he literally "picked me up." One day during my medical school years, I was leaving the campus after classes to find that someone had parked bumper to bumper behind my Volkswagen Bug. With cars on either side, the only way out was to wait for the offending driver to show up, or to drive forward onto a sidewalk. Since the sidewalk was level with the parking lot, that was not the problem. The problem was that on the other side of the sidewalk was a descending concrete stairway that would have made for a very bumpy ride. I decided to give it a try anyway, and began a tedious back and forth maneuver with the car, trying to negotiate a turn to the left.

While doing this, I noticed two tall, athletic young men observing my efforts. Rather irritated by their bemused expressions, I shouted at them that they could at least let me know how close I was getting to the stairway, and if it looked like I was going to make the turn safely or not. They both shook their heads slowly to the negative. They then exchanged a look, and without a word of warning each one took hold of either end of the Bug. They lifted up the car (startled driver included), and with little bounces turned the car so I could safely drive away.

Well, what could I do but offer them a ride somewhere if they needed it? The one who accepted still looks after me and my 1963 VW bug.

ELLEN YANKAUSKAS, MD

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Responses to "Parenthood and practice"

TO THE EDITOR: I read with great interest Dr Scanlon's[1] wonderful account of the joy she found in family life. My family was also large. I have five sisters and two brothers, with me as the oldest sister. Dr Scanlon's advice to take time with children, incorporate them into your activities, teach them responsibility, etc, were givens in my childhood. Just as Dr Scanlon sees her adult children flower, I see the joy my parents take in myself and my siblings. The isolation of an aging person without family is a common sight on the hospital floors. I have no questions as to the fate I would prefer. Unfortunately, while much of Dr Scanlon's wisdom prevails, I must presume times have changed over 44 years.

The really essential issue is child care. I have many concerns about letting a stranger have so much influence in raising my children. How does one evaluate a person's values, mental health, and abilities in parenting in such a brief period, and when the applicant has reason to make an impression? My husband and I had years to know each other to make such a decision. Years to decide on the father and a few hours to decide on a major caregiver? That scares me. Besides, I really do not want an outsider in my home. Dr Scanlon talks about making the caregiver a part of the family, loving her and including her. In the same paragraph, she refers to the person as the "help." Throughout the remaining paragraphs of valued family memories, "Mac" is not mentioned once. To me this demonstrates that the image of the caregiver as part of the family may not be how people really feel. Unless you are unbelievably lucky, the person in your home is likely to have different values and attitudes. Of course, children need to have exposure to different ideas. But I want them to have a consistent environment in their youngest years that will prepare them to be open-minded once they mature.

Let's assume that somehow my husband and I could come to terms with the fears and concerns that come with child care. Now it is time to start looking. It is my belief that even this is harder than it used to be. Every doctor who has been in practice for a number of years tells me how much harder it is to get competent secretarial help. The nursing shortage is well known. In reflecting on my contemporaries with small children, only one was really happy with her "housekeeper," who cared for her son. One friend's caregiver actually had a psychotic break while alone with the children. I think that the really mature, competent women who used to fill these roles are now going into professional careers, their own businesses, and so on. Child care may be the most important function our society must provide, but it is unquestionably devalued.

Now that I review these words, I am depressed. To me the prospects are bleak. But there are possibilities. Over the years my husband has grown to dislike his work as a designer. He has always been a musician at heart and has worked professionally in original music part-time. He shares my fears and has decided to try to become the primary caregiver. Neither of us is sure if he will adapt to this role on a full-time basis. Although chances are never good in music for new artists, if an offer were made there is no question that he would have to pursue it. There is serious interest even now, and I find myself in the unenviable position of hoping he realizes his professional dreams and fearing it as well.

The medical profession as a whole needs to address this problem as more women are becoming doctors. Rotating child care days with selected colleagues is my fantasy. As a psychiatrist, I have a pretty good idea of the quality of individuals in my medical community. I know who has the capacity to be committed, competent, reliable. Although this would create a lot of change in a youngster's life, it is not any different than my sister's use of my mother two days a week, her mother-in-law one day a week, and other relatives filling in at different intervals. It seems to be working well for her and forming a very social child!

Another possibility is to reassess the value of child care and make it desirable as a profession. My hospital has a carefully supervised day care center for children of residents. It is on-site at the hospital, in the housing. The children who live there never have to leave the grounds. If the parents could be assigned to rotate through, it would be ideal.

Roles in our society are changing. Our children are sometimes called our most precious resource. We need to build new social structures to care for and protect them. Dr Scanlon's remarks simply do not meet this challenge.

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1. Scanlon AT: Parenthood and practice. *NY State J Med* 1990; 90:326-327.

TO THE EDITOR: I read with interest Dr Scanlon's[1] article about her life as a pediatrician with ten children. However, I would have liked to see a more detailed description of exactly how it was done. I wonder if crucial details were omitted, or if times have changed so much that her world and ours seem to be two different worlds. Perhaps Dr Scanlon and her husband are just far more competent than the rest of us. I am an obstetrician-gynecologist in private practice on staff at North Shore University Hospital with five small children, and although I would have liked even more, I have made peace with what I perceive as reality. It does not seem possible to have a job—any job—let alone something as demanding as medicine, and still adequately care for many children.

Dr Scanlon states that her practice "increased more rapidly on the Island when people realized I was available all the time." How did constant availability fit in with caring for ten little ones? Perhaps part of the difference lies in the child care help that Dr Scanlon was fortunate enough to have. Most female doctors today will tell you how difficult it is to find someone faithful and hardworking, let alone someone who is "loving, intelligent, and willing to carry out your wishes on child care." And *one* person to care for ten children? Let's face it, there have to have been many times that the housekeeper was by herself and on her own. Most housekeepers today know they can easily work for people with one or two children. There are, in my experience, very few people who would even consider coming to a household with five children, let alone ten.

When I arrive home, I am usually greeted at the door by five screaming babies and children. Four are old enough presently to run into my arms and start fighting over my lap. (I, in the meantime, still have my jacket on and my pocketbook slung over my shoulder.) The fifth, at nine months, is old enough only to start crying pitifully after realizing he is going to be the last to get any individual attention. Then the three older ones, ages $6\frac{1}{2}$, $4\frac{1}{2}$, and 3, start crying because each is trying to tell me something at the same time and none of them believe I can actually hear all of them. I can only imagine the scene with ten! About the CME courses? If each physician spouse took one child, the implication is that the housekeeper and the other spouse shouldered responsibility for the other nine for several days. Again, if her husband was in the hospital with deliveries, that must have meant the housekeeper was on duty around the clock with nine children, again an unlikely situation today.

Obviously, economics have changed drastically (or Dr Scanlon was independently wealthy). Ten children, *and* vacations, *and* eight days at the Lake Placid Club, *and* a 14-room house on 75 acres?! With five children and a house near where I work, my husband and I can rarely splurge for items that are not essential. We just took our first family vacation, a four-day stay at Disney World, and some time will elapse before our next.

As for not playing bridge or golf, that is obvious. I have trouble allotting adequate time for washing my hair and brushing my teeth properly. Have times changed so much, or is nostalgia crowding out memories of the difficult times, much as I have forgotten the pain of my five labors? I am someone whose children are too young to be doing their own laundry or cooking their own meals. A description of how early childhood was survived would be most helpful!

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1. Scanlon AT: Parenthood and practice. *NY State J Med* 1990; 90:326-327.

An introduction to a current bibliography on women in medicine

TO THE EDITOR: It is surprising to find a striking paucity of articles on women in medicine in the recent medical literature. Over the last decade, when the numbers of women entering the field rose from 5% in the 1950s and 1960s, to about 50% in current medical school classes, only an average of 87 articles per year are listed. This may be an artifactual reflection of the difficulties in harvesting titles that have been variously categorized in the literature.

There are approximately 5,000 medical journals published worldwide annually. Of these, 2,400 are indexed in the *Index Medicus*, from which a computerized bibliography was drawn, and which covers the last seven years, 1983-1990. (This bibliography is not published here, but can easily be obtained through any computer network serving medical personnel with access to the great library systems.) This particular survey yielded only 480 articles. Considering the growing numbers and influence of women in medicine, this number seems to lag behind the actual presence of women in the field.

Understandably, most of the articles pertaining to the subject were published in the *Journal of the American Medical Women's Association*, the one journal consistently devoted (for many decades) to women and their professional productivity. Articles came from a wide geographic range, including Australia, New Zealand, the Soviet Union, and all of the countries of Northern Europe. In addition to scientific articles, there were editorials, letters to the editor, biographical vignettes, and appreciations.

The literature pertaining to women in medicine may yet be scarce in traditional medical journals, but has appeared elsewhere. Scholars in the fields of sociology, anthropology, social psychology, economics, and women's studies have studied women physicians more than they themselves. This is understandable, given the busy lives of women in medicine, and their lack of training for observing themselves as a social phenomenon. Although there are helpful articles for women in medicine in the paraprofessional literature and in the public press, they are beyond the scope of this survey.

The variety of articles found in the first two years of this bibliography were compared with those in the last two, to see what changes had occurred in the material presented. Of the first 147 articles during those early years, 14 dealt with pregnancy in women physicians, six covered substance abuse and women physicians, and five considered "stress" in this population. Only three referred to the issues of minority women. Although there were no articles about women in pediatrics, the most traditional clinical field for women, there were numerous articles about women in other specialties. These included eight about general practice, nine about radiology, six about psychiatry, four about obstetrics and gynecology, and two about surgery. There was only one article about women in each of the following disciplines: academic medicine, administrative medicine, internal medicine, anesthesia, and ophthalmology. Several articles were published dealing with gender comparisons between the two sexes in medicine.

The final 149 articles did more profiles but were less interested in women per se. Life cycle problems of women in medicine continued to be popular: there were 18 articles about pregnancy and family life. Twenty-one were general and survey articles, and five dealt with stress and substance abuse among this population. There were no articles comparing the attitudes or practice of men and women regarding such critical current issues as acquired immunodeficiency syndrome, nuclear war, or medical care in the inner cities. Current titles appear to speak for themselves. They include such disparate names as: "Role montage: Life patterns of the professional woman," "The Superwoman syndrome," "The taxicab theory," etc. Articles exist on professional dress codes, the choice of a name under which to practice, and even articles on motherhood when it comes as a surprise to the professional woman.

The overall impression is that women have been increasingly integrated into the mainstream of medical life without having been adequately studied, or the phenomenon of the growth spurt recorded for eventual historical analysis. Many titles seem better suited to the interests of the non-peer reviewed press. Was the phenomenon of the sudden growth of women in medicine not deemed of sufficient interest to the rest of the profession, or to society, to be studied as it should have been? Will it be too late to rectify this apparent lack, when the need becomes apparent in later years? Or has it been that women have been so busy integrating and catching up, there has been no time for the scholarly research which might have eased their lot, and provided a reasonable basis for alterations in the process of delivery of medical care, research, and teaching?

Readers interested in the phenomenon of women in medicine are urged to review the current bibliographies, with an eye to enriching the literature and taking advantage of what exists.

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Substance abuse during pregnancy

TO THE EDITOR: Substance abuse during pregnancy is particularly alarming because the problem involves two individuals, the mother and her infant. Often the problem becomes apparent only after withdrawal symptoms are detected in the newborn. This pilot study, of five months' duration, was performed to evaluate the extent of the problem of drug abuse in pregnant patients admitted in active labor to a teaching hospital in a low socioeconomic area of the South Bronx.

Research Methods and Results. Toxicologic studies were performed on 200 urine samples from patients admitted in active labor to the Bronx-Lebanon Hospital Center, an inner-city teaching hospital in a low socioeconomic area of the South Bronx. All patients subsequently delivered. Specimens were collected daily with no attempt to identify the patients; hence, no informed consent was necessary. The study was done with full approval of the hospital center's research review committee. Toxicologic analysis for cocaine, marijuana, methadone, heroin, methaqualone (Quaalude™), diazepam (Valium™), angel dust, barbiturates, and amphetamines was done over a five-month period from September 1, 1987, through January 31, 1988. Urine samples were collected by a nurse from every patient admitted in active labor and were sent directly to the hospital laboratory for routine urinalysis. Rather than discarding the remainder of the specimens, they were sent for toxicologic analysis. The first 40 urine samples, which arrived for toxicologic analysis during the first week of each of the five months of the study, were tested, ie, a total of 200 random "discarded urine samples" were analyzed.

TABLE I. Results of Analysis for Drugs of Abuse in Random Urine Samples from 200 Patients in Active Labor

Drug	Number Positive	Percent Positive	Retention Time in Urine (Days)
Amphetamines	1	0.5	2
Angel dust or PCP (phencyclidine)	1	0.5	8
Barbiturates	2	1.0	2–21
Cocaine (benzole gonine) alone	4	2.0	2–5
Cocaine plus heroin	2	1.0	2–5
Cocaine plus marijuana	33	16.5	2–21
Cocaine plus methadone	3	1.5	2–5
Heroin (diacetylmorphine) alone	1	0.5	2–4
Marijuana (cannabinoid) alone	3	1.5	5–21
Methadone (dolophine) alone	5	2.5	3
Quaalude (methaqualone)	2	1.0	14
Valium (diazepam)	2	1.0	3–4
Total	59	29.5	—

All testing was performed in the same laboratory. Radioimmunoassays were carried out using polyclonal antibodies against the drugs and/or one or more of their metabolites.[1] Whenever positive results were obtained, they were verified by repeat testing with gas chromatography. The ages of patients were noted.

Results of analysis for drugs of abuse in random urine samples showed 59 of 200 (29.5%) positive (Table I). The estimated retention time of these drugs in the urine is also shown in Table I. A variety of drugs were found, primarily cocaine in combination with marijuana (33 of 200, or 16.5%). For comparison, the urines of those babies exhibiting withdrawal symptoms in the neonatal intensive care unit were analyzed; 13% were positive for cocaine and marijuana. Table II indicates the age ranges of the patients in the study; the youngest patient was 12 and the oldest was 43. The largest group of patients, more than a quarter of those tested, were between 20 and 24 years of age. The majority of the patients with positive urines were between 20 and 29 years of age. The highest incidence of patients with positive urines was noted between the ages of 25 and 29 years.

Discussion. The antepartum complications associated with drug abuse include anemia, sexually transmitted disease, urinary tract infections, hypertensive disorders, pulmonary pathology, cellulitis or abscess, and severe edema.[2–4] Obstetric complications that have been reported include premature labor, intrauterine growth retardation, stillbirth, meconium, birth aspiration, neonatal withdrawal syndrome, abruptio placentae, extrauterine gestation, and spontaneous abortion.[3–6] One maternal death, secondary to postpartum hemorrhage, was reported by Silver et al[4] in his study of 336 women, 112 of whom were drug dependent before and during pregnancy. Little et al[7] and Chouteau et al[8] have documented the increased incidence of low birthweight infants and congenital anomalies in the newborn that result from cocaine abuse during pregnancy. Kreek et al[9] reported a 12% incidence of heroin addicts and former addicts to be chronic carriers of hepatitis B-antigen. Acquired immunodeficiency syndrome (AIDS), often associated with intravenous drug use, has become an increasing problem during pregnancy as well.

It is apparent that drug abuse in pregnant patients, particularly those admitted in active labor from the South Bronx community served by this hospi-

TABLE II. Age Range of Patients in Study and of Those Positive for Drugs of Abuse in Random Urine Samples

Age Range (yrs)	No. Patients	Patients with Positive Urines No.	Patients with Positive Urines %
10–14	4	1	25.0
15–19	39	7	17.9
20–24	56	17	30.4
25–29	50	20	40.0
30–34	31	11	35.5
35–39	14	2	14.3
40–45	6	1	16.7
Total	200	59	29.5

tal, is a serious problem. The 29.5% incidence of substance abuse found by random testing is much higher than that revealed during the routine admission interview by the nursing staff, as few patients admit to substance abuse. The 19% incidence of abuse of cocaine and cocaine combinations (Table I) is also higher than the positive test results for cocaine and marijuana in babies with a proven maternal history of drug abuse, ie, those identified by exhibiting withdrawal symptoms in the neonatal intensive care unit and who tested positive (13%). Based on these findings, testing all babies for drug abuse is being suggested to mothers to better anticipate problems and to provide better care for their babies.

It is interesting to note that a combination of drugs, rather than a single one, is used most often. This form of substance abuse has been documented by Wesson et al.[10] The most common combination in this series was cocaine and marijuana. It becomes apparent, as a result of interviewing many patients in our hospital, that this combination is attractive because it augments the euphoria and shortens the prolonged hyperexcitability of cocaine alone, which becomes intolerable. The low incidence of heroin and methadone in this series is also interesting. As individual patients were not identified, there was no way to determine how many of those with positive urines were intravenous drug users. With the high incidence of drug abuse in pregnancy found in this study, one can only speculate about the number of associated HIV positive and AIDS patients who will be encountered in the future.

Many of the patients tested may have been aware of their impending labor and delivery, but continued to take drugs. It is reasonable to assume that some of the patients stopped taking drugs just prior to admission, anticipating the onset of labor, but did not realize the long retention time of these drugs in the urine (Table I). It is alarming, yet quite possible, to consider that others may have intentionally taken drugs just prior to admission, "to make it easier to get through labor and delivery."

This random sampling of women admitted in labor in the South Bronx has revealed a high percentage of prenatal patients on drugs. Because of the high incidence of drug abuse found in this pilot study, a second phase of the study has been undertaken, with informed consent, to identify prenatal patients on drugs. Once found to have a positive urine sample during a prenatal visit, the woman will be offered a treatment plan consisting of counseling, psychological support, education, social service assistance, nutritional supplements, and specific treatment for any other existing disease problems. The third phase of the study, testing urine samples during labor of those patients treated for drug abuse during pregnancy, will assess the results of the effort to identify and treat these patients. This will also facilitate identification of expectant problems in the newborn and permit early and more effective treatment.

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Changes in state immunization requirements

Several important changes have taken place recently in the immunization requirements for protection against measles, mumps and *Haemophilus influenzae* type b (Hib). These changes primarily affect preschoolers, kindergarteners and college bound students. In addition, there is a new requirement to test all pregnant women for hepatitis B and to provide immunization for exposed newborn infants. The new requirements are summarized as follows:

Hib Vaccine Requirement for Preschoolers—Effective January 1, 1990, children enrolled in day care centers, nursery schools and pre-K programs between the ages of 18 months and five years (up to the fifth birthday) are required to show proof of vaccination against *Haemophilus influenzae* type b (Hib) in addition to other current vaccine requirements. Hib is the leading cause of meningitis in preschool-age children. There is a high rate of mortality and brain damage among children with the disease. Disease transmission can occur in day care settings. Hib vaccine is highly effective in preventing invasive disease from this organism.

Second Measles Vaccine Dose for Children Entering Kindergarten—Effective September 1, 1990, children entering kindergarten will be required to show proof of having received two vaccinations against measles. The first dose must have been received on or after the first birthday. The second dose must be given after 15 months of age (at least 30 days after the 1st dose), preferably at the prekindergarten doctor visit between four and six years of age. This is the same visit when DTP and OPV booster doses are currently given. Serologic evidence of immunity to measles or a physician documented history of measles disease is also acceptable. After 1990, this will apply to one successively higher grade each year by requiring that those entering school who were born on or after January 1, 1985, have two doses of measles vaccine. Measles, mumps and rubella (MMR) vaccine is the preferred vaccine for both doses of measles immunization. This change was prompted by widespread measles outbreaks during the 1988–1989 school year and by evidence that a single dose of measles vaccine does not provide sufficient levels of immunity to prevent outbreaks from occurring.

Measles/Mumps/Rubella Vaccine Requirement for College Students—Effective August 1, 1990, students attending post-secondary schools in New York State, who were born on or after January 1, 1957, will be required to show evidence of immunity against measles, mumps and rubella. Proof of immunity to measles will be defined as two doses of measles vaccine (same schedule as listed above), physician documented history of disease or serologic evidence of immunity. Proof of rubella immunity will mean one dose of rubella vaccine administered on or after the first birthday or serologic evidence of immunity. Proof of mumps immunity will mean one dose of mumps vaccine administered on or after the first birthday, a physician documented history of disease or serologic evidence of immunity. These new requirements will be phased in over two years: all full-time first and second year students will be covered in August 1990; all undergraduate and graduate students will be covered in August 1991 and thereafter. These requirements were made in response to measles, mumps and rubella outreaks occurring on New York State college campuses in recent years.

Hepatitis B Testing of Pregnant Women and Protection of Exposed Newborn Infants—Effective May 10, 1990, health care practitioners will be required to test pregnant women for hepatitis B surface antigen (HBsAg) to determine if they are infected with hepatitis B. The test can be done on the same blood sample taken for the required prenatal syphilis serology. Hospitals are required to obtain HBsAg test results within 48 hours for women admitted in labor who do not have test results available. The health care providers caring for newborn infants of HBsAg-positive mothers are required to offer hepatitis B immune globulin and hepatitis B vaccine for the infant within 12 hours after birth, as soon as physiologically stable, or as soon as HBsAg results for the mother are available. Follow-up doses of hepatitis B vaccine should be given at one and six months of age or at one, two and twelve months of age. This new law was enacted in response to evidence that hepatitis B infection is highly communicable from mother to newborn; is likely to lead to a chronic carrier state in the infant; and is preventable by the vaccination schedule outlined above. This is a cost effective disease prevention strategy.

Each of the requirements outlined above allows for religious and medical exemptions. Questions regarding these new immunization requirements and referral for vaccination of those unable to pay for any of these vaccines may be directed to your local health department or to the state or city Immunization Program listed below:

Buffalo (716) 847-4502
New Rochelle (914) 632-4133
Rochester (716) 423-8094
Syracuse (315) 426-7633
Albany (518) 473-4437
New York City Hotline (212) 349-2664
Long Island (516) 348-2742

Harvard Report highlights

On February 28, 1990, the New York State Department of Health released the results of the long awaited

Harvard Medical Practice Study, a comprehensive analysis of the relationship between medical injury, negligence, costs, compensation and lawsuits. The four-year, $3.1 million study, funded by New York State, focused on hospitals throughout the state, but has implications for the rest of the country's patients, physicians, hospitals and health care systems. Its purpose was to provide "relevant empirical data that will permit informed judgments and sound policymaking concerning this complex area," the authors said.

The study was mandated by the New York Legislature in 1986 to provide a factual background in the ongoing debate over the frequency and costs of medical malpractice.

Costs and Compensation

Eight times as many patients suffered an injury from negligence as filed a malpractice claim. About 16 times as many patients suffered an injury from negligence as received compensation.

An estimated 12,859 injuries from medical negligence did not lead to malpractice claims. Many cases in litigation were brought by patients whose records showed no evidence of negligence or injury caused by medical intervention.

The cost of medical injuries to New Yorkers (in 1989 dollars) is estimated at $894 million–$285 million in lost wages and fringe benefits, $103 million in uninsured medical costs and $506 in lost household production.

Injury

Approximately 4% of people hospitalized in 1984 were injured, more than one quarter of these resulted from negligence. That is 98,609 injuries, 27,179 of them—1% of all hospitalizations—from negligence.

More than half of these injuries resulted in minimal and transient disability. Some 14% of patients died at least in part as a result of their injuries. For another 9%, the disability lasted longer than six months.

Medical injury contributed, at least in part, to the deaths of more than 13,000 patients and approximately 2,500 cases of permanent total disability.

Deterrence

Physicians, hospitals and health organizations paid over $1 billion in malpractice premiums in 1988.

Physicians believe they face a 20% risk of being sued in a year, three times the actual risk of being sued. Overall, physicians believe they have a 60% chance of being sued for negligent care, substantially greater than the actual risk.

Physicians who perceived themselves to be at greater risk of being sued said in the past ten years, they had ordered more tests and procedures and reduced their practice scope.

Comparisons

People over 65 had twice the chance of being injured as those in the 16 to 44 age group. Newborns had half the injury rate of the 16 to 44 group.

Higher rates of medical injury were found in hospitals that served a higher proportion of minority patients.

The rate of injuries in upstate rural hospitals was one-third that of upstate metropolitan hospitals and less than one-fourth that of New York City.

The percent of injuries due to negligence was not significantly different across regions.

(Note: All estimates and projections are based on a review of 1984 hospital records and subsequent follow-up interviews and surveys.)

Mammography attitudes and awareness

Analyses of data collected by the New York State Department of Health continue to show that women in New York have one of the highest rates of breast cancer incidence and mortality in the United States. About one out of every ten women in the United States will develop breast cancer during her lifetime. Until this disease can be prevented, the best way women can protect themselves is through early detection and prompt treatment. Today, with the technology of mammography, breast cancer can be detected at very early stages of development, when the chance of cure is the highest.

Data on mammographic practices among women in New York State are limited. Therefore, a survey of mammography practices among women in New York State was initiated in the spring of 1989 by the New York State Department of Health, Bureau of Cancer Epidemiology. The initial survey was sent to a sample of 5,000 New York State women ages 50-74 within the 57 counties of New York State, excluding New York City, who were randomly selected from Department of Motor Vehicle files. Included with the survey was a cover letter and a stamped, self-addressed envelope for participants' use in returning the completed questionnaires. Nonrespondents to the initial letter were sent a second letter, then a short reminder card, yielding a participation response for all three mailings of over 63%.

Survey respondents were divided into three groups according to their current mammography practices: (1) annuals—women who reported receiving mammograms on an annual basis; (2) nevers—women who reported never having a mammogram; and (3) sporadic—women who reported having previously received at least one mammogram, but who did not obtain annual mammograms. Only 28% of the respondents reported receiving a mammogram each year. The remaining 72%, sporadic and never users, must be encouraged to seek or more fully utilize mammography. Because methods used to encourage the non-user may differ from methods employed with sporadic users, it is important to explore the differences in attitudes and awareness among the three groups. (See Figure 1.)

Almost all respondents in each group indicated that mammography is important. Eighty-four percent of "annuals" reported that they were afraid of breast cancer, compared to 66% of the "nevers" and 78% of the "sporadics." There was little difference among the groups regarding fear of discovering the disease through the use of mammography among the respondents in this survey. Al-

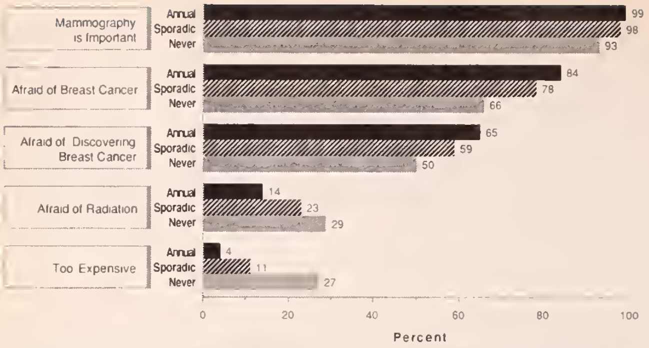

FIGURE 1. Attitudes and awareness of mammography among respondents to the NYS 1989 survey, ages 50–74.

most 30% of the nonusers indicated that they were afraid of radiation exposure, and a comparable proportion of nonusers mentioned the cost of a mammogram as a deterrent. Lower proportions of "annuals" and "sporadics" reported these concerns. When asked about insurance coverage, 60% of the "nevers" did not know if their insurance company provided coverage for mammography procedures. This compares to 10% of "annuals" and 30% of "sporadics" who reported that they were aware of insurance coverage for mammography.

Frequency of visiting a physician was also examined. Ninety-six percent of the "annual" users reported regular medical care, while only 64% of the women who had never received a mammogram reported that they receive medical care on a regular basis. Among "sporadic" users of mammography, 78% reported that they receive regular medical care. While a lower proportion of "sporadic" users and "never" users reported regular medical care, a majority of women in each group reported receiving medical care suggesting that access to medical care may not represent a barrier to mammography for these women.

Knowledge is a prerequisite for the adoption of any healthful behavior. Unfortunately, knowledge about breast cancer and the benefits of breast cancer screening in the general population is limited; therefore, the dissemination of educational interventions is of prime importance in affecting behavioral change. Since less than 30% of respondents reported receiving an annual mammogram, efforts are needed to encourage the sporadic and never users of mammography (encompassing over 70%) to adopt annual breast screening practices.

The mammography survey also included an intervention component designed to increase knowledge concerning the benefits of annual mammography screening and to evaluate the impact of providing educational materials describing mammography. Three interventions were designed to provide women with certain facts about the importance of mammography screening, information on what is involved in a mammogram and basic steps to prepare for the procedure. These interventions were mailed at six week intervals.

The first intervention consisted of the American Cancer Society (ACS) brochure on mammography that provides information about the nature and purpose of mammographic screening. This brochure contains a series of questions and answers providing information on ACS recommendations, benefits and risks of mammography, risk factors for breast cancer and the costs of screening mammography.

The second intervention, a "Mammography Fact Sheet," highlighted information given in the first intervention, and also reassured women about the safety, simplicity, and efficacy of the procedure. An insert drawing attention to the new insurance reimbursement coverage in New York State was also included, as well as a toll-free number that women could call to find where mammography is available.

The third intervention was designed to encourage asymptomatic women to include breast screening practices as part of their regular personal health regimen along with weight control, changes in diet and increased use of exercise programs.

Early in 1990 the "sporadic" and "never" groups will be resurveyed to determine the impact of these interventions. If they are successful in motivating women to participate in breast screening practices, interventions of these types may provide a low-cost and effective method of encouraging women to obtain mammograms on a regular basis.

In light of legislation recently passed by New York State requiring third party reimbursement for mammographic services, knowledge of mammography practices in New York State will be useful in directing promotional and educational campaigns to selected subpopulations currently underutilizing these services.

For additional information, contact Patricia Kraichy, Bureau of Cancer Epidemiology, (518) 473-7817.

Is lightscanning a viable alternative to mammography for detecting breast cancer?

Lightscanning involves the use of red and near-infrared light to visualize breast tissue. The efficacy of this technique, whether it is used alone or as a complement to mammography, is a controversial issue. In 1986, a Swedish team of physicians compared lightscanning with mammography in a prospective study. Their conclusion was clear: "lightscan as utilized by us was inferior to mammography" (*Cancer* 1990; 65:1671–1677).

Although mammography is an effective means of diagnosing breast cancer, it carries the risk of ionizing radiation—an important consideration in screening young women—and its diagnostic capabilities are more limited with dense breasts and in lobular carcinoma in situ and in intraductal, noninvasive cancer without microcalcification.

The Swedish study assessed the efficacy of lightscan in two protocols: the clinical study included patients with symptoms suggesting breast irregularities; the screening study included patients who had suspicious findings on a screening mammogram. In all, the one-year study encompassed 2,568 subjects and 5,087 breasts.

In the clinical study, examination of 3,178 breasts revealed 198 cancers. Mammography correctly identified 93% of these cancers, whereas lightscan identified 83%. False-positive findings of cancer were more common with lightscan (334 cases in 2,646 breasts) than with mammography (107 of 2,646). Lightscan led to the diagnosis of nine cases of cancer (4.5%) that were not detected by mammography. Lightscan was slightly more sensitive for breast cancer diagnosis in women younger than 50 years. "Calculations show that if the women ≤ 50 years of age had the same breast cancer prevalence as those >50 years, the positive predictive value for mammography would have been 78%, and for lightscan, 52%."

In the screening study, lightscan identified only 48% of the 126 cancers found by mammography in 1,909 breasts. Lightscan was especially poor in identifying small tumors. Of the 1,783 breasts with no cancer, mammography yielded false positives in 70, with lightscan erring in 156.

"In the absence of clinical information, the sensitivity of lightscan decreased in both the clinical and the screening study." The overall false-positive rate for lightscan (19.1%) compared to mammography (6.9%) is a major disadvantage of the former technique according to the authors. They cite the increased time and expense of lightscan as other negative factors. They conclude that, "It is our impression that the main contribution of lightscan is in a more careful and thorough clinical examination than is usual in clinical practice."

Antibiotics and other drugs may inhibit effectiveness of oral contraceptives

To date, there have been at least two lawsuits settled out of court that have sought compensation from dentists for patients taking oral contraceptives who became pregnant after undergoing antibiotic therapy prescribed by the dentists (*American Dental Association News* 1990; 21:1,6). There is evidence that some drugs, including several antibiotics, may interfere with the effectiveness of oral contraceptives. Despite the relatively low risk of pregnancy, the American Dental Association Council on Dental Therapeutics recommends that dentists inform female patients of the potential for a drug interaction.

Pharmaceutical companies provide warnings to both patients and physicians in the packaging materials of antibiotics and oral contraceptives. Dentists should advise female patients who use birth control pills to supplement their protection with an additional form of contraception during the menstrual cycle in which she is taking antibiotics or other drugs that are incompatible with oral contraceptives.

Two of the drugs frequently associated with reduced effectiveness of oral contraceptives are penicillin (and related antibiotics) and tetracycline—both of which are commonly prescribed by dentists to treat or prevent infection. Other drugs that may interfere with birth control pills include rifampin, ampicillin, neomycin, isoniazid, chloramphenicol, sulfonamide, nitrofurantoin, griseofulvin, barbiturates, phenytoin, carbamazepine, primidone, phenylbutazone, analgesics, tranquilizers, and medications to treat migraine headaches. It appears that the risk of oral contraceptive failure increases with the use of more than one of these drugs.

Premenstrual syndrome and diet

The physiologic basis of premenstrual syndrome (PMS) becomes clearer with each successive scientific study. The misconceptions that PMS is strictly a psychologic disorder are being replaced by data that demonstrate the complex bodily changes that women experience during the late luteal phase of the monthly menstrual cycle. Common symptoms of PMS include mood alterations such as irritability, depression, and anxiety, abnormalities in sleep patterns, bodily discomfort including bloating and breast tenderness, and food cravings. With respect to the latter, women who experience PMS frequently report cravings for carbohydrate-rich foods.

Judith Wurtman, PhD, and colleagues from the Massachusetts Institute of Technology, measured food intake and mood changes in 19 women with PMS, and compared the data with those for nine control subjects (*Am J Obstet Gynecol* 1989; 161:1228–1234). In a second study, the authors evaluated the effects of a carbohydrate-rich meal on mood and plasma hormone and metabolite levels in 18 women with PMS and 14 controls. For these studies, women with PMS scored ≥ 20 on the Hamilton Depression Score (HDS) during the late luteal phase (3–5 days before the onset of menses) and ≥ 30 on the HDS plus the addendum (which assessed changes in fatigue, appetite, carbohydrate craving, and sociability).

In the first study, performed during a 48-hour period in an outpatient facility, Wurtman et al measured the subjects' food intake; the women reported on their mood, ap-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

petite, sleep habits, and somatic complaints, and they underwent a psychiatric interview. In the second, outpatient study, the participants consumed a test carbohydrate meal during the early follicular phase (days 3–7 of the menstrual cycle) and the same meal during the late luteal stage.

The results from the first study indicate that "the subjects with PMS exhibited significantly elevated depression scores during the late luteal measurement period." Significant increases in fatigue, sociability, general appetite, and carbohydrate craving were reported, in addition to a significant decrease in work efficiency. Carbohydrate intake increased 24% ($p < 0.01$) during regular meals and 43% ($p < 0.003$) for snacks.

Women with PMS reported significant decreases in depression, tension, anger, confusion, and fatigue after the carbohydrate-rich meal consumed during the second study, as well as increases in calmness and alertness. There were no significant differences noted between PMS and control subjects with regard to postprandial plasma glucose, insulin, and amino acid levels.

The authors conclude that "individuals suffering from PMS may eat carbohydrates for their antidepressant effects." Carbohydrates increase the synthesis and release of serotonin in the brain, a neurotransmitter which may play a role in depression. Although this study was based on a very small sample population, it suggests a role for serotonin in the symptoms of PMS and a possible therapeutic role for mimicking the carbohydrate-induced increase in serotonin synthesis and release.

An attempt to predict bone mass in perimenopausal women using only clinical data

Determination of bone mass at multiple skeletal sites yields accurate estimates of mineral content when using such sophisticated techniques as single- and dual-photon absorptiometry, dual-energy roentgenographic absorptiometry, and quantitative computed tomography. Bone mass measurements have become an important diagnostic component in osteoporosis. Several studies have shown that low bone mass is suggestive of an increased risk of fracture in perimenopausal women.

Once low bone mass is detected, there are effective treatments, including calcitonin and estrogen replacement therapy. Although there is little support for screening of all perimenopausal women, it is important to identify those women with low bone mass who would benefit from available treatments. Slemenda et al sought to determine whether proposed risk factors for osteoporosis, including information obtained from a medical history, physical examination, and laboratory tests, could pinpoint women who should then undergo additional studies, such as bone mass assessment (*Ann Intern Med* 1990; 112:96–101).

Although there are no proven, quantitative risk factors for osteoporosis, the authors amassed data on suggested risk factors, including height, weight, age, time since last menstrual period, episodes of flushing, smoking, dietary calcium intake, and alcohol intake. In addition, the authors performed the following laboratory studies: urinary calcium, creatinine, and hydroxyproline. The researchers used single-photon absorptiometry to determine bone mineral content at the midshaft and distal radius as well as dual-photon absorptiometry to estimate values for the spine and hip.

Based on the data obtained, the authors assigned scores to the risk factors, "based on the strength . . . of each factor in predicting bone mass at a particular site." Finally, the authors "constructed general linear models to properly weight each of the many independent variables in multiple variable equations to maximize the correct classification of women with low bone mass."

These models were able to identify 65% of the women with low bone mass and 53% of the women with high bone mass. From this finding, the authors concluded that, "the analysis of multiple risk factors in perimenopausal women is an inadequate substitute for bone mass measurements." Missing more than 30% of women with reduced bone mass is not acceptable sensitivity. Furthermore, the model was least effective in predicting bone mass in the spine and hip, the most common sites for osteoporotic fractures. "For most candidates for estrogen therapy, a single bone mass measurement at any site provides information on which to base a therapeutic decision, information that cannot be obtained through other clinically available data."

Osteoporosis: Does sodium fluoride strengthen bone?

Sodium fluoride is a common treatment for osteoporosis; it can increase spinal bone mass, yet its ability to strengthen bone and prevent osteoporotic fractures has never been proven. The Food and Drug Administration has not approved sodium fluoride as a treatment for osteoporosis, although it is often used for this purpose (*JAMA* 1990; 1752–1753).

Studies have demonstrated that sodium fluoride increased spinal bone mass, but its ability to increase cortical bone mass and reduce one's risk of fractures is not supported by current evidence. Researchers at the Mayo School of Medicine (Rochester, Minn) have found that sodium fluoride therapy increases spinal bone mass by transferring bone material from cortical bone to trabecular bone. Based on their controlled study comparing sodium fluoride to a placebo, the researchers stated, "Our tentative conclusion is that bone that forms with fluoride therapy does not have normal strength and is qualitatively inferior." Thus, bone supposedly strengthened by sodium fluoride may actually be more susceptible to fractures.

Osteoporosis: New therapies may add bone mass

At least three new, experimental treatment regimens for osteoporosis are showing promise in early phases of testing (*JAMA* 1990; 263:1753). Clinical trials with diphosphonates suggest that they may inhibit bone loss in osteoporosis.

Injected calcitonin is an established treatment for osteoporosis in patients who cannot receive estrogen replacement, however, it is not appropriate for preventive use. Calcitonin in nasal-spray form is now undergoing clinical trials and appears to be effective in delaying or preventing bone loss in postmenopausal women.

Rather than simply inhibiting bone loss, however, a new, combined hormone therapy appears to increase bone mass density in the spine as well as increasing cortical bone mass. Preliminary findings of a two-year trial with the combination of estrogen and the progestogen norethindrone acetate are promising.

BOOKS RECEIVED

AIDS

Preventing AIDS: A Guide to Effective Education for the Prevention of HIV Infection. By Nicholas Freudenberg, DrPH. 217 pp, illustrated. Washington, DC, American Public Health Association, 1989. $22.00 (softcover)

CARDIOLOGY

Clinical Cardiology. Third Edition. By Peter C. Gazes, MD. 594 pp, illustrated. Philadelphia, Lea & Febiger, 1989. $55.00 (hardcover)

Progress in Cardiology. Edited by Douglas P. Zipes, MD, and Derek J. Rowlands, MD. 250 pp, illustrated. Philadelphia, Lea & Febiger, 1990. $39.50 (paperback)

The Nucleus of Cardiac Diagnosis: Electrocardiography and Chest X-Ray. By William A. Schiavone, DO. 287 pp, illustrated. Philadelphia, Lea & Febiger, 1990. $34.00 (paperback)

DERMATOLOGY

Pediatric Dermatology and Dermatopathology: A Text and Atlas. By Ruggero Caputo, MD, A. Bernard Ackerman, MD, and Evita Q. Sison-Torre, MD. 391 pp, illustrated. Philadelphia, Lea & Febiger, 1990. $150.00 (hardcover)

GERIATRICS

Resources for Elders with Disabilities. 168 pp. Lexington, Mass, Resources for Rehabilitation, 1990. $39.95 (paperback)

HEALTH CARE ADMINISTRATION

The Doctor's Office Guide to Medicare 1989–1990. By Philip L. Beard. 289 pp, illustrated. Willowbrook, Ill, Professional Medical Management Corp, 1989. $59.95 (softcover)

ICD-9-CM Coding Handbook: For Physician Practices. By Therese M. Jorwic, RRA. 379 pp. Willowbrook, Ill, Professional Medical Management Corp, 1989. $49.95 (softcover)

HISTORY OF MEDICINE

Ask Sir James: Sir James Reid, Personal Physician to Queen Victoria and Physician-in-Ordinary to Three Monarchs. By Michaela Reid. 315 pp, illustrated. New York, Penguin Books, 1987. $12.95 (paperback)

INFECTIOUS DISEASES

Antiviral Agents and Viral Diseases of Man. Third Edition. Edited by George J. Galasso, PhD, Richard J. Whitley, MD, and Thomas C. Merigan, MD. 800 pp, illustrated. New York, Raven Press, 1990. $150.00 (hardcover)

INTERNAL MEDICINE

Oxford Handbook of Clinical Medicine. By R. A. Hope, J. M. Longmore, P. A. H. Moss, and A. N. Warrens. 795 pp, illustrated. New York, Oxford University Press, 1989. $21.95 (paperback)

LEGAL MEDICINE

Legal Aspects of Medicine. Edited by James R. Vevaina, MD, Roger C. Bone, MD, Honorable Edwin Kassoff. 340 pp. New York, Springer-Verlag, 1989. $73.00 (hardcover)

MISCELLANEOUS

A Year-Long Night: Tales of a Medical Internship. By Robert Klitzman, MD. 242 pp. New York, Penguin Books, 1989. $7.95 (paperback)

Healing and Restoring: Health and Medicine in the World's Religious Traditions. Edited by Lawrence E. Sullivan. 468 pp. New York, Macmillan Publishing Co, 1989. $35.00 (hardcover)

Letting Blood. By Richard Platt, MD. and Orah Platt, MD. 176 pp. New York, St Martin's Press, 1988. $14.95 (clothcover)

Make Sure You Do Not Have Breast Cancer. By Philip Strax, MD. 118 pp, illustrated. New York, St Martin's Press, 1989. $15.95 (clothcover)

Occupational Therapy Assistant Career Profile. By Naomi Schubin Greenberg, PhD. 236 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1990. $37.50 (softcover)

Perspectives on Animal Research. Volume I. Edited by Stephen R. Kaufman, MD, and Betsy Todd, RN. 134 pp. New York, Medical Research Modernization Committee, 1989. $15.00 (hardcover)

Practice Made Perfect: The Physician's Guide to Communication and Marketing. By Edna Kaplan, MS. 261 pp. Boston, Mass, Barrington Press, 1990. $39.95 (hardcover)

Providing Services for People with Vision Loss: A Multidisciplinary Perspective. 129 pp, illustrated. Lexington, Mass, Resources for Rehabilitation, 1989. $19.95 (paperback)

Reporting on Risk: Getting It Right in an Age of Risk. By Victor Cohn. 65 pp. Washington, DC, The Media Institute, 1990. $8.95 (paperback)

Wisdom for Living: Your Health. By Hugh D. Patterson, MD. 97 pp, illustrated. New York, Vantage Press, Inc, 1989. $8.95 (hardcover)

NEUROLOGY

Ryan: A Mother's Story of Her Hyperactive/Tourette Syndrome Child. By Susan Hughes. 153 pp. Duarte, Calif, Hope Press, 1990. $9.95 (paperback)

Tourette Syndrome and Human Behavior. By David E. Comings, MD. 828 pp, illustrated. Duarte, Calif, Hope Press, 1990. $39.95 (paperback)

NUTRITION

Nutrition & Eating Disorders. By Catherine M. Patterson, MPH, Diane P. Whelan, MPH, Cheryl L. Rock, MMSc. Tami J. Lyon, MPH. 34 pp. Van Nuys, Calif, PM, Inc, 1989. $4.95 (paperback)

OBSTETRICS AND GYNECOLOGY

Methods of Childbirth. The Completely Updated Version of a Classic Work for Today's Woman. Revised Edition. By Constance A. Bean. 334 pp, illustrated. New York, William Morrow & Co, 1990. $12.95 (paperback)

Obstetrics & Gynecology: Annual Review 1989. Edited by Harrison H. Shield, MD. 321 pp. Elmsford, NY, Pergamon Press, Inc, 1989. $32.50 (softcover)

Tubal Reconstructive Surgery. By Ginter Sotrel, MD. 281 pp, illustrated. Philadelphia, Lea & Febiger, 1990. $75.00 (hardcover)

ONCOLOGY

Hodgkin's Disease: The Consequences of Survival. By Mortimer J. Lacher, MD, and John R. Redman, MD. 477 pp, illustrated. Philadelphia, Lea & Febiger, 1990. $80.00 (hardcover)

OPHTHALMOLOGY

The Ocular Fundus: A Photographic Documentation Atlas With Diagnostic and Therapeutic Guidelines. By Manfred R. Tetz, MD, and David J. Apple, MD. 215 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1990. $129.50 (hardcover)

PEDIATRICS

Control of the Onset of Puberty. Edited by Melvin M. Grumbach, MD, Pierre C. Sizonenko, MD, and Michael L. Aubert, MD. 710 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1990. $125.00 (hardcover)

The Role of Professional Background, Case

Characteristics, and Protective Agency Response in Mandated Child Abuse Reporting. By Gail L. Zellman and Robert M. Bell. 163 pp. Santa Monica, Calif, The Rand Corporation, 1990. $15.00 (paperback)

PHARMACOLOGY

Toxicokinetics and New Drug Development. Edited by Avraham Yacobi, PhD, Jerome P. Skelly, PhD, and Vijay K. Batra, PhD. 246 pp, illustrated. Elmsford, NY, Pergamon Press, Inc, 1989. $25.00 (hardcover)

PREVENTIVE MEDICINE AND PUBLIC HEALTH

Health Education: Principles, Preparation, and Practice. By Karen M. Lorentzen, RN, and Robert N. Neal, EdD. 124 pp. St Louis, Mo, Warren H. Green, Inc, 1990. $18.75 (paperback)

Organizing Villagers for Self-Reliance: A Study of Gonoshasthya Kendra in Bangladesh. By Jayanta Kumar Ray. 104 pp. Calcutta, Orient Longman Ltd, 1986. (paperback)

Plagues and Politics: The Story of the United States Public Health Service. By Fitzhugh Mullan, MD. 223 pp, illustrated. New York, Basic Books, Inc, 1989. $26.95 (hardcover)

PSYCHIATRY

A Day Hospital Group Treatment Program for Anorexia Nervosa and Bulimia Nervosa. Edited By Niva Piran, PhD, and Allan S. Kaplan, MD. 168 pp, illustrated. New York, Brunner/Mazel Publishers, 1990. $25.00 (hardcover)

Annual Progress in Child Psychiatry and Child Development 1989: A Selection of the Year's Outstanding Contributions to the Understanding and Treatment of the Normal and Disturbed Child. Edited by Stella Chess, MD, and Margaret E. Hertzig, MD. 576 pp, illustrated. New York, Brunner/Mazel Publishers, 1990. $50.00 (hardcover)

Brief Therapy Approaches to Treating Anxiety and Depression. Edited by Michael D. Yapko, PhD. 384 pp, illustrated. New York, Brunner/Mazel Publishers, 1989. $39.95 (hardcover)

Child Psychiatry: A Primer for Those Who Work Closely With Children. By R. Dean Coddington, MD, and Mollie M. Wallick, PhD. 254 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1990. $37.50 (paperback)

Cognitive-Behavioral Marital Therapy. By Donald H. Baucom, PhD, and Norman Epstein, PhD. 496 pp. New York, Brunner/Mazel Publishers, 1990. $47.95 (hardcover)

Computer Applications in Psychiatry and Psychology. Edited by David Baskin, PhD. 192 pp, illustrated. New York, Brunner/Mazel Publishers, 1990. $23.95 (hardcover)

Dry All Night: The Picture Book Technique That Stops Bedwetting. By Alison Mack. 176 pp, illustrated. Waltham, Mass, Little, Brown & Co, 1989. $17.95 (hardcover)

DSM-III-R Training Guide for Diagnosis of Childhood Disorders. By Judith L. Rapoport, MD, and Deborah R. Ismond, MA. 224 pp. New York, Brunner/Mazel Publishers, 1990. $17.95 (paperback)

Hypnotherapy Scripts: A Neo-Ericksonian Approach to Persuasive Healing. By Ronald A. Havens, PhD, and Catherine Walters, MA, MSW. 216 pp. New York, Brunner/Mazel Publishers, 1989. $27.95 (hardcover)

Modern Perspectives in the Psychiatry of the Affective Disorders. Edited by John G. Howells, MD. 440 pp. New York, Brunner/Mazel Publishers, 1989. $60.00 (hardcover)

Psychological Consulting to Management: A Clinician's Perspective. By Lester L. Tobias, PhD. 208 pp. New York, Brunner/Mazel Publishers, 1990. $25.00 (hardcover)

Psychosocial Interventions with Physically Disabled Persons. Edited by Bruce W. Heller, PhD, Louis M. Flohr, MD, and Leonard S. Zegans, MD. 336 pp. New Brunswick, NJ, Rutgers University Press, 1989. $17.00 (paperback)

The Crises of Competence: Transitional Stress and the Displaced Worker. By Carl A. Maida, PhD, Norma S. Gordon, MA, and Norman L. Farberow, PhD. 226 pp. New York, Brunner/Mazel Publishers, 1989. $27.50 (hardcover)

The Practical Application of Medical and Dental Hypnosis. By Milton H. Erickson, MD, Seymour Hershman, MD, and Irving I. Secter, DDS. 480 pp. New York, Brunner/Mazel Publishers, 1990. $18.95 (paperback)

Trancework: An Introduction to the Practice of Clinical Hypnosis. Second Edition. By Michael D. Yapko, PhD. 480 pp. New York, Brunner/Mazel Publishers, 1990. $42.50 (hardcover)

Trauma and the Vietnam War Generation: Report of Findings from the National Vietnam Veterans Readjustment Study. By Richard A. Kulka, PhD, William E. Schlenger, PhD, John A. Fairbank, PhD, Richard L. Hough, PhD, B. Kathleen Jordan, PhD, Charles R. Marmar, MD, and Daniel S. Weiss, PhD. 352 pp. New York, Brunner/Mazel Publishers, 1990. $19.95 (hardcover)

RADIOLOGY

Clinical Implications of Abnormal Digestive Tract Radiographs. By David A. Morowitz, MD. 235 pp, illustrated. Philadelphia, Lea & Febiger, 1990. $39.50 (hardcover)

SPORTS MEDICINE

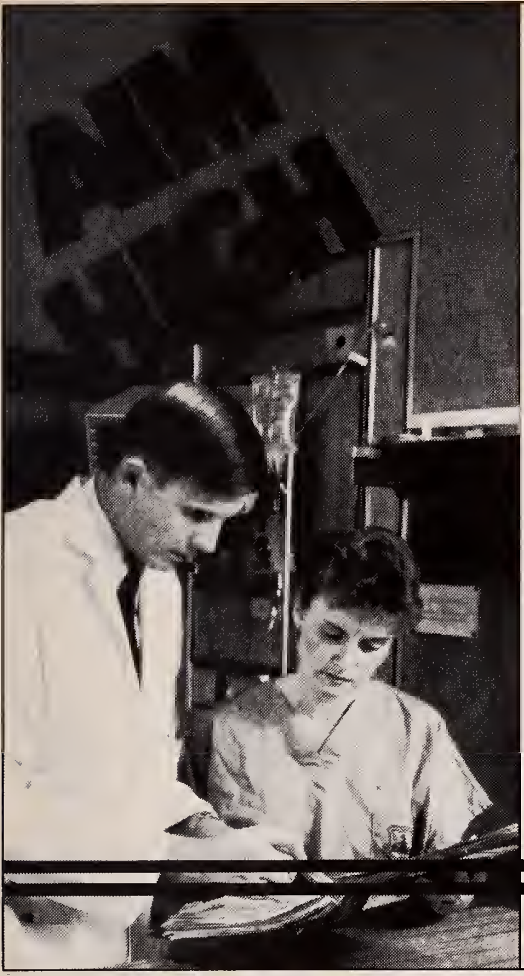
Drugs and the Athlete. By Gary I. Wadler, MD, and Brian Hainline, MD. 353 pp, illustrated. Philadelphia, F. A. Davis Company, 1989. $45.00 (hardcover)

SURGERY

Lasers in Surgery. Edited by Stephen N. Joffe, MD. 319 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1989. $65.00 (hardcover)

Trauma, Transformation and Healing: An Integrative Approach to Theory, Research and Post-Traumatic Therapy. By John P. Wilson, PhD. 368 pp. New York, Brunner/Mazel Publishers, 1989. $40.00 (hardcover)

Biliary Tract Surgery: Tactics and Techniques. By Jacob A. Glassman, MD. 416 pp, illustrated. New York, Macmillan Publishing Co, 1989. $90.00 (hardcover)



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(*continued from p 3A*)

ology. 1 Cat I Credit. Southampton Hospital. Contact: Charlotte Henry-Sullivan, Medical Staff Secretary, Southampton Hospital, 240 Meeting House Lane, Southampton, NY 11968. Tel: (516) 283-2600, ext 519.

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July 19–21. **Rheumatology and Arthritis: A Conference for Primary Care Physicians.** 13 Cat I Credits. Catamaran Resort Hotel, San Diego. Contact: Debra L. Gist, Medical Staff/CME Coordinator, Continuing Medical Education Department, Sharp Memorial Hospital, 7901 Frost St, San Diego, CA 92123. Tel: (619) 541-4530.

July 19–22. **Radiology for Non-Radiologists.** Hotel del Coronado, San Diego. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

July 23–25. **Musculoskeletal Imaging.** 14¾ Cat I Credits. *July 26–29.* **The Fourth Annual Symposium on Magnetic Resonance Imaging.** 22 Cat I Credits. Ritz Carlton Resort Hotel, Laguna Niguel. Contact: Dawne Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

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July 19–21. **Substance P and Related Peptides: Cellular and Molecular Physiology.** Mechanics Hall, Worcester. Contact: Conference Director, The New York Academy of Sciences, 2 E 63rd St, New York, NY 10021. Tel: (212) 838-0230.

July 21–22. **Depression and Suicide.** 6 Cat I Credits. New Seabury Resort, Cape Cod. Contact: Eric Schwartz, Program Administrator, Office of Continuing Medical Education, Albert Einstein College of Medicine/Montefiore Medical Center, 2201 Bainbridge Ave, Bronx, NY 10467. Tel: (212) 920-6675.

MICHIGAN

July 8–11. **22nd Annual March of Dimes Clinical Genetics Conference.** 25 Cat I Credits. The Hyatt Regency, Dearborn. Contact: Carol Blagowidow, Professional Services Dept, March of Dimes Birth Defects Foundation, PO Box 2000, White Plains, NY 10602.

TEXAS

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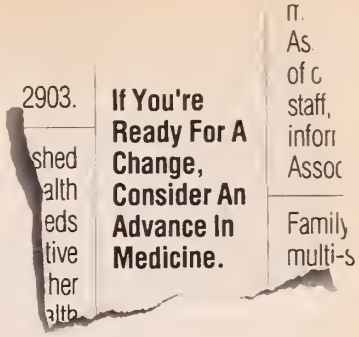

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Bell Atlantic 3A

Classified Advertising 4A, 5A, 7A, 8A, 10A

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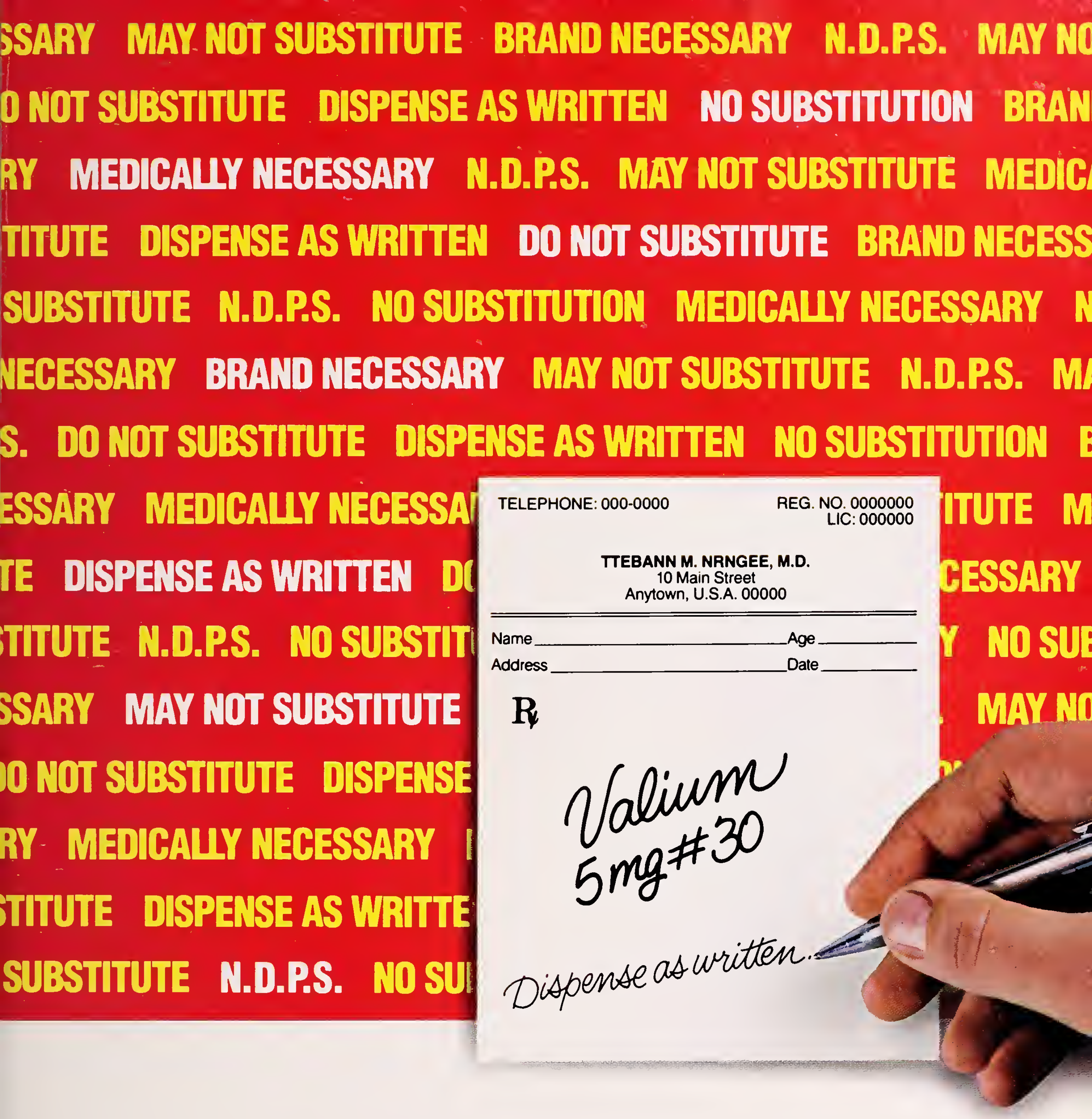
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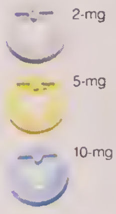

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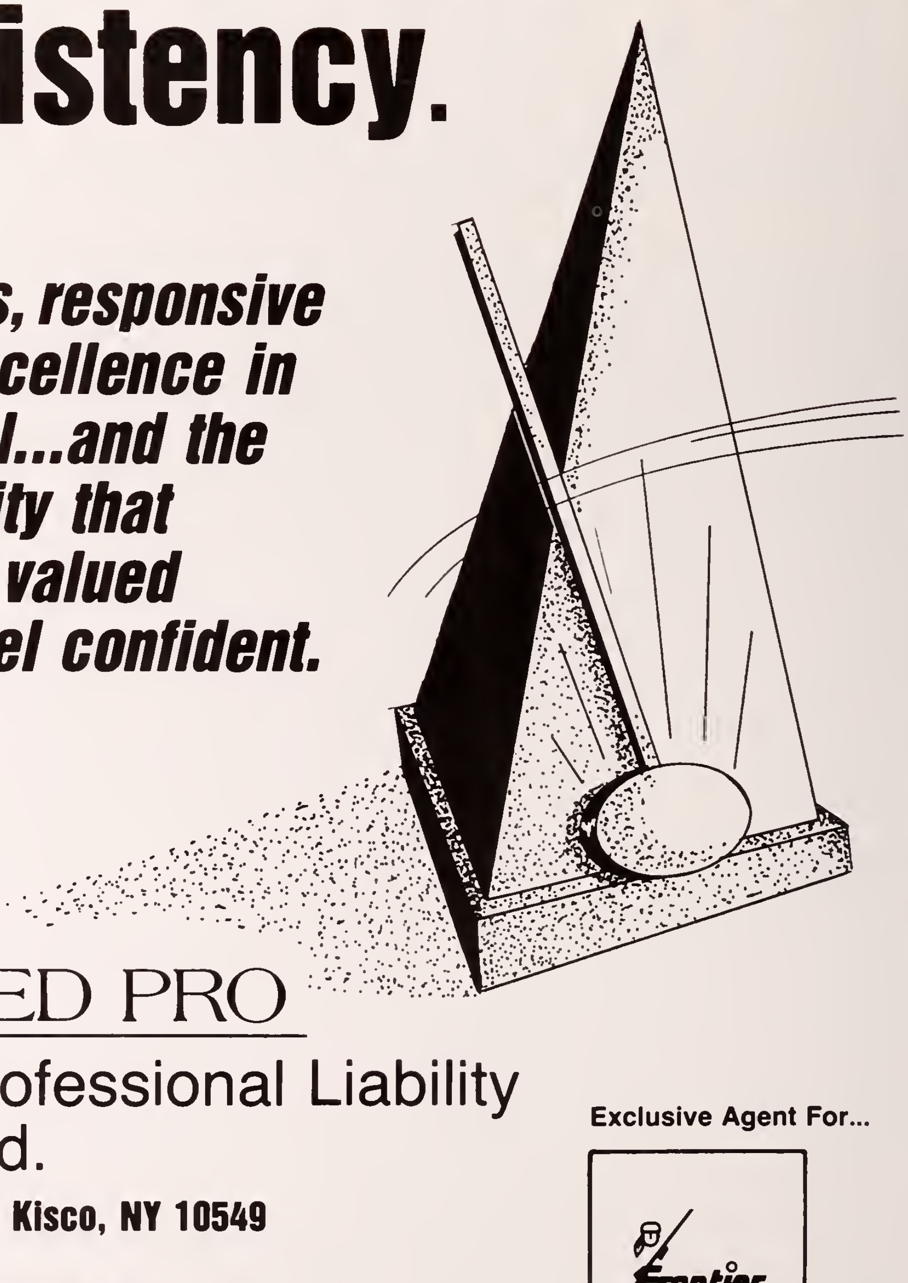

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Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

AUGUST 1990

AROUND THE STATE

BUFFALO

Aug 20–24. **The International Society of Psycho-Neuro Endocrinology.** Hyatt Regency. Contact: Dr Uriel Halbriech, SUNY-AB, 462 Grider St, K-Annex, Buffalo, NY 14215. Tel: (716) 898-3036.

COOPERSTOWN

Aug 24–26. **Third Annual Pediatric Refresher Course.** The Otesaga Hotel. Contact: Charlotte L. Hoag, Medical Education Coordinator, Office of Medical Education, The Mary Imogene Bassett Hospital, One Atwell Rd, Cooperstown, NY 13326. Tel: (607) 547-3926.

MANHATTAN

Aug 18–24. **The Ninth Annual Meeting of the Society of Magnetic Resonance in Medicine.** Contact: Lynette Corning, Acting Director, Society of Magnetic Resonance in Medicine, 1918 University Ave, Suite 3C, Berkeley, CA 94704. Tel: (415) 841-14899.

Aug 27–31. **Radiation, Physics, and Biology.** 32 Cat I Credits. NYU Medical Center. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

AROUND THE NATION

CALIFORNIA

Aug 8–11. **Sharp Surgical Review Course.** 14 Cat I Credits. Le Meridien Hotel, Coronado (San Diego). Contact: Debra L. Gist, Medical Staff/CME

(*continued on p 14A*)

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2. Standard industry new prescription audit.

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Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression.

Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.

Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.

PRECAUTIONS:

Special Risk Patients: VICODIN/VICODIN ES Tablets should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.

Cough Reflex: Hydrocodone suppresses the cough reflex; as with all narcotics, caution should be exercised when VICODIN/VICODIN ES Tablets are used postoperatively and in patients with pulmonary disease.

Drug Interactions: Patients receiving other narcotic analgesics, antipsychotics, antianxiety agents, or other CNS depressants (including alcohol) concomitantly with VICODIN/VICODIN ES Tablets may exhibit an additive CNS depression. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.

Usage in Pregnancy:

Teratogenic Effects: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN/ VICODIN ES Tablets should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nonteratogenic effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The withdrawal signs include irritability and excessive crying, tremors, hyperactive reflexes, increased respiratory rate, increased stools, sneezing, yawning, vomiting, and fever.

Labor and Delivery: Administration of VICODIN/VICODIN ES Tablets to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk and because of the potential for serious adverse reactions in nursing infants from VICODIN/VICODIN ES Tablets, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS:

The most frequently observed adverse reactions include light-headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include:

Central Nervous System: Drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, psychic dependence and mood changes.

Gastrointestinal System: The antiemetic phenothiazines are useful in suppressing the nausea and vomiting which may occur (see above); however, some phenothiazine derivatives seem to be antianalgesic and to increase the amount of narcotic required to produce pain relief, while other phenothiazines reduce the amount of narcotic required to produce a given level of analgesia Prolonged administration of VICODIN/VICODIN ES Tablets may produce constipation.

Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported.

Respiratory Depression: Hydrocodone bitartrate may produce dose-related respiratory depression by acting directly on the brain stem respiratory center. Hydrocodone also affects the center that controls respiratory rhythm, and may produce irregular and periodic breathing. If significant respiratory depression occurs, it may be antagonized by the use of naloxone hydrochloride. Apply other supportive measures when indicated.

DRUG ABUSE AND DEPENDENCE:

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OVERDOSAGE:

Acetaminophen Signs and Symptoms: In acute acetaminophen overdosage, dose-dependent, potentially fatal hepatic necrosis is the most serious adverse effect. Renal tubular necrosis, hypoglycemic coma, and thrombocytopenia may also occur. Early symptoms following a potentially hepatotoxic overdose may include: nausea, vomiting, diaphoresis and general malaise. Clinical and laboratory evidence of hepatic toxicity may not be apparent until 48 to 72 hours post-ingestion.

Hydrocodone Signs and Symptoms: Serious overdose with hydrocodone is characterized by respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, (cyanosis), extreme somnolence progressing to stupor or coma, skeletal muscle flaccidity, cold and clammy skin, and sometimes bradycardia and hypotension. In severe overdosage, apnea, circulatory collapse, cardiac arrest and death may occur.

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New York State Journal of Medicine

JULY 1990 / Volume 90, Number 7

Contents

COMMENTARIES

345 **A new cover for the *New York State Journal of Medicine***
PASCAL JAMES IMPERATO, MD

346 **The behavioral risks: Mother knows best**
MICHAEL D. DECKER, MD

347 **Uninvited guests: Cardiovascular foreign bodies**
DANIEL A. HENRY, MD

RESEARCH PAPERS

349 **Death takes a ride: Alcohol-associated single vehicle fatalities revisited**
MILLARD J. HYLAND, MD; ALBERT B. LOWENFELS, MD; CATHEY E. FALVO, MD, MPH; EACHOU CHEN, MD, MPH

351 **Drug resistant tuberculosis among the homeless in New York City**
ARIEL PABLOS-MENDEZ, MD; MARIO C. RAVIGLIONE, MD; RUGGERO BATTAN, MD; RODRIGO RAMOS-ZUNIGA, MD

356 **Fibrous disease of the breast in juvenile diabetes**
FRANK E. GUMP, MD, JANETTE McDERMOTT, MD

REVIEW ARTICLE

358 **Aeromedical emergency trauma services and mortality reduction in rural areas**
DANIEL A. FREILICH, MD, ALLEN D. SPIEGEL, PhD, MPH

SPECIAL ARTICLE

366 **Maintaining serenity in chronic illness**
JACQUELINE GALLOWAY

CASE REPORTS

368 **Hypodermic needle embolization to the heart**
MARC F. STERN MD, BARBARA G. STEINBACH, MD

(*continued on p 7A*)

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References
1. *USP DI Update, September/October 1988, p 120.*
2. *Br J Clin Pharmacol 1985;20:710-713.*
3. *Data on file, Lilly Research Laboratories.*
4. *Scand J Gastroenterol 1987;22(suppl 136):61-70.*
5. *Am J Gastroenterol 1989;84:769-774.*

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Indications and Usage: 1. *Active duodenal ulcer*—for up to eight weeks of treatment. Most patients heal within four weeks.

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Contraindication: Known hypersensitivity to the drug. Use with caution in patients with hypersensitivity to other H_2-receptor antagonists.

Precautions: *General*—1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Dosage should be reduced in patients with moderate to severe renal insufficiency.

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Carcinogenesis, Mutagenesis, Impairment of Fertility—A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of varying durations included almost 5,000 patients. Among the more common adverse events in domestic placebo-controlled trials of over 1,900 nizatidine patients and over 1,300 on placebo, sweating (1% vs 0.2%), urticaria (0.5% vs <0.01%), and somnolence (2.4% vs 1.3%) were significantly more common with nizatidine. It was not possible to determine whether a variety of less common events was due to the drug.

Hepatic—Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizabdine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to three times the upper limit of normal, however, did not significantly differ from that in placebo patients. Hepatitis and jaundice have been reported. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS—Rare cases of reversible mental confusion have been reported.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with equal frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic—Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental—Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity—As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Because cross-sensitivity among this class has been observed, H_2-receptor antagonists should not be administered to those with a history of hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance by approximately 84%.

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New York State
Journal of Medicine

371 **Leiomyosarcoma of the esophagus**
SHAILESH R. PATEL, MD, NANARAO ANANDARAO, MD

373 **Gardner-Diamond syndrome**
MAXWELL L. GELFAND, MD

375 **Volar proximal skin necrosis after radial artery cannulation**
ROBERT D. GOLDSTEIN, MD, MICHAEL J. V. GORDON, MD

LETTERS TO THE EDITOR

377 **UNY*Care**
JONATHAN B. PASTERNACK, MD

377 **Authors' academic affiliation**
NANCY E. GARY, MD

378 **Claims-made policies and physician employment contracts**
DONALD R. MOY, ESQ

378 **Avicenna, problem solving, and creativity during sleep**
JEROME M. SCHNECK, MD

379 **The turbulent years**
RANDALL D. BLOOMFIELD, MD

380 LEADS FROM EPIDEMIOLOGY NOTES

384 BOOK REVIEWS

387 NEWS BRIEFS

389 OBITUARIES

392 GUIDELINES FOR AUTHORS

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New York State
Journal of Medicine

COMMENTARIES

A new cover for the *New York State Journal of Medicine*

The *New York State Journal of Medicine* was first published in January 1901 by the New York State Medical Association (NYSMA). Five years later, after the NYSMA joined with the Medical Society of the State of New York (MSSNY), the *Journal* became the society's official publication. During its almost 90 years of continuous publication, the *Journal* has had 18 different cover designs, the first lasting from 1901 through 1905. These covers fall into three broad design formats: those displaying pictures, those carrying a complete or partial table of contents, and those listing key articles. The longest a cover design has lasted is ten years (1924–1933) and the shortest, one year (1973). The cover which has just been replaced was used for seven years, from July 1983 through June 1990. Overall, the average life span of a cover has been around seven years, lending credibility to the popular belief in a "seven-year itch." In addition to changes in cover design, the *Journal* has also undergone changes in size. From 1901 through 1933 it measured 8″ × 11″; from 1934 through 1970, 6¼″ × 9½″, and finally, from 1971 to the present, 8³⁄₁₆″ × 11″.

Covers which carry the entire table of contents are popular with many readers because they provide easy access to the information in a given issue. They are unpopular with others because they appear either cluttered or dull and often limit editors with regard to the number of articles that can be carried in a given journal issue. Clearly, there is no design that will satisfy all of the *Journal's* readers, as proven by the frequency with which the *Journal* cover has been changed over the past nine decades.

In 1989, the society's Committee on Publications, Library and Archives, responding to expressed dissatisfaction with the then current cover, undertook an in-depth study of the *Journal's* overall design. A subcommittee consisting of Philip P. Bonanni, MD (Chair), Joseph I. Cohn, MD, and Joseph F. Muratore, MD, was appointed by Milton Gordon, MD, Chair of the Committee, to help the editor in designing a new cover. The assistance of a professional designer, Joan Simpson of Simpson's, New York City, was obtained. The subcommittee instructed Ms Simpson to produce a cover design that would be both attractive and reflective of the serious peer-reviewed scientific character of the *Journal*. She submitted several designs which incorporated an early decision to use a blue background with white lettering. Some modifications were made on one of Ms Simpson's designs by Ms Anne Rhymer of Mack Printing Company and Ms Carol Moore, the *Journal's* Managing Editor. The subcommittee members and the *Journal's* editors and staff arrived at a consensus which Ms Simpson then translated into the current design. This new design was approved by the subcommittee and recommended to the committee which adopted it at a meeting on May 23, 1990.

Three changes have been made in the *Journal's* design. The cover, now the *Journal's* 19th, is blue with white type and carries the titles of important articles. The table of contents, which is now spread over two pages on the inside of the *Journal*, is set in larger type than before and in a single column format across the page. The page numbers appear to the left and rules separate the various categories of articles, providing for greater clarity and ease in finding desired items. The new cover typeface is carried over to the masthead, the table of contents, and the first editorial page. Responding to the needs of readers who like to tear out articles or photocopy them for future reference, we have added the volume and number of each issue to the running foot of each even numbered page. The odd numbered pages continue to carry the month and year of publication. A Helvetica typeface is being used for the cover and is carried over to the table of contents pages for article titles. The authors' names are set on the table of contents pages in Times Roman which is also used in the body of the *Journal*.

The basic design of the editorial pages remains unchanged, as does the organization of the editorial content.

The Committee on Library, Publications and Archives is hopeful that this new cover design will be well received by the readership. It would be appreciated if readers would write the editor and make known their views about these design changes.

PASCAL JAMES IMPERATO, MD
Editor

The behavioral risks: Mother knows best

"Don't smoke. Drink moderately. Drive safely—and never drink and drive. Eat right. Exercise."

Maybe these injunctions are the reason physicians find it so difficult to deal with behavioral health risks. Why undergo all those years of training just to sound like Mom? And when we do try to appropriately counsel our patients, don't we often see in them the reactions Mom must have seen in us? ("Sure, Mom. I promise. Bye, Mom.") The difficulty of accomplishing changes in behavior further diminishes our enthusiasm for engaging these problems. Though we suspect that fully implementing even one of Mom's admonitions would have a more beneficial impact on the nation's health than any acute care we can provide, the inclination nonetheless is to sigh, "Alas! Human nature does not change."

And indeed, progress in these areas can appear glacial. Consider, for example, the contribution by Hyland et al in this issue of the *Journal.*[1] In their 1959 report on alcohol-associated single vehicle fatalities,[2] the Medical Examiner's Office of Westchester County noted blood alcohol levels of 0.10 mg/dL or greater in 58% of studied subjects; in their current report, 59% of studied subjects.[1] Have we made no progress? Injuries are the leading cause of death for persons aged 1 through 44 years[3,4]; the majority of these deaths are due to motor vehicle crashes (MVCs),[3,4] and approximately half of these involve a driver who by law was drunk.[2,5,6] Recognition of this problem is hardly new; in the 1959 report, reference is made to a 1934 study of alcohol levels among hit-and-run drivers, in which the author comments, "the problem of controlling the drinking driver . . . is far from being solved . . . "[2] Indeed!

Yet, led by colleagues such as Dr William Haddon, Jr, first author of the 1959 report, we have acquired a much better understanding of the intricacies of the MVC and drunk driving problems. For example, the 1959 report of Haddon and Bradess[2] was the first to identify the single-vehicle fatal crash as a category uniquely well suited for the investigation of alcohol and driving; this has since become a standard technique. In fact, alcohol is so prominently involved in such crashes that the lack of change in the proportion intoxicated from 1959 to 1990 is more a testament to the stability of the relationship than to an alleged lack of progress. In the earlier study, 102 deaths occurred in eight years; in the current study, 183 deaths occurred in eight years. Yet, the number of licensed drivers in New York increased from 6.2 million in 1955 to 9.8 million in 1985; the number of licensed automobiles, from 3.9 million to 7.7 million (personal communication, Arthur Valodze, Division of Research, New York State Department of Motor Vehicles). Vehicle miles travelled have similarly increased. Thus, the *rate* of such events has declined substantially, perhaps by 25% or more.

Dr Haddon was also among the first to focus on the young driver as particularly at risk for MVCs, and at especially elevated risk when alcohol was involved. Because of growing evidence to support these concerns,[7,8] within the last few years every state has raised the minimum legal drinking age to 21 years. The effectiveness of this tactic has been demonstrated elsewhere,[9] and is further supported by the observation of Hyland et al[1] of a dramatic decline in alcohol involvement in single vehicle MVCs fatal to drivers under age 21 following the raising of the legal drinking age to 21 years in New York.

Although the slowness of the pace can understandably lead to pessimism, in fact we are making important progress with respect to many of the behavioral risk factors that so powerfully influence life and health. Exercise and dietary risk factors now receive substantial attention, and this may have contributed to the recent decline in cardiovascular mortality.[10] Of great importance, the overall consumption of cigarettes is declining (although some groups remain at high risk). The consumption of alcohol is declining.[11] Rates of MVCs, and particularly rates of alcohol-associated MVCs, are declining. These favorable trends are occurring in the context of (indeed, are attributable to) fundamental changes in societal attitudes toward the behavioral risks. Behavior that once was found amusing, or at least tolerable, is no longer socially acceptable; and there is evidence that these attitudinal changes directly result in reductions in the behavior in question.[9,12] These facts should encourage us to continue our efforts to guide both our individual patients and our society toward minimizing these behavioral risks.

Hyland and his colleagues[1] direct our attention this month toward the involvement of alcohol in MVCs. What measures have been found to be effective in reducing this problem? Raising the drinking age to 21 years has produced a prompt and gratifying reduction in alcohol associated MVCs among the affected age group; any move to repeal such laws should be opposed.[9,13] Public education campaigns based in junior high and high school may be particularly effective in influencing the behavior of those young persons just beginning to make decisions about driving and about drinking.[9] Clearly, penalties for conviction of drunk driving should adequately reflect the severity of the problem and society's concern; however, there is evidence that unusually harsh penalties are not effective in reducing drunk driving, and may be counterproductive by reducing conviction rates.[14] In contrast, efforts to increase the rapidity and certainty of punishment appear to be more fruitful. One approach that has been successful elsewhere is the use of administrative suspension, in which suspension of the driving privilege is separated from the criminal prosecution of the offense.[15] Upon arrest for drunk driving, there is an automatic suspension of the driving privilege pending a prompt administrative hearing. This assures both rapidity and certainty of response. Concerns regarding unjustified suspension can be addressed by setting a minimum standard for evidence to support the suspension (for example, a blood alcohol level exceeding the established threshold). For those who drive drunk despite license suspension, vehicle impoundment or immobilization or the use of technologic barriers may be effective solutions.

There are societies in which the major health problems are amenable to the therapeutic wizardry in the physician's bag; ours is not one of them. Fundamental improvements in the health status of our nation will depend on our success in altering the major behavioral risk factors—and our efforts are being increasingly successful. Physicians have a vital role to play in this process—and Mom would have it no other way.

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Uninvited guests: Cardiovascular foreign bodies

The discovery of an embolized intracardiac needle, reported in this issue of the *Journal*,[1] reminds us of the increasing problem of cardiovascular foreign bodies. Intravascular foreign bodies have long been a subject of interest, challenging the physician in terms of their clinical recognition, detection, anatomic localization, and subsequent management. The varied situations in which they occur can range from the battlefield to the street corner, and from the kitchen to the intensive care unit. Intravascular foreign bodies may be grotesquely apparent or virtually invisible, totally unexpected or meticulously planned, but all have the common denominator of traumatic access to the cardiovascular system.

Clinical recognition of the process is sometimes difficult, as few patients, unlike the patient reported here, will announce their suspicions of an intravascular foreign body. Diagnosis more likely depends on clinical symptoms and/or an awareness of a predisposing incident. With acute missile injuries, the approach is straightforward—careful evaluation of entrance and exit wounds and radiographs of the injured area. If all projectiles cannot be accounted for, a radiologic search for the embolized bullets or fragments is indicated. Symbas[2] has proposed a detailed algorithm for the management of projectiles in the heart and great vessels. Contemporary surgical management of intracardiac foreign bodies is offered by Culliford[3] and Rutherford and Campbell.[4]

Objects can gain access to the cardiovascular system by a more insidious or indolent means. Intravenous drug abusers are a growing population at risk for cardiovascular foreign bodies. Venous injury may be manifested by thrombophlebitis, skin abscesses, and cellulitis, while arterial injury may result in ischemia, false aneurysm formation, and arteriovenous fistula.[5] The frequency of needle embolization is less clear, with few cases being reported. This complication would be suspected based on history or symptoms, as well as by chance.[6] Once the diagnosis is considered, detection and localization are the next steps; subsequent management depends on symptoms, location of the object, and the risks and benefits of intervention.

Perhaps the most troublesome intravascular foreign bodies are those introduced iatrogenically. As technology has allowed some diagnostic and therapeutic procedures to be more invasive, numerous reports have appeared detailing lost or migrating catheter or guide wire fragments, central venous lines, Swan-Ganz catheters, coils, various shunts and valves, pacing wires, filters, angiographic dilators, and other intravascular paraphernalia.[7-9] With the increasing use of in-dwelling catheters and more outpatient and home intravenous therapy, not to mention intravascular assist devices, the frequency of iatrogenic intravascular foreign bodies will likely increase. Physician awareness of this complication in these specific patient populations is warranted.

There have been many reports of embolized catheter fragments in the literature.[8,9] These reports suggest that serious complications or death result in 61% to 75% of patients in whom embolized catheter fragments are not removed. Retrieval would appear imperative. Techniques for nonsurgical percutaneous removal, associated with low patient risk and low cost, are both available and successful,[9] and in some instances can be performed on an outpatient basis.

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Death takes a ride

Alcohol-associated single vehicle fatalities revisited

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ABSTRACT. Records from the Medical Examiner's Office of Westchester County (1980 population = 866,599) were studied for trends in blood alcohol levels in drivers who died after single vehicle accidents. Findings for all motorists (N = 102) who died in the earliest available eight-year period, 1952–1959, were compared with findings for all motorists (N = 183) who died from 1980 to 1987. The mean age of subjects in the two periods did not differ (37.3 yr ± 15.4 SD versus 35.4 yr ± 16.8 SD), and mean blood alcohol levels were nearly identical (0.14% ± 0.12 SD) versus 0.13% ± 0.12 SD). Based on the increase in vehicular miles travelled during the two periods, there may have been a 20–25% reduction in alcohol-associated mortality over a 30-year period. Following legislation raising the minimum drinking age to 21, no fatally injured drivers 20 years of age or under have had blood alcohol levels ≥0.10%.

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In 1959, the Office of the Medical Examiner of Westchester County published an early study of the relationship between drinking and fatal single vehicle accidents.[1] Postmortem blood alcohol levels revealed that approximately half of these fatally injured drivers had blood alcohol levels ≥ 0.10 g/100 mL (0.10%), a level indicative of driving while impaired. Since then, these findings have been confirmed,[1–5] and alcohol-associated vehicular accidents continue to be an enormous public health problem.[6]

The aim of this study was to review and extend the original Westchester County report and to compare the early findings with more recent observations.

METHODS

Westchester County has an area of 435 square miles and is located immediately north of New York City. It had a population of 752,406 in the 1950 census, which had increased to 866,599 by the 1980 census. In both periods, the population was located mostly in urban or suburban centers, although the northern part of the county can be considered semirural. There is a single Medical Examiner's Office, and blood alcohol determinations are performed at the time of autopsy for all motor vehicle fatalities.

Fatally injured motorists involved in single vehicle accidents who died within four hours of injury were chosen for study, because all of these subjects were autopsied. In these victims, blood alcohol levels can be considered representative of blood levels at the time of injury, enabling a determination of responsibility for the accident. During both study periods, approximately 20% of all fatalities classified as traffic deaths were attributable to high blood alcohol levels in the driver.

The group included in the initial 1959 report consisted of 83 subjects who died in Westchester County during 1950–1957 after a fatal single motor vehicle accident. Prior to 1952, some alcohol determinations were performed on tissue removed from the brain or liver, but from 1952 onwards all alcohol determinations on fatally injured subjects were based on blood samples. Therefore, for this study subjects who died during the years 1952–1959 were included and were compared with subjects who died during the years 1980–1987. The records of all subjects who died within four hours after a single motor vehicle accident were available and were reviewed to ascertain age, sex, race, permanent address, date and time of injury, as well as postmortem blood alcohol level. For both time periods, a blood alcohol level of 0.1 g/100 mL (0.10%) or greater was considered to indicate driving while impaired (DWI).

It should be noted that during the second study period (1980–1987), New York State passed a law raising the minimum legal drinking age to 21 years. This law was in effect for the years 1986 and 1987.

For statistical analysis, between-group differences were determined using t-tests, chi-square analysis, and Fisher's exact test. For multiple group comparisons, analysis of variance was performed. For all comparisons, two-tailed tests with a predetermined level of significance of 0.05 were used.

To compare mortality rates for the early and most recent periods, we used data supplied by the New York State Department of Transportation (N.S. Erlbaum, personal communication, August 2, 1989). For the entire state, the ratio of vehicular miles driven in the most recent period compared to the earlier period was 2.1/1. The expected number of fatally injured drivers in the most recent period was calculated by multiplying the number of fatally injured drivers in the early period by 2.1. The 95% confidence interval for the number of fatal injuries in the first period was obtained from available tables by regarding observed cases as a Poisson variable.[7] These upper and lower values, when multiplied by 2.1, gave the confidence interval for the number of expected cases in the most recent period.

RESULTS

For the periods 1952–1959 and 1980–1987, characteristics were compared by year. Since the intra-year variation within these two groups was not significant for any of the variables included in the study, the data for individual years within these two time periods were combined to form two groups: period I (1952–1959) and period II (1980–1987). Table I compares the characteristics of these two groups. No significant differences were observed with respect to age, sex, race, residence, or timing of accident. The mean blood alcohol

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TABLE I. Comparative Findings in 285 Fatally Injured Motorists During Two Time Periods (Westchester County, NY)

Variable	Period I (1952–1959) Number (%)	Period II (1980–1987) Number (%)
Total subjects	102 (100)	183 (100)
Males	92 (90)	151 (83)
Females	10 (10)	32 (17)
Age (yr)*	37.3 ± 15.4	35.4 ± 16.8
<21	12 (12)	30 (16)
≥21	90 (88)	153 (84)
Race		
White	94 (92)	170 (93)
Nonwhite	8 (08)	13 (07)
Resident of county		
Yes	70 (69)	127 (69)
No	32 (31)	56 (31)
Time of accident		
Day (6 AM–6 PM)	37 (36)	65 (36)
Night (6 PM–6 AM)	65 (64)	118 (64)
Weekday (Mon–Fri)	58 (57)	86 (47)
Weekend (Sat–Sun)	44 (43)	97 (53)
Blood alcohol findings		
Alcohol detected	74 (72)	120 (66)
BAC ≥ 0.1%	62 (61)	106 (58)
BAC (g/100 mL)*		
All subjects	0.14 ± 0.12	0.13 ± 0.12
Alcohol-positive subjects	0.20 ± 0.09	0.20 ± 0.09

* Mean ± standard deviation.

level in the first period was 0.14% ± 0.12 SD and was nearly identical to the observed level of 0.13% ± 0.12 SD in the second period. Furthermore, the proportion of subjects who were driving while impaired (blood alcohol level (BAC) ≥0.1%) in the first period, 61%, was almost the same as in the second period, 58%.

Seventy-two percent of fatally injured drivers in the early period had consumed alcohol prior to their accident, as compared to 66% in the second period. This difference was not significant ($p = 0.23$). The mean blood alcohol level of fatally injured alcohol consumers in the two periods was identical: 0.20% ± 0.09 SD.

Table II contrasts the total number and the number of fatally injured intoxicated (BAC ≥ 0.1%) drivers by age in the two time periods. For both periods, the largest number of fatalities occurred in the 21–30-year age group; nearly three quarters of these subjects had BAC levels greater than or equal to 0.1%. However, in 1986 and 1987, subsequent to enactment of a state law that raised the minimum legal drinking age to 21 years, none of the six fatally injured drivers age 20 or younger had blood alcohol levels greater than 0.1%.

TABLE II. Age and Alcohol-associated Motor Vehicle Fatalities in Two Time Periods (Westchester County, NY)

	Period I	Period II	
Age (yr)	1952–1959 No. Subjects	1980–1985 No. Subjects	1986–1987* No. Subjects
<21	12 (6)[†]	24 (15)	6 (0)[‡]
21-30	29 (22)	47 (35)	16 (12)
31-40	26 (18)	34 (24)	5 (3)
41-50	18 (10)	9 (4)	3 (2)
51-60	5 (4)	18 (9)	0 (0)
>60	12 (2)	15 (2)	6 (0)
Total	102 (62)	147 (89)	36 (17)

* Years in which minimum legal drinking age was 21 years.

[†] Number in parentheses is number of subjects with blood alcohol levels ≥ 0.1%.

[‡] Differs significantly from all previous years ($p = 0.021$, Fisher's exact test).

Only one subject had a positive blood alcohol test of 0.07%. This contrasts sharply with all previous years, in which we found that 21 of 36 of these youngest drivers had BAC levels ≥0.1% ($p = 0.021$, Fisher's exact test).

Table III compares observed with expected fatalities in the two time periods, after adjusting for the increased number of vehicular miles travelled in the recent study period. For subjects in whom alcohol was absent at the time of fatal injury, the mortality ratio (observed/expected deaths) appears unchanged from the 1950s to the 1980s. However, for consumers of alcohol, there appears to be a reduction in the mortality ratio to 0.77 (95% confidence limit = 0.62–0.98).

DISCUSSION

Many of the findings in this study are similar to those in our 1959 report. The mean blood alcohol level, the proportion of drivers with BAC levels ≥ 0.1%, and the ages of motorists involved in fatal single vehicle accidents are virtually unchanged. In both periods, subjects in the third decade constituted the largest age group, whereas subjects aged 65 and older contributed less than 10% of the cases.

In 1982, the fatal accident reporting system (FARS) began collecting data on alcohol involvement in motor vehicle accidents. Since then, there has been a reported 17% decrease in the proportion of drivers who were intoxicated at the time of a crash.[8] However, blood alcohol information is not available for all of these drivers, and decreases were minimal for the type of drivers most frequently represented in our sample, younger subjects involved in nocturnal accidents.

Vehicular travel data for New York State have been used to calculate mortality ratios for the two study periods. For the entire state, vehicular miles driven increased by a factor of 2.1 between the two time periods. Assuming that the same increase holds true for Westchester County, it appears that there has been little change in the mortality ratio (observed/expected number of deaths) for alcohol-free drivers, but there seems to have been a reduction in the mortality ratio for drivers who had consumed alcohol prior to injury in the period from 1980 to 1987. Nearly identical estimates were obtained when the number of motor vehicle registrations in the two periods was used for adjustment. Because detailed driving information by county is unavailable, these estimates are not adjusted for race, sex, or age. Furthermore, the increase in vehicular miles travelled for Westchester County and New York State may well differ. Nevertheless, the data from this study are compatible with a 20–25% reduction in alcohol-associated highway fatalities within Westchester County over a 30-year period.

Subsequent to a law passed in December 1985, increasing the minimum legal drinking age to 21 years, a significant reduction in alcohol-associated deaths in subjects less than 21 years of age has been observed. The results of this study are consistent with previous findings[9] and suggest that raising the minimum legal drinking age has been an effective legislative strategy.

Strict enforcement of existing laws as well as additional legislative and educational measures will undoubtedly be required to reduce the excess mortality still caused by drunk driving.

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TABLE III. Mortality Ratios for Fatally Injured Westchester County Drivers: 1980–1987 Versus 1952–1959

Alcohol	1952–1959	1980–1987		Mortality Ratio
Status	Observed	Observed	Expected[*]	Observed/Expected
Absent	28 (19-40)[†]	63	59 (40-84)	1.1 (0.80-1.60)
Present	74 (58-93)	120	155 (122-195)	0.77 (0.62-0.98)

[*] Estimated from ratio of 2.1 for average number of vehicular miles travelled for recent versus earlier period in New York State.

[†] Numbers in parentheses represent 95% confidence limits based on assumption of a Poisson distribution.

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Drug resistant tuberculosis among the homeless in New York City

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ABSTRACT. The purpose of this study was to compare the frequency of infections due to drug resistant *Mycobacterium tuberculosis* between homeless and nonhomeless populations in an urban community hospital.

We retrospectively examined the mycobacteriology registry of this hospital for culture-proven, susceptibility-tested cases of *M tuberculosis* disease from 1982 to 1987. Clinical records were analyzed for age, ethnicity, sex, residence, and history of alcoholism, intravenous drug abuse, acquired immunodeficiency syndrome, or previous tuberculosis.

Of a total of 132 cases, 53 patients were homeless and 79 were not. Drug resistance was found in 21% of the isolates from homeless individuals and 8% of those from nonhomeless persons. Resistance to isoniazid was found in 19% and 1% ($p < 0.01$), and for two or more drugs in 15% and 0% (p, 0.01), respectively.

Forty-two patients were black, and among them, resistance to one or more drugs was 26.2%, versus 6.6% in nonblacks ($p < 0.01$). Ten of 24 homeless blacks had resistant organisms, as opposed to 1 of 18 nonhomeless blacks ($p < 0.05$). No other significant differences were noted for the other variables.

To our knowledge, this is the first comparison of drug-resistant tuberculosis between homeless and nonhomeless patients using an internal control group. Forty percent of isolates from homeless blacks were resistant, two thirds to both isoniazid and rifampin. These findings support the use of an initial four-drug regimen to treat tuberculosis in this specific population.

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As in the rest of the western world, tuberculosis in the United States has markedly decreased during the last half century. In recent years, however, tuberculosis has made a comeback.[1] The acquired immunodeficiency syndrome and other factors have been cited to explain this resurgence.[2-4]

Throughout the centuries, poor people living in crowded conditions have frequently been the victims of tuberculosis. Among certain homeless populations, prevalence rates of 1.6–6.8% for clinically active disease and of 22–50% for asymptomatic infection have been reported. These rates are 150 to 300 times higher than the nationwide rate and similar to the rates in populations with acquired immunodeficiency syndrome.[5-7] In a recent outbreak of tuberculosis among the homeless in Boston, the drug resistance rate was as high as 60%, suggesting that shelters may be contagious foci for difficult-to-treat tuberculosis.[8]

Drug resistant tuberculosis is a problem for contemporary control programs and has increased since the first reports in the 1960s.[9] Several factors have been associated with resistant tuberculosis: previous treatment, recent residence in or birth in an endemic area, and exposure to drug resistant cases.[10-12] Many of these conditions are prevalent among the homeless, as are alcoholism, malnourishment, an adverse sociomedical environment, and chemotherapeutic noncompliance.[13-15]

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Since optimal antituberculosis treatment can dramatically reduce morbidity and mortality, prescribing an appropriate initial regimen is as important as proper follow-up.[16] To accomplish this in homeless patients, we considered it important to evaluate comparatively the rates of in vitro drug resistance in the hospital setting where most homeless persons are diagnosed and initially treated.

PATIENTS AND METHODS

This was a retrospective clinical study with an internal control group. It was conducted at Cabrini Medical Center, an urban community hospital of 493 beds in New York City.

All consecutive culture-proven, susceptibility-tested cases of *Mycobacterium tuberculosis* disease from 1982 to 1987 were reviewed. A total of 132 cases were analyzed. Five homeless patients were admitted twice during the study period with a diagnosis of tuberculosis, but only their last admission was considered for analysis. One other homeless patient was readmitted, but sets of data from both admissions were considered for analysis, as susceptibility patterns differed for the two admissions. One nonhomeless, 73-year-old man diagnosed in 1986 was excluded, as susceptibility patterns for his case could not be obtained.

For the purposes of this study, homelessness is defined as having no customary and regular access to a conventional dwelling or residence. We considered as homeless patients who live on the street or in a single room occupancy hotel or shelter, according to a list of such places compiled by the New York City Tuberculosis Homeless Program. Resistance is defined as the "demonstrated ability of the patient's tubercle bacilli to survive and multiply in elevated concentrations of a specific antituberculosis agent unlikely to be attained in the body of the infected host."[17] Primary resistance occurs in patients who have not received any prior antituberculosis treatment (this is attributed to acquisition of drug resistant mycobacteria), whereas secondary resistance is related to previous, failed drug therapy.

The mycobacteriology registry for the period 1982–1987 was reviewed, and all cases in which *M tuberculosis* had been cultured were analyzed. The patients' medical records and pertinent data were obtained. For this purpose, personal characteristics (sex, age, race, ethnic group), epidemiologic data (alcohol abuse, intravenous drug abuse, and history of acquired immunodeficiency syndrome or tuberculosis exposure), and microbiologic information (previous treatment, type of specimen, and susceptibility results) were tabulated. Special emphasis was given to distinguishing referred ("private") from nonreferred ("house") patients, and homeless from nonhomeless patients.

When mycobacterial growth was present, biochemical identification of the species was performed in our laboratory. The specimen was then sent to the New York City Department of Health Mycobacteriology Unit for final identification, and for in vitro drug susceptibility evaluation by the conventional method, as recommended by the Centers for Disease Control.[18] Susceptibility was tested for the following drugs and concentrations (μg/mL): isoniazid 1,2 and 1,5; ethambutol 2 and 5; rifampin; and streptomycin 2 and 5, with appropriate controls. Organisms were considered resistant to a given drug when any growth of 1% or more above the control was observed at low or high drug concentration. Ethionamide resistance was not analyzed because resistance is very prevalent (25 out of 54 in this study), and clinical use has declined; the same decision was made concerning *p*-aminosalicylic acid but for the opposite reason—all 37 cases evaluated showed sensitivity in vitro.

A prospective analysis of the cohorts was done. Variables were described with mean and standard deviation. Comparison of subgroups' characteristics was done with the chi-square test using Yates' correction for discrete variables. Continuous variables among subgroups were compared with the two-sample t test.

RESULTS

A total of 132 cases were suitable for analysis (Table I). Most of the patients (113) were male (85%), with a mean age of 49 years (standard deviation (SD), 16 yr). Seventy-four patients were nonreferred and 48 were referred; admission status was not determined for ten patients. The racial distribution showed a predominance of whites (34%) and blacks (32%), with Hispanics accounting for 19% and Asians for 11%; in 4% ethnic group was not specified (Table I). Fifty-three (40%) patients were considered homeless.

TABLE I. Personal Characteristics of the Study Population

Characteristic	Number
Admission status	
House service	74
Private service	48
Unspecified	10
Sex	
Male	113
Female	19
Race	
Asian	14
Black	42
Hispanic	25
White	45
Unspecified	6
Age (mean in years)	49
Total	132

One fifth of the tuberculosis patients were intravenous drug abusers and almost half were alcoholics, according to their clinical records (Table II). Among those for whom data were available, 23 patients (22%) admitted to a history of previous tuberculosis, although treatment characteristics and compliance could not be reliably determined. Because of this, cases could not be classified as primarily or secondarily resistant.

Seventy-eight percent of the specimens came from the lung (including seven from pleura), and among the remaining 22% there were nine from bone marrow, five from urine, three from blood, two from lymph nodes, and one from the pericardium. Reports of acid-fast bacilli stains were found in 101 patient charts: an average of 4.8 (SD, 3.5) specimens were sent per case, and 30 were reported negative (18 of which were specimens from other than sputum). According to the number of specimens reported, 5/17, 5/10, 13/19, and 48/55 cases were positive for one, two, three, or more than three specimens, respectively.

Of the 132 *M tuberculosis* isolates, 17 (13%) were resistant to one or more drugs and eight (6%) to two or more drugs. Table III shows the patterns of resistance for each of the four drugs considered in this analysis. Isoniazid resistance was found in 11 (8%) of the cases,

TABLE II. Epidemiologic Characteristics of the Study Population

Characteristic	Number
Intravenous drug abuse	
Present	23
Absent	93
Unknown	16
Alcohol abuse	
Present	52
Absent	63
Unknown	17
Previous tuberculosis	
Present	23
Absent	79
Unknown	30
Source of specimen	
Lungs	103
Other	29
Total	132

TABLE III. Patterns of Drug Resistance for Several Antituberculosis Agents Between Homeless and Nonhomeless Patients

	Number	Resistance to $\geq$ 1 Drug	Isoniazid	Rifampin	Streptomycin	Ethambutol	Resistance to $\geq$ 2 Drugs
Total	132	17 (13%)	11 (8%)	10 (8%)	6 (5%)	3 (2%)	8 (6%)
Homeless	53	11 (21%)	10 (19%)	7 (13%)	4 (8%)	3 (6%)	8 (15%)
Nonhomeless	79	6 (8%)	1 (1%)	3 (4%)	2 (4%)	0 (0%)	0 (0%)
p value		NS*	<0.01	NS	NS	NS	<0.01

* NS = not significant.

whereas resistance to ethambutol was documented in only three (2%). Resistance among the homeless patients (21%) was higher than among the nonhomeless (8%), and all eight isolates resistant to more than one drug were obtained from homeless patients ($p < 0.01$). Resistance to isoniazid was also significantly higher among the homeless (19%) than the nonhomeless (1%) ($p < 0.01$).

Drug resistance prevalence for the other clinical and epidemiologic variables considered in the study is shown in Tables IV and V. Admission status ("house" vs private service) had no effect on tuberculosis resistance frequency (11% and 10%, respectively). Drug resistance among males (14%) was higher than among females (5%), but this did not reach statistical significance. Patients of Asian or Hispanic origin had average resistance rates (14% and 12%). Isolates from blacks were resistant in 11 (26%) of the 42 cases ($p < 0.01$); seven of these 11 cases were resistant to two or more drugs. Patients with resistant isolates were slightly younger than patients with nonresistant ones, with a mean age of 43 and 50 years, respectively; those with resistance to two or more drugs had a mean age of 39.6 years ($p < 0.05$). Blacks were also younger (42 years) than whites (60 years), and this was statistically significant ($p < 0.025$); there was no significant difference in age between homeless (47 years) and nonhomeless patients (49 years).

Of the eight patients in whom a diagnosis of acquired immunodeficiency syndrome had been established, two (25%) were found to yield drug resistant isolates; four of these patients were Hispanic, aged less than 43 years, and two of them were women. In two cases, *M tuberculosis* was cultured from the blood (only three cases in the total sample), and in one from the bone marrow. Six of these patients entered the study population in 1987.

A history of intravenous drug abuse or alcohol abuse did not increase resistance prevalence in this study, as shown in Table V. Four of 23 (17%) isolates from patients with a previous history of tuberculosis were resistant, as opposed to six of 79 (8%) without a history of the disease, but this difference did not reach statistical significance. These data should be evaluated with caution because they were collected retrospectively from clinical charts, and patients' assertions may be unreliable. No difference in resistance could be established among specimens from the lung and other sources in this work.

To better define the characteristics of the 53 homeless cases, further analysis was performed (Tables VI and VII). As expected, 40 (89%) of the 53 homeless patients were nonreferred or admitted as "house" patients ($p < 0.01$), and 52 (98%) were men ($p < 0.01$). Age was similar among the two subgroups of tuberculosis patients. Blacks constituted 48% of the homeless population, but only 33% of the total study population. Twenty-four (57%) of all 43 blacks with tuberculosis were homeless. Whites represented 42% of homeless patients, and 34% of the total number of cases. Hispanics and especially Asians were underrepresented among the homeless population with tuberculosis (Table VI).

Because resistance among the homeless (21%) could be partly explained by resistance among the over-represented black population (26%), a further analysis was made. We found that among the 42 blacks with tuberculosis, 24 were homeless, with resistant disease in 42%; whereas among the 18 nonhomeless blacks, only one patient was resistant to one or more drugs ($p < 0.01$). Among the 53 homeless, 24 were black, with drug resistance in ten ($p < 0.005$). Among 90 nonblack patients, there was no significant difference in drug resistant tuberculosis between the homeless and the nonhomeless.

There were more intravenous drug abusers among the nonhomeless (23%) than among homeless tuberculosis patients (16%), but the difference did not reach statistical significance (Table VII). However, the homeless had a significantly higher prevalence of alcohol abuse (68%) as compared to the nonhomeless (31%) ($p < 0.05$). A history of previous tuberculosis was obtained for 19% of 53 homeless patients and for 16% of 79 nonhomeless patients. Specimens came from the lung in 89% of homeless cases as opposed to 68% of the nonhomeless ($p < 0.05$). The number of drugs initially ordered by physicians to treat tuberculosis was also quantified. An average of 2.3 drugs were used in homeless patients and 2.2 in the nonhomeless (no statistical difference). Twenty-three (17%) patients were not treated in the hospital, either because the patients died or because they were discharged before culture results were reported.

TABLE IV. Drug Resistance Prevalence According to Several Clinical Characteristics

Characteristic	Number	Drug Resistance Present	Drug Resistance Absent	Drug Resistance $\geq$ 2 Drugs
Admission status				
House service	74	8	66	4
Private service	48	5	43	1
Unspecified	10	4	6	3
Sex				
Male	113	16	97	8
Female	19	1	18	0
Race				
Asian	14	2	12	0
Black	42	11	31	7
Hispanic	25	3	22	1
White	45	1	44	0
Unspecified	6	0	6	0
Age (mean in years)	49	43	50	40
Total	132	17	115	8

TABLE V. Drug Resistance Prevalence According to Several Epidemiologic Variables

Characteristic	Number	Drug Resistance Present	Drug Resistance Absent	Drug Resistance $\geq$ 2 Drugs
Intravenous drug abuse				
Present	23	3	20	1
Absent	93	10	63	5
Unknown	16	4	12	2
Alcohol abuse				
Present	52	6	46	5
Absent	63	7	56	1
Unknown	17	4	13	2
Previous tuberculosis				
Present	23	4	19	2
Absent	79	6	73	3
Unknown	30	7	23	3
Source of specimen				
Lungs	103	14	89	6
Other	29	3	26	2
Total	132	17	115	8

TABLE VI. Clinical Characteristics of Homeless and Nonhomeless Patients

Characteristic	Number	Homeless	Nonhomeless
Admission status			
House service	74	40	34
Private service	48	5	43
Unspecified	10	8	2
Sex			
Male	113	52	61
Female	19	1	18
Race			
Asian	14	1	13
Black	42	24	18
Hispanic	25	4	21
White	45	21	24
Unspecified	6	3	3
Age (mean in years)	49	48	49
Total	132	53	79

DISCUSSION

Although the incidence rate for tuberculosis in New York declined by 72% between 1960 and 1978, since then rates have increased by 50%, especially among young black males. Epidemiologic and clinical data have shown that the acquired immunodeficiency syndrome may predispose to tuberculosis.[2–4] Other possible explanations for the apparent increase are a more vigorous case finding campaign, an influx of immigrants from countries with endemic tuberculosis (Mexico, Philippines, Haiti, Korea, and Vietnam), and an increase in poverty and intravenous drug abuse.[2]

Medical progress has not equally reached all levels of society. The British Registrar General's statistics dramatically show that for males aged 15–64 years, the standardized mortality ratio for pulmonary tuberculosis is 25 for professionals and 250 for unskilled workers.[19] As McAdam et al[5] expressed it, "At times it seems the homeless and shelter residents live in the preantibiotic era because of the difficulties they have in negotiating the complex care system."

Unfortunately, there is no clear epidemiologic surveillance of the homeless. As a working definition does not exist, the federal government estimates are understatements of the real magnitude of the problem.[20–22] The first rigorous attempt to apply social science research to study the homeless was undertaken in Chicago in 1985–1986.[23] The point prevalence estimate of the city homeless population was about 0.07%, although the annual prevalence estimate is three times higher due to the high "turnover rate" of this population. Homelessness and congregate shelter living have increased in New York City, and this has contributed to the high case rates of tuberculosis seen recently, especially in Central Harlem and the Lower East Side.[2]

TABLE VII. Additional Epidemiologic Characterization of Homeless and Nonhomeless Patients

Characteristic	Number	Homeless	Nonhomeless
Intravenous drug abuse			
Present	23	7	16
Absent	3	38	55
Unknown	6	8	8
Alcohol abuse			
Present	52	30	22
Absent	63	14	49
Unknown	17	9	6
Previous tuberculosis			
Present	23	10	13
Absent	79	29	50
Unknown	30	14	16
Source of specimen			
Lungs	103	47	56
Other	29	6	23
Total	132	53	79

Several factors influence the likelihood of drug resistant tuberculosis, including previous treatment (especially if it failed), birth or recent residence in an area endemic for tuberculosis (Asia, Latin America, Africa), and exposure to cases of drug resistant disease (common in shelters).[10,11] Primary drug resistance (no previous treatment) is estimated to be present in 8.6% of the cases of tuberculosis in the United States, with variation depending on the geographic area and ethnic population.[24,25] Prior therapy can increase drug resistance to 7–53%,[12] and cases secondarily resistant are considered more contagious.[26] Some useful algorithms have been developed to reduce the chance of treatment failure.[10]

Among 132 cases of tuberculosis seen at Cabrini Medical Center during a seven-year period, 53 patients were recorded as homeless (40%) and 79 as nonhomeless (60%). Resistance to one or more drugs in this population was 13%, and to two or more drugs was 6%, which is in accordance with results of other epidemiologic studies.[27] We did not observe a cluster of resistant cases in any specific year or shelter. Isoniazid and/or rifampin resistance was found in 8% of the isolates. When we analyzed frequency of resistance, we found it to be almost three times higher in homeless than in nonhomeless patients (21% vs 8%).

Referral status and sex did not influence drug resistance. However, patients with resistant isolates were younger than those with nonresistant isolates; this has been previously described.[25] The fact that nonwhite patients with higher drug resistance rates are also younger than white patients with tuberculosis emphasizes this relationship. We can hypothesize that a high prevalence of drug resistant tuberculosis among the homeless, although possibly generated by poor therapeutic compliance (secondary resistance), may be explained or magnified by acquisition in shelters of primary tuberculosis (with primary drug resistance) by young nonwhite individuals; in fact, exogenous reinfection has been reported in a shelter for the homeless.[13]

Contrary to expectations and other reports,[28] the few Asian and Hispanic patients in this study failed to show increased resistance rates. On the other hand, resistance was exceedingly rare in white patients (2.2%) and remarkably common in black patients (26.2%); in both cases, the results reached statistical significance. To exclude the possibility that the high resistance rate among blacks is explained simply by their over-representation among the homeless (48%), a subanalysis was performed that showed that resistance was truly higher in black homeless than in black nonhomeless patients ($p < 0.05$). Similarly, among the 53 homeless patients, resistance was present in ten of 24 blacks and in only one of 29 patients of other race ($p < 0.005$). Thus, there appears to be no doubt that being homeless and black predisposes one to higher resistance rates. We do not know why blacks had a higher rate of drug resistant tuberculosis. Associated social determinants may play a role.

A previous history of tuberculosis, in most cases with treatment failure or noncompliance, predisposed to increased resistance (17.4% vs 7.6%). The lack of statistical significance should be judged with caution, since the compared numbers were small and a retrospective study could fail to allow precise evaluation of this variable, which certainly would help to tailor more detailed recommendations. Alcohol abuse and intravenous drug abuse were not found to increase the frequency of drug resistance. Once again, these data must be interpreted with caution, for the same reasons as discussed. Only a prospective study would demonstrate the importance of these variables, the significance of which has been previously recognized.[5,29]

As seen in other surveys, the lungs were involved in most of the cases of tuberculosis; this was significantly remarkable among the homeless in this study, with their obvious risk for infectivity.

Only eight patients in this study population had a diagnosis of acquired immunodeficiency syndrome. Two of them had drug-resistant tuberculosis, and two of the three isolates from blood belonged to these patients, showing that systemic dissemination is common in patients with AIDS. It is possible that several isolates from organs other than lungs could have been from patients with undocumented human immunodeficiency virus infection.

Certainly the problems of the homeless are complex. Specifically, tuberculosis control faces several hurdles: lack of compliance on the one hand, and lack of globally effective strategies on the other. Several approaches have been suggested,[6,30,31] including the use of public health laws to confine patients unable to follow proper outpatient treatment protocols and who thus become a hazard to the community.[6,32–34] Education and motivation are important tools, as well as choosing the proper drugs for effective treatment.

The present study demonstrates the high frequency of drug resistance, especially to isoniazid and rifampin, among homeless patients. Moreover, it shows that black individuals who live in shelters, single room occupancy hotels, and on the streets of New York City carry highly resistant strains of mycobacteria. In fact, 40% of isolates from homeless blacks were drug resistant, and two thirds of these were resistant to both isoniazid and rifampin.

In this institution, an average of 2.3 drugs was initially being prescribed for patients with tuberculosis. This study supports the use of an initial multidrug regimen to treat tuberculosis in homeless individuals, as recommended by the Centers for Disease Control,[7,35] and allows one to conclude that four drugs are mandatory when a young homeless black patient presents with this disease. Although the study did not evaluate clinical outcome, it seems safer to keep a high risk patient in the hospital until he becomes negative for acid-fast bacilli or until results from susceptibility testing are available.

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Fibrous disease of the breast in juvenile diabetes

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ABSTRACT. The association between Type I diabetes mellitus and breast masses whose extreme hardness suggests cancer has only recently been recognized. The present report consists of 11 cases of patients with juvenile diabetes who were identified by a retrospective review of 3,219 patients seen in our breast clinic over an eight-year period. All 11 patients were referred for possible biopsy and this was carried out routinely before the condition was recognized. The patients with diabetes were carefully reviewed in order to identify specific features of the breast lesions and a management plan was devised that should reduce the need for biopsy in these patients in the future.

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The lesion classified as fibrous disease of the breast is a localized but not encapsulated proliferation of breast stroma—it is really not a disease entity. Clinicians know this condition as one part of what was formerly called "fibrocystic disease," but is now more accurately regarded as nodularity characteristic of breast tissue during the reproductive years. We have recently recognized that this process may progress to such an extent that it mimics cancer in young women with Type I diabetes mellitus. A fuller awareness of the association between Type I diabetes and suspicious breast masses should help physicians better evaluate the need for breast biopsies in these patients.

The first report linking fibrous disease of the breast with Type I diabetes was by Soler and Khardori and appeared in 1984,[1] and the second one by Byrd and his colleagues was published in May 1987.[2] We independently noted the same relationship. Once this association is made, management of most of these patients is possible without the need for biopsy confirmation. This report confirms the findings of the two previous studies and identifies specific features of these lesions that should aid in differentiating between fibrous tumors and cancer.

PATIENTS AND METHODS

After seeing two juvenile diabetics with suspicious breast masses in the course of one week, we undertook a systematic review of all premenopausal women presenting to the Breast Service from January 1, 1980, to September 1, 1987. A review of 3,219 charts led to the identification of 11 patients with Type I diabetes. The clinical mammographic and pathologic features of these 11 patients were recorded in an effort to characterize more precisely the breast lesions associated with Type I diabetes. Emphasis was placed on the breast, since the Breast Service questionnaires did not provide sufficient information about the course and complications of the patients' diabetes.

RESULTS

The cases included in this report are summarized in Table I. The mean age was 35 years at the time of presentation with an abnormal breast mass; this is considerably younger than the usual age of patients seen on the Breast Service. All patients had long standing Type I diabetes mellitus, and the average duration between onset of the disease and the appearance of a breast mass was 23 years. Tests for thyroid microsomal antibodies were not performed; however, three of the 11 patients were known to be hypothyroid and were on thyroid replacement therapy.

Table I also details the clinical features of the breast lesions. Fine needle aspiration cytology was carried out in nine patients and was negative in seven. One patient was thought to have a fibroadenoma, while another had atypical cells suspicious for neoplasm. As would be expected, the typical aspirate contained few cells because of the fibrous nature of the tissue. There were no false-positive cytologic reports, and the absence of false-positives would be expected given the nature of the lesion.

Mammography was performed on ten of the 11 patients despite their youth, because the need for biopsy was seriously considered. All participating radiologists commented on the density of the breast tissue, but adverse findings were recorded in only one instance. Patient 2 was reported to have an area of parenchymal distortion, but it was present on only one view.

Bilateral breast masses were noted in four of the 11 patients at the time of presentation, but patient 3 had bilateral breast biopsies one year apart. Patient 4 had a suspicious lesion on the right with a possible area of thickening in the other breast. The remaining five patients had unilateral lesions that were considered to be dominant masses.

The physical findings led to biopsies in six patients; biopsy was advised in a seventh patient who refused. The bilateral and symmetrical nature of the findings convinced us that biopsy was unnecessary in two patients. The last two patients did not have biopsies, because they were the ones that led us to an appreciation of this entity.

DISCUSSION

The association between diabetes mellitus and breast tumors has only been appreciated in the past few years. The original report by Soler and Khardori[1] came from an endocrine clinic caring for patients with diabetes, and the 12 patients described constituted 13% of all female Type I diabetic patients between 20 and 40 years of age. It is noteworthy that the patients were identified by performing breast examinations on the juvenile diabetic women who attended the clinic. Not surprisingly, most patients had bilateral lesions (10 of 12), and concern about carcinoma was low. Six of the patients were sent for mammography, and only four actually had biopsies.

A very different perspective appears when such patients present to a Breast Service. Byrd's report described a total of eight patients from a group of several thousand breast

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TABLE I. Features of the Breast Lesions in Women with Juvenile Diabetes

Patient	Age (yr) at Presentation with Mass	Age (yr) at Onset of Diabetes	Breast Findings					
			Fine Needle Aspiration	Biopsy	Mammography	Right	Left	Bilateral
1	32	10	Negative	No	Negative		X	
2	41	12	Not done	No	Possible mass			X
3	39	15	Fibroadenoma	Fibrosis	Negative			X
4	40	12	Negative	Fibrosis	Negative	X	?	
5	28	14	Atypical cells	No	Negative			X
6	37	12	Negative	Refused	Negative	X		
7	27	11	Not done	No	Negative			X
8	35	14	Negative	Fibrosis	Negative	X		
9	40	12	Negative	Fibrosis	Negative		X	
10	35	8	Negative	Fibrosis	Negative	X		
11	27	9	Negative	Fibrosis	Not Done			X

biopsies done over a ten-year period.[2] All these patients had biopsies because of concern about carcinoma. In fact, a total of 12 biopsies were performed on these eight patients. The high biopsy rate reflects the fact that the association between Type I diabetes and fibrous disease of the breast was not appreciated when these patients were encountered.

The same conditions prevailed in this series, with the exception of the last two patients, who served to focus attention on the true nature of the breast lesion. Biopsy may be avoided by awareness of the association between juvenile diabetes and breast lesions, and diagnostic guidelines can be developed based on a review of these patients.

Our experience suggests that about half of the diabetic patients present with bilateral masses, and despite the extreme hardness of these tissues, it would not be necessary to perform biopsies on such patients provided the process is symmetrical. Patients with unilateral lesions constitute a more difficult clinical situation and may require modification of the dictum that all dominant breast masses require a tissue diagnosis. Fine needle aspiration should always be done, despite the accepted fact that there are false negatives. This procedure constitutes a worthwhile first step in such patients. Mammography would follow, and although Byrd believed these lesions to be indistinguishable from breast malignancy on radiographic evaluation, that has not been our experience. Mammograms done on ten patients showed only dense fibrous tissue without microcalcifications or other adverse findings. Follow-up mammograms would depend on the patient's age, since we have limited such studies in women under the age of 30. Ultrasound would avoid radiation, but none of the patients in this series were studied with this technique.

Similarly, the clinical findings were not suspicious, other than the extreme hardness of many of the lesions. Specifically, there were no skin changes or distortion of the breast contour. However, a hard dominant mass is generally considered a clear indication for a biopsy. At times, the age of the patient may be helpful, in that the incidence of breast cancer is extremely low below the age of 25. In women older than 25, the unusually firm texture of the contralateral breast tissue, even though a dominant mass is lacking, may be sufficient to convince the clinician of the presence of diabetic mastopathy.

Finally, it may be possible to combine diagnostic techniques, all of which can produce false negatives, but all of which provide different information about a breast mass. If physical examination reveals a mass with a low index of suspicion, carcinoma can still be present. If one adds a negative cytologic report and mammogram, the possibility of carcinoma becomes remote, even though false negatives are a recognized problem. The combination of questionable physical findings with negative cytologic findings and mammography can be utilized to avoid biopsy in most juvenile diabetics.

Fibrous disease of the breast mimicking cancer has recently been recognized as a unique feature of some women with Type I diabetes mellitus. Features characteristic of these patients included an average age lower than that of the usual patient presenting with a breast mass, frequent bilaterality, extreme hardness of the tissue, and, generally speaking, negative aspiration cytology and mammographic studies. We have proposed a diagnostic approach designed to avoid biopsies in these patients.

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REVIEW ARTICLE

Aeromedical emergency trauma services and mortality reduction in rural areas

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A congressional report estimates that each year in the United States 140,000 people die from trauma and 80,000 suffer permanent disabilities. Fifteen percent to 20% of that toll could be averted if care were provided in designated trauma centers.[1] Budget restrictions, centralized coordination, and service improvements create challenging opportunities for aeromedical emergency medical systems (EMS) today. To appreciate the magnitude of this challenge, it is important to understand the history of aeromedical prehospital transportation, the specific problems encountered in rural settings, and the efficacy of EMS in terms of increased survival.

THE HISTORY OF EMERGENCY MEDICAL SYSTEMS

Napoleon's surgeon, Dominique Jean Larrey, pioneered the modern concept of treating the wounded as quickly as possible. Soldiers received battlefield care and were transported on "flying ambulances" (speedy light carriages).[2(p35)] In the United States, Jonathan Letterman, medical director of the Army of the Potomac during the Civil War, created the horse-drawn "Ambulance Corps" to transport casualties to nearby hospitals.[2(p35)] Initially called "Accident Rooms," emergency rooms emerged in the 1940s as a civilian outgrowth of the successful battalion aid stations during World War II.[2(p35)]

In 1962, the father of EMS in the United States, orthopedic surgeon Joseph D. Farrington,[3-6] started the first EMS training program, in collaboration with Dr Sam W. Banks, and under the auspices of the Chicago Committee on Trauma. Their curriculum initiated a standard for EMS instruction that was adopted by the Department of Transportation in 1969.[2(p35-36)] Federal legislation prompted creation of the Interdepartmental Committee on Highway Safety in New York State. Studies by that committee stimulated a systematic approach to EMS in New York State. In 1974, Article 30 of the New York State Public Health Law was changed to formalize the EMS concept.[7,8]

During the 1960 presidential campaign, a comment by John F. Kennedy inflamed a public issue: "Traffic deaths constitute one of the greatest, perhaps the greatest, of the nation's public health problems."[9(p555)] At that time, about 50% of the ambulances were staffed by morticians.[10(p11)] In 1966, a landmark report by the National Academy of Sciences, *Accidental Death and Disability: The Neglected Disease of Modern Society*, initiated a scientific and systematic approach to prehospital and hospital management of the injured patient.[2,9,11,12] Subsequently, the Highway Safety Act was passed and signed by President Lyndon B. Johnson.[9(p555)] That act encouraged state regulation of emergency medical services and permitted the use of highway safety funds for them.[10(p12)] In 1973, the Emergency Medical Services Act provided funds for the purchase of vehicles and communication equipment for ground EMS services[12] as well as for trauma care regionalization.[2(p45),10(p12)]

President Reagan's 1981 Omnibus Budget Reconciliation Act transferred funding responsibilities to state and local governments while consolidating several acts into one preventive health and health services block grant. As federal contributions remained relatively constant, a 1986 US General Accounting Office[10(p20)] study of six states reported a 34% decrease in total EMS expenditures between 1981 and 1983, followed by a 28% increase between 1983 and 1985. However, total EMS expenditures appropriated during the Reagan administration decreased.

Traumatic death from accidents "kills more people than all other diseases combined" among young Americans up to age 34.[1] As policy makers attempted new approaches to EMS, aeromedical EMS expanded enormously.[12]

AEROMEDICAL EMERGENCY CARE

Aeromedical emergency services also originated in military history. Supposedly, the first air evacuation of casualties occurred via a hot air balloon during the Prussian siege of Paris in 1870.[6,13] An exhaustive and comprehensive review by Lam[13] proves conclusively that the evacuation from Paris by hot air balloons never happened. In a similar vein, but in a somewhat tongue-in-cheek manner, on April 1, 1989, Cottrell and Garrand[14] suggested replacing noisy helicopter ambulances with lighter-than-air blimps or hot air balloons.

Air evacuation of battlefield casualties in the US began in World War I and was frequently used during World War II. In the Korean War, air evacuation to mobile army surgical hospital (MASH) units became the norm. During the Vietnam war, aeromedical transportation turned into a fast and sophisticated life-saving tool.[11,15,16(p33)]

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Mortality rates dropped from 4.5 per 100 casualties in World War II to 2.5 per 100 in the Korean War and to less than 1 per 100 in Vietnam.[12] Rapid evacuation of casualties in the larger helicopters used in Vietnam, combined with sophisticated medical care en route, partially accounted for the dramatic decrease in mortality.

A Military Assistance to Safety and Traffic (MAST) program[17] analyzed applications of military aeromedical transportation systems to civilian emergencies. That study concluded that response "to civilian medical emergencies is entirely feasible from both the military and civilian viewpoint."

Civilian applications of aeromedical EMS occurred in the 1960s, mainly by state police departments. Cowley et al[18] described the combined University of Maryland/state police helicopter evacuation program. Until 1972, no helicopter service was exclusively designed for medical purposes.[12] During the early 1970s, the growth of these systems was slow. St Anthony Hospital (Denver, Colo) claims to be the oldest operating civilian hospital-sponsored aeromedical helicopter service in the United States. That hospital began its "Flight for Life" program on October 12, 1972.[19] By 1976, only Houston and Denver had full-time helicopter services[20]; by 1980, 30 hospitals, and by 1983, 56 hospitals had aeromedical EMS programs.[11] Since 1972, 140 hospitals have established a helicopter transport service.[21]

TREATING TRAUMA

Considering the high cost of aeromedical EMS, effectiveness and efficacy analysis is vital. Studies were next to impossible at first, because there was no objective means of comparing data. Until traumatic injury was considered to be a physiologic insult to the body, like other illnesses, progress was limited. Teasdale and Jennett[22] first designed objective trauma scores, but these were restricted to head trauma. Both physiologic and anatomic scoring means were later developed with reasonable prognostic value: Trauma Score (TS) and the Injury Severity Score (ISS). The TS and ISS are used extensively in studies of aeromedical efficacy.[12]

In 1981, Champion et al[23] described the TS, the most commonly used system in the prehospital setting.[24] TS is a physiologic score that includes assessment of the status of the cardiovascular and respiratory systems as well as an index of central nervous system function.[2(p66)] Adding the weighted codes yields a TS ranging from 1 to 16, with the higher score indicating a better prognosis. Table I illustrates the weighting of the scoring system and indicates the severity of the trauma linked to the assessments. Gilmore et al[25] of LDS Hospital in Salt Lake City reported Glasgow Coma Scores (GCS) for 202 blunt trauma patients. Results showed a GCS of 7 for those who died, 10 for patients with morbidity, and 12 for patients who underwent successful rehabilitation.

Physiologic scores may be temporarily normal due to compensatory mechanisms—this is particularly relevant in younger patients. Therefore, physiologic scores, such as the TS, may not accurately reflect the extent of anatomical injury. Baxt et al[24] found that TS was an accurate predictor of mortality, with greater than 85% sensitivity and specificity. Among surviving patients, the TS prediction was less than 70% accurate in sensitivity and specificity. Investigators concluded that TS is a good indicator of mortality but a poor indicator of actual anatomic injury among survivors.[24,26]

The Injury Severity Score assigns weighted values to the patient's three most critical anatomic injuries.[2(pp67-68)] These weighted values, Abbreviated Injury Scale values (AIS), are squared and totaled. For example, a patient with a ruptured spleen (AIS = 5), rib fractures (AIS = 2), lung contusion (AIS = 3), and a fractured femur (AIS = 3) has an ISS of 43 ($5^2 + 3^2 + 3^2 = 25 + 9 + 9$).

A major handicap of the ISS is the limitation of up to three anatomic injuries, therefore not adequately addressing the problem of multiple injuries.[2(p68)] Gilmore et al[25] found a mean ISS of 39 resulting in death; a mean ISS of 36 resulting in morbidity; and a mean ISS of 23 resulting

TABLE I. Trauma Score

		Rate	Codes	Score
A. Respiratory rate—number of respirations in 15 sec, multiply by 4		10-24	4	
		25-35	3	
		≥36	2	
		1-9	1	A. ____
B. Respiratory effort		0	0	
Retractive-use of accessory muscles or intercostal retraction		Normal	1	
		Retractive/ none	0	B. ____
C. Systolic blood pressure		≥90	4	
Systolic cuff pressure—either arm, auscultate or palpate		70-89	3	
		50-69	2	
		0-49	1	
No carotid pulse		0	0	C. ____
D. Capillary refill				
Normal—forehead or lip mucosa color, refill in 2 sec		Normal	2	
Delayed—more than 2 sec capillary refill		Delayed	1	
None—no capillary refill		None	0	D. ____
E. Glasgow Coma Scale		Total GCS		
1. Eye opening		Points	Score	
Spontaneous	____ 4	14-15	5	
To voice	____ 3	11-13	4	
To pain	____ 2	8-10	3	
None	____ 1	5-7	2	
2. Verbal response		3-4	1	E. ____
Oriented	____ 5			
Confused	____ 4			
Inappropriate words	____ 3			
Incomprehensible sounds	____ 2			
None	____ 1			
3. Motor response				
Obeys commands	____ 6			
Localizes pain	____ 5			
Withdraw (pain)	____ 4			
Flexion (pain)	____ 3			
Extension (pain)	____ 2			
None	____ 1			
Total GCS points (1 + 2 + 3) ______		(Total A + B + C + D + E) Trauma Score ______		

Source: Champion HR, Sacco WJ, Carnazzo AJ, et al: Trauma score. *Critical Care Medicine* 9:672-676. © Williams & Wilkins, 1981.

in eventual rehabilitation. In general, scores greater than or equal to 15 are considered "major trauma," with the patient requiring care in a trauma center.[24] In 1988, Copes et al[27] reviewed the ISS system and affirmed the ISS as a viable and accurate triage mechanism.

Gormican[28] developed an Apgar-like CRAMS scale for field triage of trauma victims. CRAMS uses observational aspects of *c*irculation, *r*espiration, *a*bdomen, *m*otor, and *s*peech factors, with up to two points for each category. Scores of nine or 10 indicate minor trauma; eight or less is considered major trauma.

Recently, Burney et al[29] evaluated 1,081 hospital-based aeromedical patient transfers using the Therapeutic Intervention Scoring System (TISS). TISS calculates scores based on therapeutic events that occur within specific time periods. With TISS, only 18–28% of the flights had a 20% probability of being appropriate; 42% had a 99% probability of being appropriate, and 35% had a 50% or better probability of being appropriate.

RURAL EMERGENCY MEDICAL CARE SERVICES

In 1966, the National Academy of Sciences noted that 70% of fatal US motor vehicle accidents (MVAs) occurred in rural areas.[11,30] In the early 1980s, the National Highway Traffic Safety Administration recorded fatal MVAs in rural areas at 58%.[2(p26)]

Reports by Waller in 1964 and 1967 pointed out the geographical inequality of trauma survival in the United States. In 1983,[2(pp32–33)] the overall accidental death rate per 100,000 population was highest in Alaska (88.8), Montana (65.3), Mississippi (59.4), New Mexico (57.8), Wyoming (56.8), and Idaho (52.9). Lowest death rates occurred in Rhode Island (26), New Jersey (29.8), New York (excluding New York City, 30.4), Hawaii (30.5), Michigan (32.8), and Illinois (32.9). Despite the exclusion of New York City, urbanization in New York State is still extensive and may bias the New York State figure.[31] Certainly, most of the states with high death rates are predominantly rural.

On a county level, Baker et al[31,32] studied MVA death rates from 1979 to 1981. Results showed an inverse and multifactorial relationship between population density and MVA death rate. Baker et al[31] concluded that "differences in road characteristics, travel speeds, seat-belt use, types of vehicles, and availability of emergency care may have been major contributors to these relations." MVA death rates typically varied by a factor greater than 100.[32]

Mueller, Rivara, and Bergman[33] reported death rates in pedestrian-vehicle accidents in rural areas (1981–1983) of Washington State to be 2.3 times greater than in urban areas, even after controlling for the major discrepancy, the increased vehicle speed in rural areas. Generally, all studies indicate a poor prognosis for rural trauma victims as a result of two major handicaps: geographic factors and inadequate medical care.[34]

Obviously, rural vs urban and/or suburban areas is relevant when discussing geographic factors. Rural, according to the dictionary, refers to the country and is distinguished from cities and urbanized areas by the density of population and buildings. However, the literature on EMS does not usually distinguish or describe the geographic locality. This is probably a valid assumption and readers will realize the differences.

Geographic factors affect the distances for patient transfers and the time required for volunteer ambulance crews to assemble and to travel to the location of the accident.[35,36] Subtler variables involve notification and the quality of care.

Often, there are long delays before a passerby spots the accident and telephones the authorities. In 1976, only 27% of the nation had "911" emergency phones available; in 1978, 40%; and just under 50% in 1986.[10(pp31,38)] A 1986 US General Accounting Office (GAO)[10(p31)] study found that six of nine urban areas and only two of nine rural areas had complete "911" coverage. High installation costs, about $250,000, and high operating costs present significant impediments to "911" initiation in rural areas.[10(p32,38)] Furthermore, the GAO reported that an "enhanced 911" service, a central computer that automatically relays the telephone number and address of the caller, is especially rare in rural areas.

Inadequate medical care is quantitative as well as qualitative. In urban centers, paramedics usually provide prehospital services. Volunteer basic emergency medical technicians (EMTs) generally render that care in rural America.[34,36] In 1986, the GAO[10(p42)] study found that nine of nine urban areas and only four of eight rural areas had more than 50% of their population covered by EMS services staffed by personnel trained in advanced life support (ALS).

In rural areas, small case loads inhibit cost effectiveness and maintenance of skills by ALS personnel.[(10p42)] After an ALS program was started in Boston, there was a 9% increase in survival for people with cardiac emergencies.[10(p41)] Respiratory obstruction or inadequate ventilation and hypovolemia are the two most preventable causes of trauma mortality.[37] Basic EMTs are not trained to provide proper airway management and fluid replacement along with other ALS therapies.[36] Because of this difference in ALS training in rural areas, trauma patients must be transferred initially to local hospitals where immediate ALS can be started.

Trauma level III hospitals, less specialized trauma centers, do operate at a deficit as compared to trauma level I hospitals, which are tertiary care trauma centers. Physicians in local level III hospitals see few trauma patients and get little practice in ALS resuscitation and management techniques, resulting in inadequate care at this critical juncture. One index of trauma survival involves the severity of head trauma,[38] but few neurosurgeons are available at trauma level III hospitals. That creates a critical void in rendering adequate care to people with severe head injuries. In addition, deficient blood banks often cannot meet the need of an exsanguinating patient.[36]

"Rural populations suffer especially from inadequate trauma care," according to a spokesperson of the American College of Surgeons. Considering these quantitative and qualitative EMS deficiencies, it is no wonder that mortality from trauma is significantly greater in rural areas than in urban areas.

AEROMEDICAL SYSTEMS IN THE RURAL SETTING

Logically, the success of military aeromedical EMS should foster a natural extension into civilian populations, particularly in rural areas.[15,18,19,39] In addition to rapid transport of critically ill patients to regional trauma centers, helicopters also deliver highly skilled medical teams directly to the patient.[40–42] Since almost 30% of trauma mortality occurs in the early hours after the incident,[33,35,43] the quality of prehospital care can be as vital as the quality of the definitive care obtained in the emergency and operating rooms. The time between injury and initial stabilization, the "golden hour," is the single most important factor in patient survival.[34,39]

West et al[44] reported that "Survival rates for major trauma can be improved by an organized system of trauma care that includes the resources of a trauma center." In separate investigations, Fortner et al,[45] Jacobs et al[46] and Klauber et al[47] all showed improved outcomes when personnel trained in ALS render emergency care as compared to those trained only in basic life support. That view is seconded by the experience of Black et al[41] in operating a pediatric aeromedical system at Primary Children's Medical Center in Salt Lake City (Utah): "In planning our system. . . the critical issue is not how long it takes the child to arrive at the regional pediatric center, but rather how long it takes the center's expert care to be delivered to the child."

Certo et al,[48] from the University of Vermont, found that 22% of patients who arrived alive and later died in the emergency room could have been saved. Investigators concluded that "stabilization and resuscitation are necessary before transfer." However, in the view of Certo and his colleagues, bypass to a trauma center would not have been a significant factor for most of their patients. Notably, the use of helicopters staffed by a sophisticated EMS medical team was not addressed in this study. Had helicopters been used, stabilization and resuscitation techniques could have been applied, and the rapid transport to a trauma center by helicopter could have been efficacious. According to the GAO[10(p51)] report, "There are fewer specialized trauma care facilities that can provide appropriate care for the most severely injured patients. . . Therefore, systematically routing trauma patients to appropriate hospitals is important."

In addition, the prehospital care of MVA victims requires complex care that necessitates "expert" administration. Both Fischer's team[26] and Anderson et al[49] found that almost 50% of their physician-staffed helicopter missions to MVAs required a major procedure such as endotracheal intubation, tube or needle thoracostomy, central venous pressure line, cardiopulmonary resuscitation, pericardiocentesis, or cricothyrotomy. Based on these concepts, West Germany created a sophisticated aeromedical system for treatment and transportation of trauma patients along the Autobahn highway.[35] The helicopter EMS integrates the patient's care at the rural accident scene into an extended and sophisticated trauma care system. Since the EMS helicopter team delivers a highly skilled level of care on the scene, the critically traumatized patient can bypass the local hospital. Patients go directly to the regional trauma center for definitive care.

In summary, the aeromedical team delivers rapid and highly skilled medical care to inaccessible accident victims in a rural population spread out over a large geographic area.[12,40,42,49–51]

EFFECT OF AEROMEDICAL EMERGENCY SYSTEMS ON RURAL TRAUMA MORTALITY

Studies consistently demonstrate a significant decrease in mortality for patients transported to a trauma center by helicopter. However, that decline occurs mainly for seriously injured patients who require rapid and sophisticated medical care. There is no decrease in mortality for the most severely injured patients who usually die en route or soon after arrival at the hospital. Effectiveness of the helicopter team is also related to whether the team was used in an urban or rural area. Helicopter EMS teams have been proven to reduce mortality *only* in rural areas. There are conflicting data for urban regions.

In an early mortality study, Mackenzie et al[52] studied 760 patients transported by helicopter directly from the site of a MVA. There were no controls, such as trauma patients transported by ground. Mackenzie et al concluded that "Since 99 percent. . . were endotracheally intubated and mechanically ventilated, optimum oxygenation was maintained throughout the period of resuscitation and stabilization. . . The overall mortality of 22.7 percent therefore, must be assumed to be nearing the ideal for management of automobile trauma patients." Although there was no increase in survival, the study showed that airway management was paramount in the prehospital treatment of trauma patients. All helicopter teams were well trained in airway management techniques to increase survival.

In 1983, Baxt and Moody,[12] from the University of California at San Diego, reported comparative mortality statistics from a controlled study. Their research revealed an overall 52% decrease from predicted mortality with a physician/nurse helicopter team as compared with a paramedic land team caring for 150 patients suffering blunt trauma. Survival probability trauma scores were assessed on the basis of the first physician contact with the patient. Baxt and Moody indicated that "the major improvement in survivability occurred in the more severe classification groups."

No significant difference in survival occurred between the two groups with respect to patients with a high probability of survival. Survival progressively increased in the helicopter-assisted patients as survival prediction decreased (Table II). Obviously, survival significantly increased among severely injured patients who had a poor prognosis,[12,42] although "these groups represent less than 10 percent of all injured patients."

Increased survival was less dramatic in patients with the worst prognosis. Many probably had severe head trauma that could not be treated any better by the helicopter EMS group. Obviously, appropriate triage of accident victims is needed to maximize the efficiency of a helicopter team. Baxt and Moody[12] realized that the two compara-

TABLE II. Survival and Mortality Predictions Compared to Actual Mortality

	Helicopter	Ambulance	Helicopter	Ambulance
50+%	4	2.77	3	6
Less than 50%	5	2.35	2	4
Less than 25%	7.09	7.03	4	6

tive groups were not optimally chosen; that transportation distance, initial care, and care during transport to the hospital were all different.

A subsequent multicenter study by Baxt and Moody[50] reported on 1,273 patients transported by helicopter directly from the injury scene. Unlike the previous study, the helicopter teams used varying combinations of physician, nurse, and paramedic teams. Researchers compared survival of these patients to predicted survival in an ongoing "Major Trauma Outcome Study" (MTOS) sponsored by the American College of Surgeons' Study Committee on Trauma. Baxt and Moody[49,50] found a 21% decrease in expected mortality in their helicopter groups as compared to the MTOS norm.

MTOS information "represents the treatment experience of a large segment of the country"—111 hospitals in the US and Canada and more than 47,000 trauma patients.[27] More than 3,000 blunt trauma cases in 45 trauma level I and level II hospitals throughout the country were analyzed. MTOS predicted survival is currently used as a national norm for investigating trauma care outcome.

Two studies by Urdaneta et al[11,53] at the University of Iowa demonstrated the efficacy of the helicopter team in decreasing mortality and morbidity among severely injured patients. There were 569 traumatized patients in the first study and 916 patients in the second study. Patients were retrospectively divided into four groups relative to the value of the medical care rendered (Table III).

Clearly, patients with severe injury benefited most from the helicopter team's care—about 27% in both studies (essential and helpful). Still, the vast majority were not helped by the helicopter service because their injuries were relatively trivial, approximately 55% (not a factor). Among the most severely injured, for whom sophisticated medical intervention was not a factor, about 17% died in both studies. This investigation supports the data of Baxt and Moody.

Moylan et al[34] at Duke University compared the survival of 136 air-transported patients with 194 ground-transported patients, both directly from the accident scene. Because trauma scores greater than 12 predict high survivability, only patients with trauma scores of 12 or lower were included. Patients were classified into four trauma score groupings. Overall survival was evaluated for each of the groups transported by air or by ground as shown in Table IV.

By rearranging these numbers, Moylan et al reported that "there was a statistically significant survival advantage for air-transport patients with trauma scores between 10 and five (82.8 percent survival vs. 53.5 percent)." These data support earlier studies finding significantly increased survival for helicopter-transported patients with severe traumatic injury but not for the hopeless critical patients. Although there were more survivors in the air-transported group with a trauma score of three or less, there was no statistical difference for this group. In this study, the efficacy of air transport was greatest for patients in the middle range of trauma scores.

TABLE III. Value of Helicopter Transport in Two Studies Using Glasgow Coma Scale Ratings

GCS at Pick-up First Study	GCS at Pick-up Second Study	Value Category	Medical Value First Study	Medical Value Second Study
12.0	12.0	Essential	16.5%	14.0%
11.5	11.4	Helpful	10.9	12.9
13.7	13.6	Not a factor	54.7	56.6
5.9	5.7	Died	17.9	16.5

TABLE IV. Comparison of Survival by Air or Ground Transport by Trauma Score

Trauma Score	Air Transport No.	Air Transport %	Ground Transport No.	Ground Transport %
10–12	78	97.4	92	92.3
7–9	16	81.3	46	47.0
4–6	19	63.0	36	27.0
0–3	23	15.0	20	5.0
Total	136	89.0	194	61.0

At St Joseph's Hospital in Phoenix, Schiller et al[38] found that a helicopter team in an urban center may be less efficacious. Data showed an overall mortality of 18% in the 347 air-transported patients, as compared to 13% in the 259 ground-transported patients. Researchers concluded that "there is no survival advantage in the helicopter transported group in an urban area with a sophisticated prehospital care system." On the other hand, Fischer et al,[26] from the University of Texas Medical School at Houston, studied urban helicopter flights within Houston. Fischer's helicopter team included a flight physician, which enabled the prehospital care to be viewed as an extension of the hospital's emergency room. Fischer et al concluded that helicopters are indicated in urban areas where "care beyond the ALS level is required." Certainly, triage accuracy is a factor in these situations. Conflicting data suggest that additional investigation of the urban use of helicopters is warranted as the issue is still debatable.

In summary, there appears to be agreement that mortality from injury is significantly lowered by helicopter transport in the following types of patients: patients who are injured in rural areas; and patients who are seriously injured, but not the most severely injured patients.

LOGISTICAL ISSUES CONFRONTING AEROMEDICAL SYSTEMS

Various investigations have indicated that there are a number of logistical issues facing aeromedical EMS. The problems deal with triage, safety, cost, and the staffing of EMS teams.

Triage. To prevent misuse and inefficiency in an aeromedical EMS that is both costly to set up and to maintain, it is vital to establish an accurate and effective triage procedure.[16(p33)] Critics complain that some hospitals misuse their helicopter for public relations purposes, by dramatic "pick-ups" that are not medically indicated.[56] Because helicopters are "second responders" to a call from the police, fire, or other medical personnel, that rare event does occur. Certainly, effective flight protocol and medical supervision is necessary.

Generally, flights to the trauma scene cannot be justified unless a higher level of medical expertise or fast transportation will improve patient outcome.[55(G-2)] In rural areas, the prevalence of basic EMTs and long distances make this a common justification.[51] Mattox[2(p36)] identifies four situations in which helicopters are effective: when ground transport time is greater than 35 minutes; when no ambulances are available; when a physician or other medical personnel are needed at the accident scene; and when rural areas are inaccessible by land.

Entrapment of MVA victims, frequently with serious injury, is common in rural areas.[3] During extrication, the helicopter team can fly to the accident scene and use that long delay effectively to render care.[51] However, severity of injury must be a compelling factor in calling for helicopter transport.

Trauma scores of less than 12 usually indicate that a patient will benefit most from helicopter transport directly to a trauma center.[51,56,57] While realizing that mortality is decreased by aeromedical systems, in 1984 Urdaneta et al[11] felt that one cannot differentiate patients who will benefit from helicopter transport. Fischer et al[26] reported that about one third of 577 trauma patients had a trauma score greater than 16 and probably did not need transport by helicopter. In 1985 Schwab et al[58] found high injury severity scores (average ISS of 36) associated with most flights, implying accurate triage. Since many studies demonstrate a significant relationship between head injury and trauma death,[38,52,57,58] it is not surprising that level of consciousness is considered a particularly effective triage parameter.[52,57] Rhodes et al[57] found "unresponsiveness to verbal stimulation" to be the most sensitive and specific triage tool for helicopter transport. Time and/or distance was also effective, but Rhodes et al reported vital signs and entrapment to be less reliable.

Generally, transportation distance and time, level of consciousness, and severity of injury calculated by the TS or ISS are most effective in deciding which patients will benefit from helicopter transport from the accident scene to the trauma center.

Safety. People question whether aeromedical EMS may in fact increase risk of injury to the patient.[51,54] Burney and Fischer[51] reported that 47 flight crew personnel died in 25 aeromedical accidents between 1980 and 1985. Recently, newspapers publicized a helicopter crash in Texas in which two nurses and a pilot were killed.[59] Rescue flights are dangerous because they may require flying in unfamiliar and dangerous terrain, often at night, with trees and electrical wires that may easily be overlooked.[55(G-2)]

Reporting from Birmingham, Ala, Carraway[60] noted about "25 EMS helicopters damaged with 26 deaths and 16 people injured." He also observed that the numbers are increasing: 10.8 accidents per 100,000 patients transported in 1984; 14.9 in 1985; and 18.4 in 1987. Obviously, the safety of the flight crew is paramount, and rescue missions should be approved only by the helicopter pilot. Institutions such as the University of Pittsburgh use a "blind" protocol for decisions. Pilots are not informed of the severity of the patient's condition and flight decisions are unaffected. In any event, state or federal government regulations should apply to ensure appropriate flight safety.

Cost. In this era of cost containment, prospective payment systems (PPS), and diagnosis related groups (DRGs), it is imperative to assess the cost-effectiveness of aeromedical EMS. Estimates for program start-up costs range from $900,000 to $2.2 million, while annual mainenance costs run from $500,000 to $2 million.[51,54,61] A 1960s Georgia Institute of Technology[2(p37)] study and subsequent recent studies found that 24-hour-a-day, seven-days-a-week ground ambulance coverage in an urban area with a four- to six-minute response time costs $250,000 per year per ambulance unit. A priori, the common occurrence wherein more than one private ambulance arrives at the accident scene is obviously economically unacceptable for helicopters.

Burney and Fischer[51] reported that reimbursement for aeromedical EMS services typically ranges from 30% to 50% of total operating expenses. Moylan et al[34] found that payments met only 15% of the actual helicopter operational costs with the deficit generated from in-hospital patient revenue. Schwab et al[58] noted that of a total of 45 trauma patients, air-transported patients had a length of stay of 28 days vs 20 days for the ground-transported patients. Investigators concluded that "although there is a high 'capture' rate (69 percent) of new patients, the overall financial gain to the hospital is not great." Evidently, treating air transported patients is expensive and "small hospitals may be overwhelmed by these patients."[58] Furthermore, today's PPS and DRG reimbursement mode, by its cost containment nature, may discourage some hospitals from taking patients with major trauma. While Thomas et al[62] found only 11 of 283 air-evacuated trauma patients covered by Medicare, those few patients incurred a $249,601 deficit for the hospital. DRG trauma reimbursement covers only 16% of the cost. If other third party insurers adopted the DRG payment schedule, the results could be economically devastating for trauma centers.

Who can support costly helicopter transport systems? Adding administrative aspects, Barbash et al[63] stressed management control for efficient and economical operation of this high cost resource. Perhaps only larger institutions will be able to afford to divert inpatient revenue to air transport systems as part of their commitment to provide major trauma services.

STAFFING EMERGENCY HELICOPTERS

Aeromedical EMS in the rural setting has a unique quality of delivering a high level of emergency care over a wide geographic area. Most programs have teams composed of flight nurses and/or paramedics. Much of the data about emergency helicopter systems that support decreased mortality studied these teams. Problems arise with aeromedical teams that infrequently perform emergency transports, such as state police helicopter teams. Currently, there is no data showing decreased mortality with the police teams.[42]

Who should be included in the emergency helicopter team? Should a physician be an EMS team member? Critics claim that physicians do not work well in an uncontrolled environment and lack the "street wise" expertise to perform effectively as part of the emergency helicopter team.[54] Others put it more succinctly: "Physicians venturing forth into the prehospital setting often were viewed as eccentrics looking for excitement."[64] Pepe, of Baylor College of Medicine, and Stewart, of the University of Pittsburgh,[64] concluded that the use of "in-field" EMS physicians fosters an environment for research in addition to the "obvious benefits of quality assurance and supervision." EMS is seen as "the practice of medicine through physician surrogates." Pepe and Stewart agree with Henry and Krisky's[7] view that EMS is an integral part in the continuum of proper emergency care.

Studies support the use of physicians not only as supervisors, but as part of the aeromedical team. Baxt and Moody[65] randomized 574 blunt trauma patients into groups transported by a nurse/paramedic helicopter team or by a nurse/physician team. There was a 35% reduction in predicted mortality for the patients treated by the nurse/physician team; "significantly lower mortality than that of the flight nurse/paramedic staffed helicopter." Furthermore, Baxt and Moody[65] found that physician psychomotor skill and judgment were the two factors most instrumental in lowering mortality; aggressive procedures were performed more frequently by physicians. In a six-month study of 176 helicopter transports, Champion et al[66] found that physician input had significant implications for the control of overtriage of patients to trauma centers.

Fischer et al[26] agreed that many necessary invasive procedures were performed by their unit's physician-led helicopter team. Reporting on an urban helicopter team, Fischer found that a "physician in attendance was deemed medically desirable for one-half of the flights." Rhee et al[67] reported that "a flight physician made a unique and important contribution to the care of the patient" in 22% of helicopter transport cases. Interestingly, this assessment was made by the flight nurse. A retrospective analysis of dispatch sheet data by Carraway et al[68] showed that a "physician was needed" in 21% of the flights. Review of flight medical charts revealed this number to actually be 51%. McCloskey et al[69] concluded that 91% of pediatric air transports required a physician. In studying the aeromedical transport of 884 military and civilian casualties in Israel, Dreyfuss et al[70] concluded that "the 24 resuscitations and 17 emergency operations. . . could never have been carried out without the participation of a flight surgeon." Noy-Man et al[71] add additional Israeli medical air-evacuation experiences during the 1982 Lebanon war.

Stewart, Paris, and Heller[72] discussed the University of Pittsburgh's emergency medicine residency program, ranked among the strongest in terms of prehospital care teaching. That formal EMS curriculum combines extensive didactic teaching and "in-field" experience. In addition, second and third year residents are on call as part of the aeromedical team a few times every month. Recognizing that EMS provides care that is only the first stage of complete emergency care, many states recently required supervision of major urban EMS systems by physicians.[64]

Only one out of every 50 patients would be affected by having physicians aboard EMS helicopters. Therefore, Baxt and Moody[65] concluded that the value of staffing all EMS helicopter missions with a physician must be questioned. Regarding pediatric critical care transport, McCloskey et al[69] also raised questions. Thomas et al[73] compared 26 nurse/physician and 21 nurse-only hospital aeromedical evacuation programs. They found that aeromedical nurses performed advanced life support procedures similar to those performed by flight physicians. Dedrick et al[74] noted that 60 of 69 helicopter transport team members performed defibrillation safely without hesitation while observing standard safety precautions.

In summary, the traditional role of the physician in prehospital care has changed. Prehospital EMS is now thought of as an integral part of a continuum in emergency care. Many physicians are actively involved as part of aeromedical emergency teams. Emergency medicine residencies include aeromedical EMS staffed by residents as part of their curriculum. Studies conclusively demonstrate decreased mortality among injured patients when transported by physician-staffed helicopter teams.

CONCLUSIONS

Death secondary to traumatic injury remains epidemic in the United States. Aeromedical EMS proliferated from 1970 to 1980. That proliferation occurred after the initial expansion of EMS nationally and was bolstered by the trauma epidemic's becoming a public issue.

EMS in general, and aeromedical rescue in particular, originated with and were developed by the military. Trauma scores most commonly used in analysis of these systems have been the Trauma Score and the Injury Severity Score. Mortality secondary to trauma is still significantly higher in rural areas of the United States as compared to urban areas. Long transportation distances and inadequately trained emergency medical personnel are the most important reasons for the increased mortality in rural areas. Emergency helicopter services, when staffed by highly trained personnel, enable the delivery of a high level of care over a wide geographic area. Inclusion of a physician as a part of the helicopter team seems to result in a decrease in mortality among trauma victims.

Helicopters specifically address the problems inherent to rural trauma. Several studies support the use of EMS helicopters in rural areas, showing decreased mortality among trauma victims transported by air. The effective use of aeromedical transport requires appropriate triage to prevent costly overutilization. Government regulation and inspection is needed to ensure adequate mechanical safety of the helicopters and the training of pilots.

Initiation and maintenance of helicopter EMS are expensive and rarely generate sufficient funds to pay for operational costs. In reality, only a major trauma center can afford a helicopter EMS.

In a comprehensive review of emergency medical services in the *New York State Journal of Medicine*, Mustalish[75] examined 20 years of growth and development, from 1966 to 1986. He identified and explained the significant economic, political, and social factors, events, and accomplishments that resulted in modern prehospital EMS systems. However, Mustalish does warn that "an absence of national funding and of a national focus for leadership may result in a slowing, if not a reversal, of the progress since 1966."

In colorful language, in the same issue of the *Journal*, Stewart[76] commented on the Mustalish article and the challenge of the 1980s for EMS physicians and systems. Stewart noted that emergency medical physicians and systems must prove themselves through research and evaluation of outcome. "Otherwise, they will find themselves still shuffling along the dusty road, oil and wine in their packs, kindly seeking to help the victims of thieves, their donkeys trotting along behind them."

Although New York State is often thought of as an urban metropolis because of the visibility attached to New York City, and also cities such as Albany, Buffalo, Rochester, and Syracuse, much of the state is rural. It would certainly be appropriate for health care providers to investigate further utilization of helicopter emergency medical care teams to reduce mortality in trauma situations.

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Maintaining serenity in chronic illness

JACQUELINE GALLOWAY

The diagnosis of a chronic illness changes a patient's relationship with the doctor. It brings to the patient both a sense of intensification of the relationship and a new attitude of dependence. One's spirit of wholeness is compromised when a recurring sickness is diagnosed and many patients feel that they must tap an inner energy to deal with their suffering and to affect the process of healing.

When I was diagnosed with systemic lupus erythematosus (SLE) and fibromyalgia, I keenly felt the need of more than my physician's treating my presenting symptoms. I knew I had first rate treatment, for my rheumatologist was recognized as having a solid background in treating lupus and was involved in several research studies at a noted university. I also knew I did not need empathy to fill the sense of need. What, then, was I seeking?

In the course of several office visits with my physician, I became aware of the "baggage" I brought along. I was not only weighed down with complaints related to the symptoms of my disease as it progressed, but I was also challenged by many other issues. I could identify the shock of learning I had SLE, and it was apparent that it immediately affected my career goals. My social life style was changing as I found making ordinary future plans impractical, since the profound fatigue in SLE is unpredictable, as is the joint swelling and pain. There was a certain amount of anger, some fear, and many unanswered questions about my future.

I felt that surely some of the contributing factors that brought SLE to manifest itself were yet undiscovered. With introspection and palliative support from my physician, where a total cure was impossible at this point in time, I felt certain coping mechanisms could be discovered and developed. I also felt that in the broader context, there was evidence to support my conviction that my body indeed needed more than medicine. I did not feel that seeking some other discipline such as psychotherapy was necessary—nor did my physician make such a suggestion. I was also fully aware that there is no magical formula today to totally arrest SLE. I began to explore the fact that a team approach to my new schedule of doctor visits could tap an inner energy to meet the needs not just of my body, but of my mind and soul.

What I discovered was that as my doctor stood in solidarity with me, sharing in the search for my relief/comfort, I felt more like a co-partner and less like a dependent patient. When my doctor encouraged me to talk about my life, my accomplishments, my goals (however impractical), rather than just of this ache or that pain, the element of dehumanization I once felt was erased. Most of us have been programmed to act a certain way in a doctor's office. We were never encouraged by physicians to share our hopes, victories, failures, or sensitivities. Each of these incorporate our spirit. Our spirit, as well as our feelings, are intrinsic parts of us, for the body and mind are different expressions of the same information. And so in the course of treatment, ideally the physician should become interested in understanding how our disease has affected our lives.

When a physician facilitates such a dialogue and encourages us to bring not only our physical self into the office, but our entire spirit, we can eliminate that feeling so many of us traditionally experience—being just another SLE patient with a grocery list of symptoms, meeting a doctor who has his grocery list of prescriptions. Our goal is not unrealistic, we seek only to attain a sense of peace and find an inner strength to deal with our illness with integrity and dignity.

We need to find our doctors a part of our team not only because of their expertise in medicine, but also because of their concern and openness to us as total persons and not just patients with a chronic disease. I did not want my disease to define me. I felt that would paralyze me and cause negativity which in turn would lead to depression. I was seeking a way to mobilize rather than paralyze me to unlock the way to patience, hope, and strength. This is not the same as a radical shift to pathetic resignation, but a passionate acceleration to explore. The first step in this quest has to come from within. If we patients regard our illnesses as a stepping stone rather than as a stumbling block, and seek a constructive way to accept the course of a disease, we can take our extraordinary situation, face it squarely, and rise up to meet the challenge and work with our options. This is exactly the point at which our physicians can help us to make appropriate and realistic choices. Adversity, used properly, can bring great growth to the patient and to the doctor's inventory of productive treatment.

A few recognizable and vital components of our psyche are love, happiness, fear, anger. Our emotions, whether logical or irrational, are inseparable from our physical body until brain death or actual death. It therefore appears to be consistent with total treatment for an ailment to include the *spirit*. To take this one step further, modern science identifies the left side and right side functions of

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our brain. Who I am and what I am about includes far more than just my body, my brain, this organ or that organ. When I cry, it is not the tear ducts which provide the impulse. When I laugh, it is not merely an exercise of my facial muscles. As has been succinctly said, what is important is "not what sort of disease does this person have, but what sort of person has this disease."

A well body is indisputably valuable. The entire make-up of a well being is not merely having a body free of disease but it includes an integrated spirit.

Physicians and patients can be pioneers energizing each other. There are moments of celebration in the ebb and flow of remissions and these remissions may not be solely because of the protocol of treatment. Probing beyond the medical charts and laboratory results in evaluating the total patient should include the hopes, expectations, wishes, and fears of the treatments prescribed.

There is often a direct correlation between the attitude and ambience of the treating physician and the eventual course of a disease process. Discussing spirituality in conjunction with medicine may at first appear to be incompatible, but a spiritual emphasis is the major component in the course of therapy on the road to recovery in a disease affecting millions today, alcoholism. The essential tool in controlling alcoholism is the scientifically accepted (by even nonreligious doctors) formula that includes and recognizes the necessity of a "higher power."

In 1990, an era of enlightened, high-tech scientific discoveries, it is not extraordinary to acknowledge that there also exists an inordinate search for the key to inner peace and inner strength. A visit to any bookstore reveals the many ways offered to lead one inward. The modern press conjures up images of weird psychics and phenomenal yogis. It is not the fanatical movement that captivates many chronically ill people and pulls them out of touch with reality. It is the serene look inward where true peace is found. It is a resource especially vital when an illness strikes.

This journey inward to discover serenity is not what some drug enthusiasts call "tripping out" but it is more like "jumping in." What we are dealing with is the *real self*, in the *real world*, with the *real truth*. Simply stated, we are seeking a personal experience in solving the problem of how to cope with the reality of our disease. It is not so much a matter of trying to delete our problem, but rather how to deal with it honestly and nobly.

The key word in dealing with our real self is self-knowledge. When we know, understand, and accept ourselves in truth, we can move to the inner self where we play no games.

In the course of my treatments for lupus, I began to notice a decided change in the way I dealt with my painful joints, profound fatigue, abdominal distress, and the side effects of some of my medications. I needed to have all the information known available to me. As I combed the *Physicians' Desk Reference*, the medications prescribed seemed more tolerable, for the side effects were predictable and not uncommon. Joining the Lupus Foundation led me to rap sessions, which gave me a support system beyond my home and doctor. The Foundation hosts general information meetings where the latest discoveries are presented by the leading research doctors. The biomedical library at the local state university, as well as the medical book store, were excellent sources for pertinent data on SLE. With these resources I was able to share my knowledge with my doctor and I was not regarded as obsessive, but was respected.

It is generally accepted that part of the mind's effect on health is direct and conscious. The extent to which we love ourselves determines whether or not we smoke, wear seat belts, eat the right foods, etc. On the other hand, we also have unconscious factors that cause our mind to send the body wrong messages.

Admittedly, not everyone who has a stressful life or suffers a tragic loss develops SLE or other chronic illness. But when we do not deal with our emotional needs (especially our stresses), we may be setting ourselves up for an illness.

One question we could ask ourselves as we strive inward to find serenity and upward to meet the challenge is why do we need this illness? Sickness sometimes gives us permission to do something we might otherwise be inhibited from doing. Sickness is our body's way of telling us that our needs are not being met.

The first step in our journey towards our goal should take place in the doctor's office, searching in each other for a high level of trust and openness. Our doctor not only needs to listen to us, he also needs to hear us. We cannot expect our physician to be introspective if we have not been encouraged or inclined to clarify our inner self.

A typical patient with SLE no longer has control of his or her life, since each aspect of the disease is so unpredictable. We (as patients) can discover the ways to become exceptional and to be receptive, to have a high degree of self-esteem and healthy self-love. It is up to us to then allow our intelligence and our inner strength to stimulate and guide us. As we discover new direction, we can reset our parameters and be exceptional in every way. Our hope is to bring our doctor along on the journey with us.

Something we should ponder is, did the discovery of million dollar diagnostic machines crowd out the capacity of our doctors to be human and gentle? A cure without care is like a gift without love.

Hypodermic needle embolization to the heart

MARC F. STERN, MD, BARBARA G. STEINBACH, MD

There have been numerous reports of foreign bodies contained within the cavities of the heart.[1] Some foreign bodies arrive directly through the ventricular wall, while others originate from more distant sites in the vascular tree and lodge in the heart. In wartime, most such objects were missiles, but in peacetime, most have been catheter fragments; in addition, household objects have been described in the heart. Because items in this latter group are inadvertently introduced into the body by the patients themselves, the route of embolization remains speculative. We describe a case of an intravenous drug abuser whose broken hypodermic needle travelled to and remained in the right ventricle without major sequelae. To our knowledge this represents the first such reported case.

CASE REPORT

In September 1985, a 24-year-old man presented to the emergency room stating that a piece of a hypodermic needle had broken off into the antecubital fossa of his left arm while he was self-injecting cocaine intravenously about 48 hours earlier. There was nothing unusual about his injection technique this particular time. The patient thought that the needle, which was originally new when he obtained it, had been used four or five times. When presented with a variety of syringes and needles, he believed that his was most similar to a 25-gauge insulin syringe. The patient was asymptomatic at the time of presentation.

He had a three-year history of intravenous cocaine abuse and occasionally abused benzodiazepam and alcohol as well. His medical history was otherwise negative.

On initial examination, he gave a healthy appearance. His blood pressure was 120/50 mm Hg; heart rate, 76 beats/min; and oral temperature, 36.3°C. Examination of his heart, lungs, and skin was unremarkable. The antecubital fossa on the left was slightly tender. Initially, radiographs were obtained of the soft tissue of the left antecubital region. When these proved negative, further soft tissue radiographs were immediately obtained of the upper arm and shoulder region, which were also negative. A chest film was obtained (Figs 1, 2). The 1-cm linear, radiopaque shadow overlying the right ventricle was clearly shown under fluoroscopic examination to be a foreign body moving synchronously with the heart and presumably in the chamber of the right ventricle.

The patient was admitted to the hospital. His complete blood count (CBC), differential, and electrolyte levels were all within normal limits. His creatinine was 130 μmol/L (1.5 mg/dL), and his erythrocyte sedimentation rate (ESR) was 2 mm/h. Hepatitis B core antibody and surface antigen were negative. Two blood cultures were sterile. No electrocardiogram was obtained. It was decided to leave the foreign body in situ due to the lack of symptoms and the difficulty of retrieval. The patient received prophylactic treatment with a seven-day course of nafcillin, 1.5 g intravenously every six hours.

The patient was seen for follow-up in May 1986 and was asymptomatic at that time. A CBC, ESR, and one set of blood cultures were unremarkable. In September 1986, and again in August 1988, the patient presented to the emergency room complaining of atypical chest pain. On both occasions, heart, lung, and skin examinations were unremarkable. An electrocardiogram on the latter visit was remarkable only for a shortened PR interval (108 msec). A chest film revealed that the needle fragment had not moved in the 36 months since its lodgement. On both occasions it was the impression of the treating physician that the patient's chest pain was of noncardiac origin, and antacids were prescribed. After each visit, the patient was lost to follow-up.

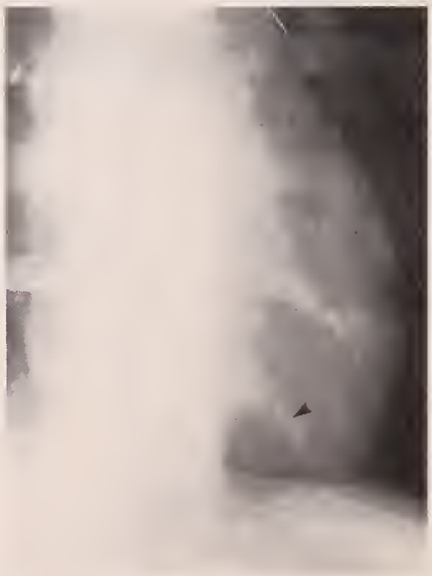

FIGURE 1. Close-up of posterior chest radiograph demonstrating a thin metallic density projected over the posterior left 11th rib.

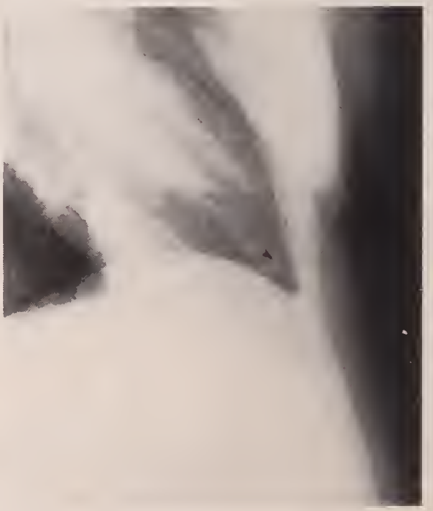

FIGURE 2. Close-up of lateral chest radiograph shows the needle to be anterior in the region of the right ventricle.

DISCUSSION

A variety of foreign bodies have been described in the heart. We limit the present discussion to the following objects: those unexpectedly found in the heart (not items such as prosthetic valves); those that arrived in the heart via embolization; those that are at least partially, if not wholly, intracavitary; those that are potentially migratory (no knife stabbings); and those that did not result in the immediate demise of the patient.

The nature of intracardiac foreign bodies varies with the era and current

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events. The earliest cases were documented or collected in the literature of the late 19th and first half of the 20th centuries.[3-5]

The offending objects cited include sewing needles, safety pins, nails, hairpins, splinters, thorns, toothpicks, bone fragments, teeth, and dental plates. Reports of intracardiac bullets and shrapnel naturally abounded as a result of World War I,[5,6] World War II,[7-9] the Korean War,[10,11] and the war in Lebanon.[12] The frequency of iatrogenic foreign bodies has paralleled the aggressiveness of medical technology. A radium needle, which migrated from the neck to the right atrium and then, in a retrograde fashion, to an iliac vein, was the first such reported case in 1929.[1] To our knowledge, only two cases of iatrogenic hypodermic needle embolism have been described.[1,13] In both cases the needle was introduced in the antecubital vein and travelled to the right ventricle. The fragments then migrated to the lung or to the pericardial fat pad, respectively.

Polyethylene catheter embolization to the heart was first described in 1954,[14] and since then scores of iatrogenic emboli to the heart have been reported,[15] including such objects as central venous pressure catheters,[16,17] guidewires,[18,19] ventriculovenous shunts,[20,21] Mobin-Uddin caval umbrellas,[22] and Greenfield filters.[23] Finally, drug abuse behavior has brought with it needle embolization. In the only two such cases described,[24,25] the needle fragments apparently embolized from an antecubital and femoral vein, respectively, to the right ventricle. Unlike the case presented here, in which the needle remained in the heart, the first patient's fragment presumably re-embolized to the lung within five months, while the second patient's needle migrated transmyocardially.

Foreign bodies reach the cardiac chambers either by direct penetration or by embolization from the vascular space. The former group is composed mostly of missiles, ie, bullets,[1,26] shrapnel,[27] and pins or needles.[1,28] The latter group is broader and includes these same missiles, the aforementioned iatrogenic materials, and a variety of other objects, such as toothpicks,[2,29] hair clips,[1] and bristle,[30] which are usually shown or presumed to have entered the body through the gastrointestinal tract and then burrowed transmurally to a vein. Though most migratory foreign bodies arrive as antegrade emboli from the venous circulation of the extremities and trunk, other possibilities have been reported; a bullet from the cranial circulation to the right ventricle[31]; paradoxical emboli from the venous circulation to the arterial circulation[1]; retrograde embolus from the aorta to the left ventricle[32]; embolus from the pulmonary venous circulation to the left heart[13,26]; or retrograde embolus from the pulmonary artery to the right ventricle.[26]

The fate of most intracardiac foreign bodies is removal. Gerard, in 1834, was reported to have performed the first surgery to remove a cardiac foreign body.[4] Since then, open or closed heart surgery has been supplanted by cardiopulmonary bypass surgery[33] and a variety of percutaneous and perivenous techniques,[34] including use of loop snares,[16,35] bronchoscopy forceps,[18] the Dormie ureteric stone catcher,[19] hook-shaped catheters,[36] myocardial biopsy forceps,[37] the Berens pacing electrode,[38] and variations and combinations of the above. Visualization and localization of foreign body fragments by the routine chest radiograph have been enhanced by fluoroscopy, as in the current case, angiography,[10] one-dimensional[28] and two-dimensional[39,40] echocardiography, digital subtraction angiography,[12] and computerized axial tomography.[41] Operative ultrasonography, which has so far only been used successfully for an intramural cardiac foreign body,[42] may prove useful for intracavitary objects.

The fate of intracardiac foreign bodies is a function of numerous factors including size, shape, chemical inertia, and porosity of the object, position of the patient, speed of blood flow, location within the heart, and chance. While foreign bodies left in the heart or embolized location for years have been well tolerated,[24,5] infection,[43,44] re-embolization of the fragment[45] or of an attached thrombus[28] with vascular occlusion or abscess, cardiac neurosis,[13] myocardial perforation or rupture, and arrhythmias can occur.[46,47] Hermoni et al[41] describe coronary-ventricular fistula, with small needle fragments in the vicinity of the fistula.[41] However, it is possible that fistula formation occurred when the fragments were still attached to the larger whole needles.

Patient Management. Recommendations in the literature are not consistent for asymptomatic foreign bodies, and guidelines have changed as improved surgical and nonsurgical techniques of fragment recovery have been developed.[47] Furthermore, due to the relative rarity of intracardiac foreign bodies, it is exceedingly difficult to find previous studies describing a comparable situation, especially studies that are not very old.

In approaching a patient with a possible peripheral foreign body that cannot be found, we recommend maintaining a high level of suspicion that a foreign body really exists and may have formed an embolus. This is particularly true when the injury was self-inflicted or resulted from illicit behavior; these patients have a high threshold for presenting to a physician, so when they do they are convinced of the presence of a foreign body.

The search for any radiopaque object should begin with plain radiographs of the site of entry and continue along the venous afferent route to the heart and lungs. Failure to find the object should be followed by examination under fluoroscopy. This technology is ubiquitous and is easily accessible in most institutions. In this case, though the needle was seen on plain radiographs, it was much more easily identified and its anatomic position determined on the fluoroscope. One- or two-dimensional echocardiography has been successfully used in identifying and localizing intracardiac foreign bodies, and should be considered as the next step if radiography fails. It may also be helpful for cases in which the object is not radiopaque. Other methods of detection, including conventional and digital subtraction angiography and CT scanning, may prove helpful in certain elusive cases.

If the foreign body is found at a peripheral site, the decision about whether or not to remove it should be guided by first answering the question, "Would there be a strong indication to remove this same object if it were located more centrally?" If the answer to this question is affirmative, then the object should be removed. A discussion of methods of removal from peripheral vessels is beyond the scope of this article, but with the exception of the use of a proximal balloon-tipped catheter[48] to block forward movement and/or retrieve the material, most available methods are the same as those described above for intracavitary intracardiac objects. The method of choice will depend on the vessel involved, the available technology, and the experience of the operators. When objects lie in tissue, proximal pressure on the afferent vessels may be appropriate to prevent central embolization during manipulation.

For those foreign bodies found to be centrally located it should be determined whether or not the object should be removed. The risks of invasive intervention need to be weighed against the risks of leaving the object in situ. Needless to say, there is a dearth of good data based on controlled studies in the recent literature to help answer this question. Further, reported cases undoubtedly skew the data toward over-representation of cases with successful outcomes (following either conservative or aggressive management). The recommended approach to management depends on the type of object.

Porous, Irregularly Shaped Objects. There are two recent reports of a toothpick[29] and a fishing nail[43] left in situ. In the first case, the object was both intramyocardial and intracavitary and resulted in *Candida albicans* emboli and sepsis. No recent reports of attempts to remove such objects could be found. However, we believe that it would be wise to consider such objects as highly susceptible to infection and candidates for early removal. In general, they will have to be removed surgically.

Metallic Missiles. The recent literature about such emboli within the heart shows mixed experiences. A review of 156 cases of objects left from four months to 52 years revealed that none of these objects caused subsequent problems.[1] However, 137 cases summarized in other case reports and reviews revealed 53 serious complications when objects were not removed initially.[5,49–52] Data on surgical mortality and morbidity for removal of embolic foreign objects from the heart in the era of cardiopulmonary bypass and advanced surgical and anesthetic technique are surprisingly scant. Shannon et al[49] reviewed the literature from 1930 to 1987, finding 20 of 20 successful attempts at primary extrication. Morton et al[33] described three other cases of bullets removed via cardiopulmonary bypass. Another bullet was removed by right ventriculotomy without bypass.[53] There is only one reported case of percutaneous removal of a bullet with the Caves-Schulz bioptome.[37] In the case of metallic missiles, then, the need to remove them will depend largely on the overall condition of the patient and the available surgical expertise. While a percutaneous approach may be possible, most experience has been with direct surgical intervention.

Catheters, Guidewires, and Shunts. Based on a review of 180 cases, Bloomfield[15] concluded that, in general, guidewires are more thrombogenic than catheters. Our review of cases in which this category of object was left in place found that 41 of 90 patients had serious complications, including 19 deaths, directly attributed to the foreign object.[44,46,54] Conversely, we were able to find 289 cases in which such objects were primarily removed by a percutaneous method with only ten complications: six transient or minor dysrhythmias; one serious dysrhythmia; one pulmonary embolism; and two fragmentations of the object with subsequent further embolization.[15,34,46,55–66] These data clearly indicate that, with rare exceptions, a primary attempt should be made to remove catheters, guidewires, and shunts that have lodged in the heart using a percutaneous route. While there is little data available regarding the success of more aggressive surgical approaches, we believe that the high incidence of complications from retained catheters indicates that such an approach is warranted if more conservative attempts fail.

Hollow Needles. We found only four reported cases of embolus to the heart.[1,13,24,25] The lengths of two of the needles were 1.3 cm and 3.0 cm. Two caused no problems; one migrated to a bronchus causing hemoptysis; while the fourth caused a life threatening hemopericardium. Though no recent attempts at operative or percutaneous needle retrieval have been described, Hermoni[41] reported being unable to locate during surgery three fragments that remained from multiple intracardiac sewing needles. The review by Harrison et al[42] states that 30% of intracardiac foreign bodies could not be located at surgery. It is not known how many were needles or had formed emboli. In the absence of better data, we think that each case will have to be judged individually. We agree, however, with Sbokos et al,[67] who stated that although the size of a cardiac foreign body should not be the sole determinant for its removal, the larger the object, the more likely it is to cause significant damage.

In the patient presented in this report, the object measured 1.0 cm. It appeared most likely that the needle lay wholly or partially embedded in myocardium or entangled in trabeculae. Percutaneous methods seemed futile either because of inability to find or grasp the needle or inability to remove it without the sharp ends causing trauma. We decided not to remove it surgically for the following reasons. Because of its small size, it was unlikely to cause significant local damage. If it formed an embolus, it was unlikely to cause hemodynamic compromise. As it was constructed of a noncorrosive material (J. Fink, Becton-Dickinson, personal communication, 1986), it was less thrombogenic and less prone to harboring infection. Finally, it would be difficult to locate during surgery.

If a foreign body is left in situ, should the patient be given prophylactic antibiotics, and if so, which one(s)? No useful data could be found in the literature to address this question. Single reports of infections from a variety of organisms have been reported with no obvious pattern, other than that the likelihood of infection increases as the porosity and surface complexity of the object increases and whenever there is accompanying debris, as in the case of a bullet, with possible clothing fragments. We would support empiric antibiotic therapy based on either the portal of entry or, as in this case, a specific risk group. This patient's history of intravenous drug abuse places him at high risk for *Staphylococcus epidermidis* endocarditis,[68,69] which was the basis for choosing nafcillin.

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Leiomyosarcoma of the esophagus

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Malignant esophageal tumors that present as mass densities on chest radiographs and are not accompanied by dysphagia are rare and are often difficult to differentiate from pulmonary and other mediastinal tumors. We report a case of primary leiomyosarcoma of the esophagus.

CASE REPORT

A 50-year-old man was referred to the chest clinic of this hospital because of a dry cough, a feeling of tightness in the lower chest of two weeks' duration, and an abnormal chest radiograph. He denied fever, rigors, hemoptysis, dysphagia, and weight loss. He smoked cigarettes (1 pack/day for 32 years); there was no history of exposure to asbestos. The only abnormal physical findings were dullness to percussion and decreased breath sounds over the left posterior lower chest. A complete blood count, biochemical profile (Sequential Multiple Analyzer (SMA) 18) and urinalysis were normal. A 5 turberculin unit purified protein derivative intradermal test was negative.

Posteroanterior and lateral roentgenograms of the chest (Figs 1, 2) showed a large, well-circumscribed mass measuring 10 cm in the left lower lung field. The inferior margin of the mass could not be visual-

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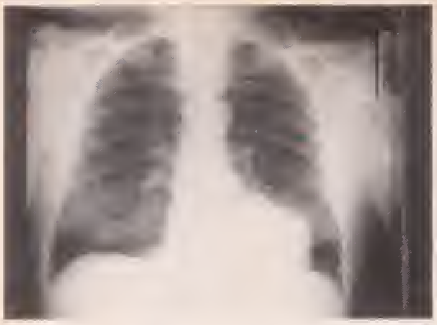

FIGURE 1. Posteroanterior roentgenogram of the chest, showing a large mass density in the left lower lung field.

ized. An esophagogram (Fig 3) demonstrated extrinsic compression of the distal esophagus. A computed tomogram (CT) of the chest (Fig 4) demonstrated a large mediastinal mass with solid and cystic areas. No calcification was seen. Bronchoscopy revealed extrinsic compression of the lower lobe bronchi. At thoracotomy, an 8 × 5 × 3-cm encapsulated, well-circumscribed, firm mass with areas of liquification was found, originating from the esophagus. The mass was partially excised. Microscopic examination showed spindle-shaped cells with hyperchromatic nuclei and mitotic figures, characteristic of leiomyosarcoma.

The patient was advised to undergo radiotherapy but refused the treatment. Six months later the patient underwent an esophagectomy for local recurrence of the tumor. He was lost to follow-up. He returned to the hospital 18 months later with evidence of widespread metastases. He refused further treatment and left the hospital; he died five days later at home.

DISCUSSION

Leiomyosarcoma of the esophagus is a rare tumor. It accounts for about 0.5% of all malignant esophageal tumors.[1] It was first described by Howard in 1902,[2] and since then about 45 cases have been reported in the English literature.[3,4]

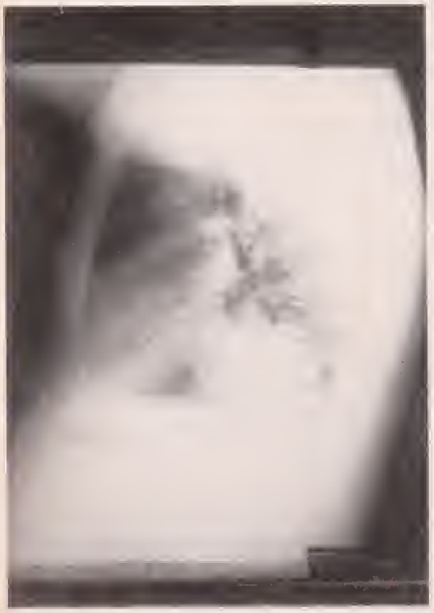

FIGURE 2. Lateral roentgenogram of the chest.

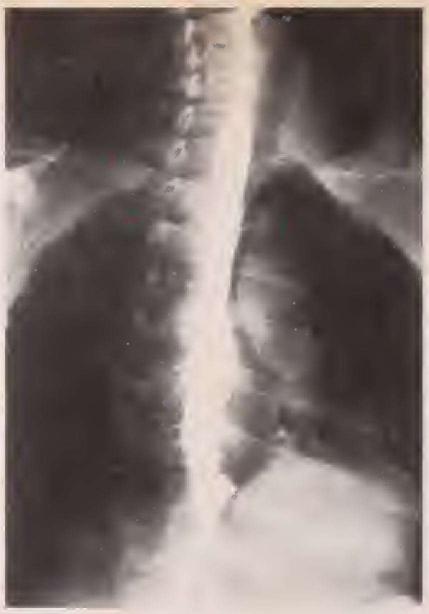

FIGURE 3. Esophagogram demonstrating extrinsic compression of the distal esophagus.

The sex distribution shows a slight male predominance, and the ages of patients range from 32 to 78 years. The tumor is more commonly found at the lower third of the esophagus, followed by the upper and middle third.[3,4] Pathologists classify this tumor into two types according to gross pathologic findings: polypoid and infiltrative, the former being more common.

Most patients present with symptoms, with dysphagia being the most common symptom, found in 85% of cases, followed by weight loss, chest pain, and melena.[3-10] Tumors found in the absence of any gastrointestinal symptoms are very unusual.[4] The chest roentgenogram often reveals a posterior mediastinal mass. Dense calcification within the mass has been also reported.[5]

Balthazar[6] classified the radiographic appearance (esophagogram) in two categories. About 60% of esophageal leiomyosarcomas present as a single, lobulated polypoid defect without demonstrable ulcerations. Although they usually exhibit a broad base of attachment, pedunculated, fully intraluminal lesions are not uncommon. The remaining 40% of reported cases present as a large infiltrating and ulcerating lesion that is indistinguishable from primary or metastatic carcinoma of the esophagus. Rarely, a barium swallow test shows a well-circumscribed, circular defect with an intact mucosa, an appearance indistinguishable from leiomyoma.[3]

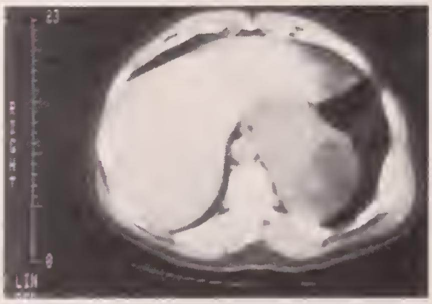

FIGURE 4. Computed tomogram of the chest, showing a large mediastinal mass with solid and cystic areas.

The CT appearance of this tumor is unusual. Balthazar,[6] in his review article, described a single case of leiomyosarcoma appearing as a well-circumscribed soft tissue mass in the posterior mediastinum without local spread or mediastinal lymphadenopathy. In the patient presented here, the CT was suggestive of a soft tissue mass in the posterior mediastinum with areas of liquification. The latter could be helpful in differentiating leiomyosarcoma from leiomyoma. Esophagoscopy is an essential component of the work-up. Biopsy is helpful when the mass is intraluminal or in cases in which there is ulceration of the mucosa.[3,7]

Surgical resection is the preferred mode of therapy. Simple excision can be as effective as radical excision, especially in a well-localized lesion.[3] There have been reported cases of prolonged survival after treatment with radiotherapy and chemotherapy.[1] Polypoid lesions in women have a better prognosis than in men.

Leiomyosarcoma is a rare tumor but should be considered in a patient with a posterior mediastinal mass.

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Gardner-Diamond syndrome

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The Gardner-Diamond syndrome, also known as autoerythrocyte sensitization, was first described in 1955.[1] Since then, individual cases have been reported predominantly in young women; it has also occasionally been seen in males.[2] The outstanding feature of this entity is purpura, which is usually confined to the arms and legs. In addition, there may be a variety of systemic manifestations, including joint pain, gastrointestinal symptoms, low grade fever, and renal and neurologic symptoms. The fact that these symptoms are so varied and indistinct has led some observers to label this disorder psychogenic purpura.[3] Following is a report of two new cases and a discussion of the possible pathophysiology of this entity.

CASE REPORTS

Case 1. A 35-year-old woman was admitted to the hospital on June 1, 1988, with multiple areas of purpura and arthralgia following a flu-like syndrome six weeks earlier. Her medical history included rheumatic fever at the age of 12, for which she had received intramuscular penicillin. Bronchial asthma had been present for many years but had not been active recently. She had undergone bilateral benign breast tissue removal and had recurrent cystitis.

Physical examination of this anxious woman complaining of pain in the wrists, forearm, and legs was performed. The head, eyes, nose, and throat were normal. The heart was not enlarged, the rhythm was regular, and the patient's blood pressure was 118/70 mm Hg. There was a soft, I-II/VI diastolic murmur heard at the apex. Examination of the abdomen revealed no masses, and no organ edges were palpable. The patient's extremities demonstrated numerous areas of purpura of various sizes, up to 25 cm (Figs 1, 2). A warm, stinging pain, which progressed into superficial nodular areas over the thighs and forearms, preceded the purpura. Swelling of the right knee was noted, with slight effusion and a superficial thrombophlebitis of the right leg.

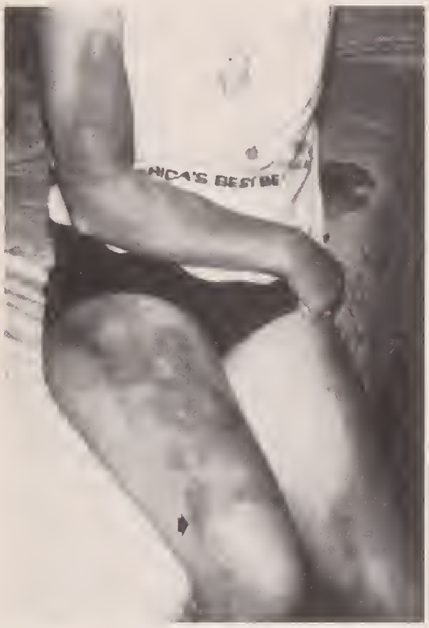

FIGURE 1. Purpura of the upper and lower extremity of Case 1.

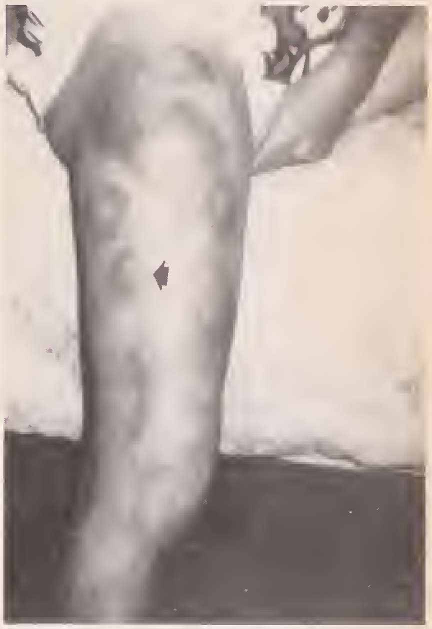

FIGURE 2. Purpura of the lower extremity of Case 1.

The laboratory data were as follows: white blood cell count, 11.4/mm^3; hemoglobin, 13.6 gram %; hematocrit, 40.3%; platelets, 637,000/mm^3; prothrombin time, 10.8 sec; and partial thromboplastin time (PTT), 22.8 sec. The blood smear and urinalysis showed no abnormalities. The blood electrolyte levels, thyroid function tests, and electrocardiogram were all within normal limits.

A coagulation profile showed an increase in the platelet count and a normal bleeding time. Platelet aggregation studies showed normal reactivity to all agonists. Plasma coagulation tests showed a normal PTT. Levels of blood fractions II, V, VII, and X were within normal limits. No significant hemostatic defect was detected.

A chest film was normal, and radiographic evaluation of the right knee showed synovial swelling.

Intradermal skin testing was performed with 0.1 cc of the patient's own red cells and a similar amount of normal saline injected as a control. Injection of the red cells produced a large area of erythema and wheal formation which required 24 hours to fade, whereas the saline gave no reaction (Fig 3).

The patient was treated with prednisone, 10 mg four times a day, while in the hospital, with slight relief of the pain and reduction of the number of lesions. At times the pain be-

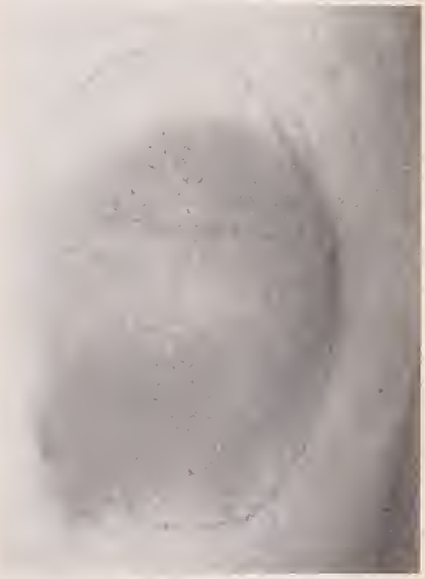

FIGURE 3. Erythema and wheal formation to 0.1 cc of autologous blood of Case 1.

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came very sharp before the eruption appeared, so she was given hydromorphone, 2 mg orally twice a day. The purpura continued to develop and increase in severity. She was given amitriptyline, 10 mg orally twice a day, and the prednisone was increased to 20 mg four times a day.[4,5] However, the regimen was not effective. The lesions recurred and lasted about six months, after which they spontaneously disappeared.

Case 2. A 30-year-old woman presented on August 29, 1988, complaining of fatigue, weakness, weight loss, and purpura of both thighs and legs. Her medical history indicated that she had been having these symptoms since she had discovered that her father had carcinoma of the lungs. She had had to give up her job in order to help care for him. She had previously lived with her male friend, but had been forced to relinquish her apartment and live near her father. She had had no previous illness or systemic disease to explain the purpura.

Physical examination revealed a rather thin woman who was markedly anxious and depressed. Examination of the eyes, ears, nose, and throat was within normal limits. Evaluation of the neck revealed no bruit or thyromegaly. The heart was not enlarged, and no murmurs were audible. The rhythm was regular, and the blood pressure was 110/74 mm Hg. The abdomen was soft, and no organ edges could be felt.

The lower extremities revealed numerous tender, slightly raised purpuric lesions on both thighs and legs (Fig 4). As in Case 1, these had been preceded by a warm, stinging pain which progressed into superficial nodular areas of various sizes that were tender to palpation.

Radiographic examination of the chest revealed a heart of normal size and clear lungs. The electrocardiogram showed normal sinus rhythm with a rate of 72 beats/min and normal axis. No T-wave abnormalities were noted.

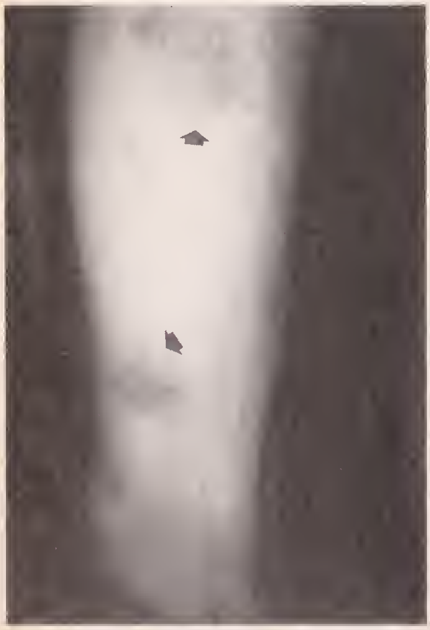

FIGURE 4. Purpura in the lower extremity of Case 2.

Laboratory tests, including a complete blood count, plasma prothrombin time, and bleeding and clotting times, were within normal limits. The capillary fragility test was also normal.

All the electrolyte levels were within the normal range. The lupus erythematosus cell test was negative. Levels of blood fractions II, V, VIII, and X were all within normal limits. The 3P test was also negative. Platelet aggregation studies demonstrated a brisk normal reactivity to all agonists. A consulting hematalogist discovered no significant hemostatic defects in this patient.

The patient was treated with oral prednisone, 10 mg four times a day, for several weeks. However, the lesions continued to recur, as did the pain. The prednisone dosage was raised to 40 mg four times a day orally, but provided no relief. She was given amitriptyline without benefit. A skin test with her red cells was not performed because of her fear of any procedures involving a needle.

The lesions recurred from time to time, but after several months, she did not return for follow-up. Her sister reported that about six months later the lesions spontaneously disappeared.

Discussion

The Gardner-Diamond syndrome, or autoerythrocyte sensitization, is an uncommon purpuric disease that occurs mostly in young women and is characterized by painful inflammatory ecchymotic lesions unrelated to blood clotting or vascular abnormalities.[1] Occasionally, it has been seen in young men. The lesion is heralded by a burning, stabbing, tingling, and throbbing sensation before the painful purpuric eruption appears. At times there is a fever associated with the syndrome, as well as systemic manifestations, including gastrointestinal bleeding, epistaxis, cutaneous bleeding, abdominal pain with or without nausea or vomiting, and renal and neurologic symptoms.

The eruptions usually occur on the extremities, although some observers have also noted lesions on other parts of the body, except for the face. Gardner and Diamond[1] first noted that the intracutaneous injection of 0.1 cc of the patient's red cells or stroma can produce erythema and wheal formation. The purpura may clear after a period of time, only to recur after an injury or emotional upset. The duration of the outbreak is unpredictable, and eventually it may spontaneously disappear.

The cause of this syndrome has been a matter of controversy. Gardner and Diamond[1] postulated that there is a fixed tissue antibody that reacts with red cell stroma to produce edema, increased capillary permeability, and further extravasation of blood into the tissues. The lesion is then self-perpetuating by subsequent contact of red cells at the periphery with sensitized tissue. This proposed allergic basis for the syndrome is not universally accepted, for the status of the antigen is not definitely known, and no circulating antibodies toward erythrocytes or red cell stroma have been demonstrated.[4]

Ratnoff and Agle[3] called attention to a psychiatric factor as the cause. It is recognized that many of these patients present with some psychogenic disturbance. Ratnoff and Agle have suggested that these patients express emotional problems in a physical form through both hysterical mechanisms and psycho-physiologic reactions. McDuffie and McGuire[6] evaluated the psychologic features by administering the Minnesota Multiphasic Personality Inventory test to six women aged 17 to 38 with characteristic purpuric skin lesions and abdominal pain. They found a remarkably uniform pattern characterized by difficulty in handling aggressive feelings, alternating between repression and hysteria.

The possibility of self-trauma, so called factitious purpura, in the etiology of this disorder has also been considered. However, in a well-studied case by Gottlieb et al,[7] a patient with the Gardner-Diamond syndrome was placed in a cast to preclude self trauma, and it was shown that the lesions continued to develop under the cast. McIntosh et al[8] have suggested that the autosensitization syndrome may have an immunologic component, although the pathogenesis would be heterogenous. Although immune deposit nephritis has not been reported in such patients, there is evidence of hematuria in some cases.

The therapeutic options for this syndrome have as yet proven unsatisfactory. Administration of corticosteroids, antihistamines, and antimalarials, as well as attempts at erythrocyte desensitization, have been unsuccessful.[6] Plasmapheresis and a placebo procedure have also been tried, with no favorable result.[6]

The two cases presented here closely resemble the description of the Gardner-Diamond syndrome, in as much as the patients are young anxious women who presented with numerous areas of purpura on their extremities and other systemic manifestations. A skin test with the patient's red cells in case 1 demonstrated a positive reaction. Treatment was unsuccessful with a variety of agents.

It is apparent that the diagnosis of Gardner-Diamond syndrome relies on a variety of criteria. For example, it occurs predominantly in anxious young women. The purpura, frequently preceded by pain and tingling, is usually distributed over the extremities, but can occur on any other part of the body except the face. Furthermore, there is no hematalogic abnormality to account for the purpura and the patients usually exhibit a positive reaction to intracutaneous testing, with their own red cells or stroma.

Acknowledgment. The author thanks David Levine, MD, for referring Case 1, and Ms Sonya Stanislow for her assistance in preparing this report.

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Volar proximal skin necrosis after radial artery cannulation

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Percutaneous cannulation of the radial artery at the wrist is a commonly used technique for monitoring blood pressure and sampling arterial blood gases in critically ill patients. The incidence of ischemic complications related to radial artery cannulation,[1-3] as well as the effectiveness of the Allen test in identifying those at risk for ischemic complications, have been reviewed by others.[4-7] This report presents a patient with ischemia to the volar forearm proximal to the site of radial artery cannulation, with a proposed mechanism for such injury.

CASE REPORT

A 74-year-old woman underwent exploratory laparotomy for a large, poorly differentiated adenocarcinoma of the left ovary. The patient underwent a bilateral salpingo-oophorectomy and a sigmoid colon resection. During surgery, an arterial line 20-g angiocatheter was placed percutaneously in the right radial artery at the level of the wrist. Arterial cannulation was accomplished in a single attempt. An infusion of 1,000 U heparin in 500 cc normal saline was maintained as a flush. On the second postoperative day, the arterial line was removed. Three days later, an area of induration was noted on the right volar forearm.

A vascular surgery consultation was recommended, and upper extremity pulse volume recordings (PVRs) were performed; they were entirely normal. The following day, the ulnar pulse was noted to be strong, but the radial pulse was noted to be absent. Digital pulse volume recordings were obtained, and these were also noted to be normal. The opinion of the vascular surgeon was that the discoloration was related to subfascial bleeding that occurred after the catheter was removed.

On the 12th postoperative day, there was demarcation of the skin of the forearm, and a plastic surgery consultation was obtained (Fig 1). At this point, the patient had mottling of approximately 50% of the volar surface of the right forearm, extending from the wrist crease proximally. There was decreased sensation over the area of mottling, and part of the area had an eschar. In addition, there was decreased sensation in the distribution of the right median nerve.

The patient was treated with topical 1% silver sulfadiazine cream. The majority of the area healed; however, a 3 cm × 2 cm portion of the eschar eventually separated, with obvious full-thickness loss (Fig 2). A skin graft was recommended, but was refused by the patient. During the next three months, the wound eventually contracted and new epithelium grew at the site (Fig 3).

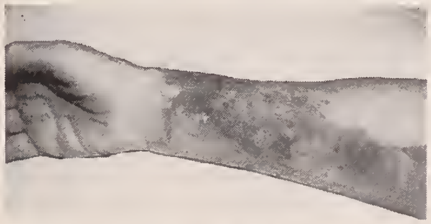

FIGURE 1. Area of ischemia to volar surface of forearm on 12th postoperative day.

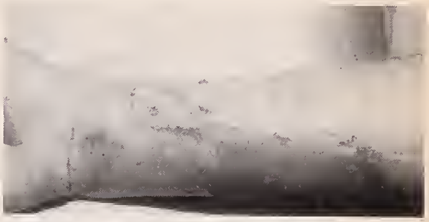

FIGURE 2. Tissue damage approximately eight weeks after surgery and after eschar separation.

Electrodiagnostic studies showed evidence of nerve compression at the level of the distal forearm in the vicinity of the tissue loss.

DISCUSSION

It is well accepted and documented that radial artery cannulation is a low-risk procedure, with substantial potential benefit. Several factors may predispose a patient undergoing radial artery cannulation to hand ischemia. These include female sex, low cardiac output, the use of vasoconstrictive drugs, and

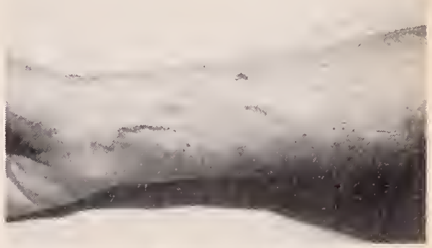

FIGURE 3. Fully healed forearm at six months.

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pre-existing peripheral vascular disease. It is recommended that the cannula be short, and it has been shown that multiple attempts at cannulation or hematoma formation yield an adverse prognosis.[4-7]

This case is similar to the seven cases presented by Wyatt, Glaves, and Cooper in 1974.[8] In these patients, the patency of the arterial line was maintained by a slow infusion (less than 4 mL/hr) of physiologic saline containing heparin (1 U/mL using a roller pump). Histologic evaluation of the postmortem samples showed subendothelial fibrin deposition. An adjacent thrombus was present, to varying extents, proximal to the position of the cannula.

The radial artery in the lower forearm is superficial and adjacent to the tendon of the flexor carpiradialis muscle. At this level, it is covered by skin and fascia. Numerous small branches arise from the distal radial artery and extend to the skin of the forearm. These small cutaneous branches off the radial artery form anastomoses with similar branches from the ulnar and interosseous arteries. Small branches off the radial and ulnar arteries carry the blood supply to the volar forearm skin.[9]

Based on the pathologic findings in this case and the current knowledge of the arterial anatomy of the volar forearm, we believe that the condition develops from proximal migration of thrombi around the catheter tip. The ischemia developed proximal to the arterial catheter, and we suspect that microthrombi adjacent to the arterial catheter tip migrated up the radial artery and into the small cutaneous branches that supply the volar forearm skin.

Of the eight patients discussed here, six had full-thickness skin loss, one required skin grafting, and one patient refused recommended skin grafting.

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ANATOMICAL DONATIONS

The Anatomical Committee of the Associated Medical Schools of New York (AMS) is currently carrying on a public education campaign to encourage anatomical donations. More than 300 cadavers are needed each year to serve the educational and research needs within the city, and in recent years, only half that need has been met by donations. The AMS Anatomical Committee represents 11 medical and other health professions schools in the New York Area. The committee was established to provide information to the public about anatomical donations and to provide assurance that cadavers used for educational and research purposes would be cared for with the utmost respect and sensitivity.

AMS functions as a clearinghouse of information when patients inquire about anatomical donations. For further information on how to pledge an anatomical donation, physicians or their patients can contact Liz Rivera at 212-643-8950, the Associated Medical Schools of New York, 70 West 36th Street, Suite 302, New York, NY 10018.

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

UNY*Care

TO THE EDITOR: If any issue should bring New York State physicians to the barricades, it should be the spector of UNY*Care. The prospect of the state as the exclusive payor for all medical services and goods exceeds the malpractice issue as a threat to the future of practice in New York.

In the October 1989 issue of the *Journal*,[1] the leadership of the Medical Society of the State of New York (MSSNY) speaks of "great appeal in terms of efficiency" in a one-payor system. It also states such a system "must be carefully constructed," implying that it is a given that it will come to pass. I fervently hope that I am never a practitioner in such a system, at the political mercy of the state and whatever wind is blowing the strongest in the legislature, or at the department of health.

The MSSNY should oppose a one-payor system at all costs, or else physicians will surely all be the certain victims of an inefficient bureaucracy.

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[1] Foy DF, Menghini MV: The confidential New York State Department of Health Universal New York Health Care (UNY*Care) proposal: Medical Society of the State of New York staff observations. *NY State J Med* 1989; 89:10:572–575.

In reply. The prospect of a New York State Department of Health (NYSDOH) initiative, UNY*Care, has been a source of concern to the Medical Society of the State of New York (MSSNY) since early 1989. For example, the MSSNY Ad Hoc Committee on Insuring the Uninsured has diligently reviewed and commented on the department's proposal as an element of its overall study of the New York State uninsured problem. The ad hoc committee, furthermore, invited key representatives of the NYSDOH to its October 5, 1989, meeting to elaborate on the UNY*Care initiative. Numerous questions posed by the committee members went unanswered, and others were either partially or generically answered.

While the issue of UNY*Care continues to be foremost in our deliberations concerning the problematic state health care delivery system, we are encouraged by the negative responses to the initiative emanating from the business, private, hospital, and health insurance sectors. We believe that the Society's UNY*Care analysis which I coauthored, and which appeared in the October 1989 issue of the *Journal*, was instrumental in motivating these groups to study the proposal closely and to make public commentary.[1] Since the analysis appeared in the *Journal*, and was disseminated to the leadership of these groups, we received numerous requests for copies of our UNY*Care assessment. Despite the October 1989 publication date, the findings and premise of the article continue to be applicable today—with one exception.

Although we had initially commented about the potential "appeal" of the UNY*Care single-payor system, with the procurement of further details we have grown even more skeptical and concerned about such a state-controlled arrangement. One major health insurer's recent analysis cites such a monolithic single-payor authority as potentially the largest such system in the country requiring yet undeveloped supporting technology at anticipated great cost. The insurer further cites lack of data to allow projections relative to the time required for the establishment of such a system; errors in estimates of cost savings in view of minimal savings through cost efficiencies, and substantial duplication of effort causing significantly increased administrative costs for existing payors and the UNY*Care single-payor authority itself. This, and other assessments, have raised doubts concerning the viability of a single-payor system which appears to be conceptually flawed.

Even more fundamental to the incomplete UNY*Care proposal (which, to date, has not revealed its minimum benefits structure) is the fact that the state's budgetary problems appear to preclude implementation of the system in the foreseeable future.

With state budgetary deficits ranging from $1 billion by the end of the fiscal year on March 31, and estimates from the business sector and the aforementioned health insurer to fund the UNY*Care system ranging from $2.2 billion to $3.7 billion, respectively, UNY*Care does not appear likely to gain the necessary funding and support to permit its implementation. Whether it will eventually be implemented on an incremental basis still remains uncertain, but such a possibility would also face similar obstacles.

In summary, reports from MSSNY representatives who have participated in recent UNY*Care meetings sponsored by the state, and have monitored the deliberations of the 20-member expert panel established by the NYSDOH to review and assess the proposal, indicate that skepticism and criticisms about UNY*Care were notably prevalent during these sessions. Its general credibility is seriously questioned—particularly in view of the state's fiscal crisis and its Medicaid track record.

However, despite UNY*Care's rather hostile reception, the MSSNY will remain vigilant and monitor further significant developments.

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TO THE EDITOR: The March 1990 issue of the *Journal* contained a letter to the editor, "AIDS legislation needed."[1] The two authors of this article, Raymond A. Smego, Jr, MD, and Elizabeth A. Barranco, MD, list their affiliation as the Albany Medical College. They have not been affiliated with the Albany Medical College since August 1, 1989. Their views in no way reflect the position of the Albany Medical College on AIDS legislation.

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[1] Smego RA, Barranco EA: AIDS legislation needed. *NY State J Med* 1990; 90:163.

In reply. Drs Smego and Barranco wrote and submitted their letter at a time when they were still affiliated with the Albany Medical College. It is for this reason that this affiliation was listed on their published letter.

PASCAL JAMES IMPERATO, MD
Editor

Claims-made policies and physician employment contracts

TO THE EDITOR: The Office of General Counsel of the Medical Society of the State of New York has received a number of inquiries from physicians concerning whether a promise by the employer to provide the employee with liability insurance obligates the employer to purchase "tail coverage" when the claims-made insurance policy is cancelled. This ordinarily occurs when the employment is terminated. The employee-physician will usually argue that the promise obligates the employer to pay for tail coverage. The employer-physician will argue that he only intended to provide claims-made coverage without tail coverage. If the parties cannot amicably settle their disagreement, a legal action based on breach of contract may result. Disagreement of this nature can be avoided by drafting an unambiguous employment contract that clearly defines the agreement of the parties. A recent appellate court ruling, summarized below, may be instructive to physicians and attorneys who draft physician employment contracts.[1]

In the spring of 1986, Doctor C made an oral agreement to employ Doctor F. This agreement was confirmed in a letter written by Doctor C dated July 29, 1986. The letter also stated, "liability insurance will be provided to you."

Doctor C purchased a claims-made insurance policy on behalf of Dr F. Because of legislation enacted in 1986, claims-made insurance was the only type of liability insurance available. Generally, an occurrence policy provides coverage for all claims arising out of incidents that occur during the policy period no matter when reported. A claims-made policy provides protection only for claims that arise out of incidents that occur and are reported while the coverage is in force. As a result of Section 9 of Chapter 266 of the Laws of 1986, beginning in July 1986, every insurer that issues or renews policies for professional medical liability insurance must issue such policies on a claims-made basis. Physicians who have occurrence policies may elect to continue coverage on an occurrence basis. The statute requires the insurer to provide tail coverage without charging additional premiums at the time of issuance under certain circumstances. Under a claims-made policy, coverage is provided only if a claim is made during the existence of the policy. If such policy is terminated, coverage can be extended under certain circumstances. If the insured prematurely retires, however, coverage for prior acts or omissions is only available through the acquisition of extended reporting period coverage ("tail coverage").

The employment continued until February 1988 when Dr C notified Dr F that the employment was being terminated. Dr F decided not to continue in private practice and canceled her insurance policy. Dr F asked Dr C to pay for tail coverage but Dr C refused. As a result of Dr C's refusal, Dr F commenced an action for breach of contract. Dr C contended that the letter of July 29 was not a legally enforceable contract. In the alternative, Dr C contended that even if the letter was an enforceable contract, he had complied with his obligation to provide liability insurance by providing claims-made insurance without tail coverage.

The Appellate Division, Third Department, held that the July 29 letter was an enforceable contract and that Dr C was required by the contract to pay for the tail coverage. The Appellate Division interpreted the phrase "liability insurance will be provided to you" as meaning that Dr C had agreed to protect the employee against any liability the employee might be subjected to for work done while in his employ. The only way this could be done was for tail coverage to be provided. The Appellate Division reminded Dr C of the often cited principle of contract: any ambiguity in a contract will be construed against the party drafting the contract.

Physicians contemplating an employment relationship should fully discuss the terms and conditions of employment, and the written agreement should clearly reflect the intention of the parties. When one party agrees to provide liability insurance to the other, both parties should discuss whether the agreement includes provision for tail coverage if the policy is cancelled prematurely. The employer and employee should each consult legal counsel. Attorneys drafting or reviewing physician employment contracts should be familiar with the differences between occurrence and claims-made policies.

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[1] *Frederick v Clark*, 541 NYS2d 660 (3d Dept, 1989).

Avicenna, problem solving, and creativity during sleep

TO THE EDITOR: One of the earliest self-descriptions by a physician regarding problem solving during sleep was written by the renowned Persian, Ibn Sina (980–1037), called Avicenna by medieval writers. He studied medicine when very young and at 18 years of age was already famous. When he was 21 years old he wrote a 20-volume encyclopedia. His most important medical publication was *The Canon of Medicine.* It was divided into five sections dealing with the theory of medicine, the simpler drugs, special pathology and therapeutics, general diseases, and the pharmacopoeia. The distinguished medical historian Henry E. Sigerist quoted him in his book, *The Great Doctors*, subtitled *A Biographical History of Medicine.* Avicenna stated,

> At home at nights, by lamplight, I read and I wrote, and when I grew so sleepy that I felt my powers of work were failing me I drank a glass of wine to restore my energies and resumed my labors. When at length I fell asleep, I was still so full of my studies that often on waking I found that problems which had perplexed me had been solved during slumber. Thus I continued my studies until I had attained a complete knowledge of dialectics, physics, and mathematics. Then I turned to theology and metaphysics.[1]

One of the outstanding examples of such problem solving involved the conception of the benzene ring, arrived at by the German chemist Friedrich August Kekulé von Stradonits (1829–1896). Kekule's account was related by Beveridge when he wrote about the art of scientific investigation.[2] Kekulé visualized atoms flitting before him, then in rows turning like snakes, and finally one snake seizing its own tail. "As though from a flash of lightning I awoke; I occupied the rest of the night in working out the consequences of the hypothesis."

S. Weir Mitchell (1829–1914), the eminent American neurologist and neuropsychiatrist, was also a novelist, short story writer, and poet. His creative experiences composing poems during sleep, two of which were commented on by him, were published.[3] It is of interest that Ibn Sina, "the Prince of Physicians" as he was called by his contemporaries, and regarded by Sir William Osler as "one of the greatest names in the history of medicine," was also a poet. He wrote his scientific works in Arabic but his highly regarded poetry in his native Persian. It has been alleged that several quatrains ascribed to Omar Khayyam were composed much earlier by Avicenna.[4]

Problem solving during sleep by fictional characters occurs in novels by S. Weir Mitchell, among others.[5]

Creativity and problem solving during sleep deserve more scientific attention and study than they have merited heretofore.

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The turbulent years

TO THE EDITOR: The signs are already there. The next decade will in all likelihood be turbulent years for physicians. The initiatives that emanate from Washington, DC, and Albany are an understandable mix of proposals and prayers. They are put forth with the prayerful hope that they will not turn out to be disasters. From innumerable conversations with legislators it is apparent that they are only too well aware that the consequences of some of their actions may lead to serious problems of access to health care. As never before, they need to hear from the physicians in their districts as to what these proposals will mean to patients. This problem is even more pronounced in rural and inner city areas.

As law makers consider the possibilities, they need to understand that physicians cannot be made the scapegoats and the burden carriers for all the ills of a health care system in deep trouble. Physicians can no longer passively accept plans that do not carry the imprimatur of practicing physicians.

It is true that in the past, generations of physicians merely had to provide care for their patients and a grateful society protected them from the slings and arrows. This no longer obtains and unless we, as physicians, become an integral part of the process, we will be viewed as part of the problem.

The sad fact is that legislators often want to hear from the physicians in their districts and only get called at crisis time. There needs to be an ongoing dialogue, not a last minute attempt to sway elected officials. These men and women are intelligent, concerned individuals who are seeking to make health care available and accessible to their constituents. We are uniquely trained to furnish the input and to forgo this opportunity is tantamount to surrendering our patients and ourselves to a fickle fate and an uncertain destiny.

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Eosinophilia myalgia syndrome

As of February 23, 1990, 1,321 cases of the eosinophilia myalgia syndrome (EMS) have been reported by all 50 states, the District of Columbia and Puerto Rico to the Centers for Disease Control (CDC). New York State has reported 132 EMS cases, second only to California's 236 reported cases. Fifteen deaths caused by EMS have been reported to the CDC, including four deaths among New York State residents.

EMS was first identified in fall 1989, when the New Mexico Department of Health was notified of three patients with eosinophilia and severe myalgias, who were taking oral preparations of the amino acid L-tryptophan (LT). Subsequent investigation revealed an additional 30 cases in New Mexico, prompting the CDC to issue an advisory.

According to CDC guidelines, "EMS is an illness characterized by (1) eosinophil count >1000 cells per mm,^{3} (2) generalized myalgia (at some point during the course of illness) of severity sufficient to affect a patient's ability to pursue his or her usual daily activities, and (3) absence of infection or neoplasm that could account for 1 or 2 above." (Trichinosis is the most common infection causing eosinophilia and myalgia.) Note, the use of LT is not an element of the case definition.

Almost all cases of EMS in the United States have been associated with the use of supplemental manufactured L-tryptophan. Approximately 2% of patients nationwide did not use LT. The CDC reported that they received preliminary reports of EMS in three patients who used only L-lysine supplements, one patient who used niacin and one patient who had used a topical tanning preparation containing LT.

Most manufactured LT distributed in the United States is produced by five Japanese companies, utilizing a fermentation process. EMS may be caused by a substance contaminating LT during manufacture. Preliminary epidemiologic evidence from two states links LT used by EMS patients to particular lots of LT manufactured by one company. A Japanese site visit by the Food and Drug Administration (FDA) as well as extensive laboratory analysis by both the CDC and the FDA have not uncovered a contaminant.

The epidemic curve of EMS cases in New York State (Fig 1) shows a sharp increase in early summer 1989 (86% became ill as of June 1989), suggesting that a material may have recently contaminated LT sold in the United States. Yet, ten EMS patients (10%) became ill prior to January 1989; the first reported illness was in September 1987. Only two cases were reported for December 1989 and none since then, reflecting the effect of a ban on the sale of LT imposed in mid-November.

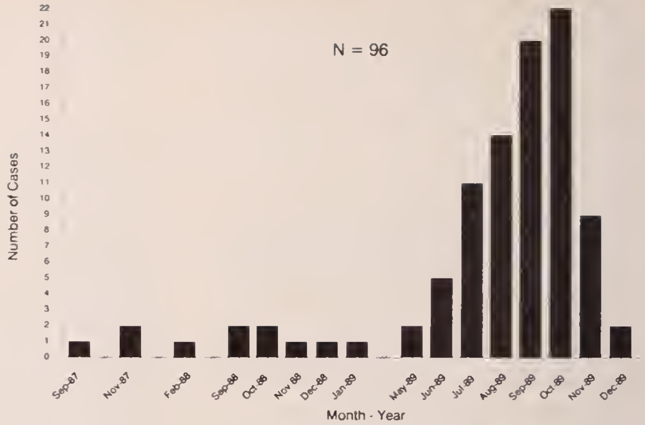

FIGURE 1. Date of onset of illness, EMS patients, New York State.

On November 17, 1989, the State Department of Health banned the sale and distribution of products containing manufactured LT. This order was modified on November 24, 1989, to allow the medically supervised use of products containing LT for purposes of providing essential nutrition (eg, parenteral and enteral nutrition). The New York ban applies to products containing less than 100 mg. On March 22, 1990, the FDA revised its ban to include products containing less than 100 mg. Although no EMS case reported thus far involved essential nutritional products, the sources of LT in a product may be the same as those in dietary supplements associated with EMS.

This report summarizes the clinical aspects of EMS experienced by the first 96 New York patients who were reported to the State or City Departments of Health. Most information has been gathered through questionnaires. Other information was gathered from telephone conversations with EMS patients and their physicians or from the CDC. All information is preliminary and certain clinical and/or pathologic reports have not been confirmed.

Demographics, Host Factors and LT Use

Patients range in age from 22 to 80 years (mean: 51 years); 85% are female, 97% are white, 2% are Hispanic and one patient is black. Patients have been reported from 26 New York counties; the greatest number reported are from Long Island (32%) and second greatest number from New York City (23%).

Only 15% of patients reported having a nonpsychiatric chronic illness prior to developing EMS. Many reported a history of psychiatric disorders including depression, eating disorders, schizophrenia, anxiety disorder and/or alcohol and substance abuse. Of the 73% of patients who supplied information regarding allergies, 49% reported an allergic history. No patients were pregnant while symptomatic with EMS.

All but one patient reported using LT prior to developing EMS symptoms; dosages ranged from 500 to 8,400 mg/day (mean: 2,032 mg). Nationally, the lowest reported LT dose used by an EMS patient was 26 mg/day. Duration of use ranged from two weeks to eight years before onset of symptoms (mean: approximately 1.5 years). Although most patients developed symptoms prior to discontinuing LT, two developed symptoms two weeks after their last use of LT. Eighty percent of patients used LT daily while 20% used it intermittently. Patients reported using 24 different brands of LT-containing pills or capsules; two brands accounted for nearly half of the cases. Most (82%) reported they used LT for treatment of insomnia, with other less frequently cited indications including treatment of premenstrual symptoms (PMS), depression, anxiety, headaches, sleep apnea, impotence and/or eating disorders, as well as a diet aid.

Clinical Signs and Symptoms

EMS patients manifest a wide variety of signs and symptoms. Every organ system may be involved. Signs and symptoms reported by EMS patients include:

- severe myalgias (87%);
- arthralgias (49%);
- fever (40%)—usually low grade;
- cough or dyspnea (65%)—many patients reported an upper respiratory infection or flu-like illness before or along with other EMS symptoms; several patients, who did not meet the EMS case definition because they did not have myalgias, had eosinophilia with severe respiratory symptoms;
- rash (68%)—described as erythematous, maculopapular, and/or urticarial; some patients reported pruritus without a definite rash;
- neuropathy or neuritis (29%)—the most serious complication of EMS is the development of an ascending polyneuropathy, which may continue to progress despite discontinuing LT; one patient, who subsequently died of a possible arrhythmia, developed a near-total quadriplegia and bifacial hemiparesis; another patient is currently on a respirator because of a Guillain-Barré like syndrome;
- periorbital edema (26%);
- peripheral edema (55%)—described as a brawny nonpitting edema, often extreme with >10-15 pounds of weight gain;
- sclerodermiform skin changes (28%)—these skin changes often developed following the initial presentation of EMS;
- alopecia (27%).

Other signs and symptoms reported by patients include extreme fatigue, anorexia, weakness, itching, skin burning, muscle tenderness and cramping, finger spasms, scalp tenderness, bone pain, mouth lesions, sicca, tachyarrhythmias, nausea, vomiting and diarrhea.

Laboratory Features

The following abnormal laboratory features have been reported:

- leukocytosis (85%)—WBC count ranged from 5,900 to 35,200 cells/mm^3 with a mean of 15,400 cells/mm^3;
- eosinophilia (100%)—the percentage of eosinophils in the WBC differential ranged from 13% to 88% with a mean of 39%; absolute eosinophil counts (WBC × %Eosinophil) ranged from 1,250 to 26,750 cells/mm^3 with a mean of 6,570 cells/mm^3;
- abnormal chest X-ray—72% of patients had a chest X-ray; of these, 13% had an effusion and 22% had an infiltrate;
- elevated erythrocyte sedimentation rate (33%)—modest elevation (20-50 mm/hr);
- elevated aldolase (66%)—most between 2-3 times normal;
- elevated CPK (8%);
- abnormal liver function tests (48%)—most between 2-3 times normal;
- pathology—a muscle biopsy was obtained from 42% of the patients. Pathologic findings have included vasculitis, perivascular inflammation, eosinophilic fascitis, myositis and muscle atrophy. Several biopsy specimens from the skin, lungs and cecum of EMS patients have revealed inflammatory infiltrates with a predominance of eosinophils. Bone marrow examinations have been normal or have shown eosinophilia.

Prognosis and Treatment

A wide spectrum of illness has been reported in EMS patients, with symptoms ranging from mild to totally incapacitating. Patients with milder symptoms appear to improve more rapidly and completely than those with more severe symptoms.

Forty percent of New York patients with EMS have been hospitalized at least once. Although many patients report that they are slowly improving, some patients are still quite ill several months after stopping the use of LT. Many patients report that their symptoms are intermittent, often worsening as the day progresses.

The clinical manifestations of some EMS patients resemble those seen in victims of the toxic-oil syndrome (TOS), an illness that affected 20,000 individuals in Spain during 1981, which was associated with the ingestion of contaminated rapeseed oil. Some patients with chronic TOS developed peripheral neuropathy (mononeuritis multiplex), thromboembolic phenomena, sclerodermiform skin changes, joint contractures and pulmonary hypertension. The specific contaminant linked to the development of TOS has never been elucidated.

Four EMS patients, all women, have died; the first had an unwitnessed sudden death two months after the development of an ascending polyneuropathy. A second patient, with multiple medical problems and the onset of symptoms of EMS in November 1987, died recently of an apparent myocardial infarction. A third patient (in whom exposure to LT is uncertain) died of an apparent hemorrhagic stroke approximately three months after developing EMS symptoms. A fourth patient, who was HIV negative and had no risk factors for developing HIV infection, died of *Pneumocystis carini* pneumonia five months after developing EMS symptoms.

The most important therapeutic intervention in EMS patients is the elimination of L-tryptophan use. Many physicians have been treating their patients with high dose steroids. They report that while the eosinophilia resolves rapidly following the initiation of steroid therapy, symptomatic improvement often lags. Symptoms and/or eosinophilia often recur in patients who are too rapidly weaned from steroids. Some patients have been maintained on at least low dose alternate day steroid therapy for months. Other experimental therapies are currently being evaluated.

The State and City Departments of Health are currently conducting several studies evaluating the clinical course of EMS and possible etiologic factors. Physicians who know of possible cases are encouraged to contact the Department of Health at (518) 458-6228 or the City Health Department at (212) 566-7187. Confidentiality of all medical information will be maintained pursuant to the Public Health Law.

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Injury mortality in New York State

Nationwide, injuries are the fourth leading cause of death, and the leading cause of death for people between the ages of 1 and 44. The National Committee for Injury Prevention and Control estimates " . . .on an average day, more than 170,000 men, women, and children are injured seriously enough to need medical care; nearly 400 die as a result of their injuries."[1] In 1985, there were 155,665 injury deaths in the United States, giving an annual rate of 65.7 deaths per 100,000 residents.[2] Since injury mortality rates vary with sociocultural factors and among geographic regions, this article examines injury mortality more closely on a statewide basis.

Injury mortality data were obtained from death records of state residents between 1984 and 1988. Five years of data were examined to minimize random fluctuations in smaller subsamples and injury categories. Injury deaths were defined as those deaths listing an ICD-9-CM E code (E800-999) as the underlying cause of death. A small number of deaths were excluded, such as surgical/medical misadventures and complications (E870-879), adverse therapeutic drug use (E930-949), legal intervention (E970-978) and operations of war (E990-999). The remaining injury deaths were classified into three groups: unintentional, intentional (suicide and homicide) and undetermined intention. Unintentional deaths were further classified into subcategories based on the agent of death (eg, motor vehicle).

Annual population size estimates were obtained from the Bureau of Biometrics of the New York State Department of Health. Average annual rates were obtained by dividing the cumulative number of deaths by the cumulative population size.

For the five-year period 1984–1988, there were 43,886 injury deaths in New York State, an average of 8,777 deaths per year. The annual rate of all injury deaths in New York State is 49.5 per 100,000 residents. The ten leading causes of injury death, shown in Table I, account for 95% of all injury deaths in the state.

Statewide, the leading cause of injury death is motor vehicle incidents (24.7%), followed by homicide (22.0%), suicide (18.0%), falls (10.2%) and those where intention is unknown (in 8.6% of these, the major subcategory is poisoning deaths). The annual injury death rate for New York City (55.8 per 100,000 residents) is significantly higher ($p < 0.001$) than that of the remainder of the state (45.1 per 100,000 residents). The leading cause of injury

TABLE I. Top Ten Causes of Injury Death in New York State, 1984–1988

		New York State			New York City			Remainder of New York State		
Category	ICD-9 codes	Number	Rank	Rate	Number	Rank	Rate	Number	Rank	Rate
Unintentional										
Motor vehicle	E810-819	10853	1	12.2	2788	3	7.7	8065	1	15.4
Drowning	E830, 832, 910	1081	8	1.2	330	7	0.9	751	9	1.4
Poisoning	E850-869	906	9	1.0	82	9	0.2	824	7	1.6
Falls	E880-888	4488	4	5.1	2084	5	5.8	2404	3	4.6
Fire	E890-899	1645	6	1.9	616	6	1.7	1029	5	2.0
Environmental	E900-909	277	10	0.3	57	10	0.2	220	10	0.4
Choking/suffocation	E911-913	1171	7	1.3	220	8	0.6	951	6	1.8
Intentional										
Suicide	E950-959	7919	3	8.9	2708	4	7.5	5211	2	9.9
Homicide	E960-969	9644	2	10.9	7846	1	21.7	1798	4	3.4
Intent undetermined	E980-989	3771	5	4.3	3004	2	8.3	767	8	1.5
All other unintentional		2131		4.9	464		1.3	1667		3.2
All injury deaths		43886		49.5	20199		55.8	23867		45.1

Note: the number of deaths refers to the 5 year total. Rates are annual rates per 100,000 residents.

death in New York City is homicide, whereas the leading cause of injury death for the remainder of the state is motor vehicle incidents. The statewide rate of injury deaths is 74.8 per 100,000 male residents and 26.7 per 100,000 female residents, a highly significant difference ($p < 0.001$). In New York City, the male rate is 90.0 and the female rate is 26.7. For the remainder of the state, the male rate is 64.8 and the female rate is 26.7.

Further breakdown is presented in Table II, where the leading cause of injury death by sex, age group, and region is listed. The leading cause of injury death statewide is homicide for males and motor vehicle incidents for females. In New York City, the leading cause of injury death for both sexes is homicide, and for the remainder of the state the leading cause of injury death (both sexes) is motor vehicle incidents. In New York City, homicide is the leading cause of injury death for all age groups except for those 5–14 (motor vehicle) and those over 65 (falls). For the remainder of the state, fire is the leading cause of injury death for those 0–4 years of age, motor vehicle incidents for those 5–44 years of age, suicide for those 45–64 years of age and falls for those over 65 years of age. The results of this analysis indicate that, while injuries are a major cause of death in New York State, specific causes of injury death vary by sex, age and region. This information is useful in surveillance efforts, in targeting injury prevention programs to those at risk for specific causes of injury deaths and in measuring the success of these programs.

Further information is available from the Injury Control Program, Division of Epidemiology, (518) 473-1143.

TABLE II. Leading Cause of Injury Death by Sex, Age and Region, 1984–1988

Age Group By Region	Males	Females	Total
New York State			
0–4	Homicide	Fire	Homicide
5–14	Motor Vehicle	Motor Vehicle	Motor Vehicle
15–24	Motor Vehicle	Motor Vehicle	Motor Vehicle
25–44	Homicide	Motor Vehicle	Homicide
45–64	Suicide	Motor Vehicle	Suicide
65+	Falls	Falls	Falls
Total	Homicide	Motor Vehicle	Motor Vehicle
New York City			
0–4	Homicide	Homicide	Homicide
5–14	Motor Vehicle	Motor Vehicle	Motor Vehicle
15–24	Homicide	Homicide	Homicide
25–44	Homicide	Homicide	Homicide
45–64	Homicide	Suicide	Homicide
65+	Falls	Falls	Falls
Total	Homicide	Homicide	Homicide
Remainder of New York State			
0–4	Fire	Fire	Fire
5–14	Motor Vehicle	Motor Vehicle	Motor Vehicle
15–24	Motor Vehicle	Motor Vehicle	Motor Vehicle
25–44	Motor Vehicle	Motor Vehicle	Motor Vehicle
45–64	Suicide	Motor Vehicle	Suicide
65+	Falls	Falls	Falls
Total	Motor Vehicle	Motor Vehicle	Motor Vehicle

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MERRITT'S TEXTBOOK OF NEUROLOGY

Eighth Edition. Edited by Lewis P. Rowland, MD. 964 pp, illustrated. Philadelphia, Pa, Lea & Febiger, 1989. $67.50 (hardcover)

For three decades this book has been recognized as a standard in clinical neurology. Conceived as a basic yet comprehensive volume, the book was authored solely by Dr Merritt for the first six editions. This revision contains the work of 75 contributing authors, many of them students of Dr Merritt who have become leaders in the field. Editor Dr Lewis Rowland, a renowned expert in neuromuscular disease, provides the editorial prowess which unifies the book into a coherent work. The authors' collective goal is simple: "to provide a historical document by Dr Merritt's students and to do that in a way that he would have approved of: making a textbook that would be useful."

The work is encyclopedic with 162 sections categorically grouped into 22 chapters. The sections are subject specific and can be read as independent entries. Every section concludes with a pertinent list of references.

The first chapter develops a clinical approach to common neurological complaints. Alterations in consciousness and cognition, aphasia, apraxia, pain, paralysis, involuntary movements, and disorders of gait and of the special senses are individually considered. Differential diagnoses are presented with taut clarity. Although the lack of anatomical illustrations may hinder the nonspecialist reader, the chapter is memorable for its lucid presentation and completeness.

Subsequent chapters detail the fundamental disease categories. Infectious, vascular, tumor-related, traumatic, degenerative, neuromuscular, demyelinating, paroxysmal, pediatric, and movement disorders are presented, along with the neurology of systemic illnesses.

These chapters are contemporary, authoritative, and comprehensive. A generous use of tables allows for rapid assimilation and comparison, which is especially useful in the pediatric sections. Numerous photographs highlight the topics. Their reproduction is of exceptional quality and permits demonstration of precise detail. Clinical, pathological, and radiographic illustrations are presented. The emerging diagnostic importance of magnetic resonance imaging is evidenced by the number of scans included in this revision. Discussions of current pharmacotherapeutics appear in the various treatment paragraphs. Duly noted is the expansion of the neurological pharmacopeia by calcium channel blockers, MAO B inhibitors, botulinum toxin, antivirals, and newer generation antibiotics.

Novel or revised entries for this edition include the following sections: HIV infection, Lyme disease, neuro-oncology, the neurology of pregnancy, mitochondrial and peroxisomal diseases, sleep disorders, drug abuse, tropical myelopathies, and intracranial hypertension. The differential diagnosis of cerebral degenerations of childhood and respiratory care in neurological patients are particularly well done and informative. However, the intrinsic complexities of molecular biology, neurogenetics, and critical care neurology would be better served by individually dedicated sections. Likewise, a section exploring the theoretical origins of electrodiagnostic techniques (EMG, evoked potentials, quantitative EEG) would be useful in clarifying their clinical utility.

This edition of *Merritt's Textbook of Neurology* continues the book's legacy as a premier text and an authoritative reference work. It remains a required acquisition for medical libraries and for serious students of clinical neurology.

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THE TRUTH ABOUT HERPES

Third Edition. By Stephen L. Sacks, MD. 216 pp, illustrated. Seattle, Wa, Gordon Soules Book Publishers Ltd, 1988. $14.95 (paperback)

Sexually transmitted diseases have always lent themselves well to "bathroom humor" and inaccurate rumor. Infection with herpes virus can be painful, debilitating, and a source of great embarrassment, especially to those patients who are affected but remain unaware of the true nature of the disease. In his book, *The Truth About Herpes*, Dr Stephen L. Sacks attempts to clear up the misconceptions surrounding the disease and inform the public at the same time.

Dr Sacks is associate professor of medicine at the University of British Columbia, and the founder/director of its herpes clinic. He is a specialist in infectious disease with specific interest in genital herpes. Although not a medical textbook in the strictest sense, this book provides quite detailed and generally accurate information about the disease. Inevitably, Dr Sacks' individual philosophy about various management options is presented in the book, though he is quick to point out which statements involve personal bias.

This book is especially well suited to the lay community. It is generally written in a "question and answer" format, making it easy to find information on a specific topic. Specific discussions include a description of the virus itself, potential symptoms, methods of diagnosis and modes of transmission. One chapter that may prove especially useful is that of the "psychology of herpes," in which Dr Sacks explores the normal feelings of anger and guilt associated with the disease and offers suggestions about its discussion.

Description of the herpes virus is given in a complete yet easily comprehensible manner. The subject of herpes infection is discussed in great detail for a nonmedical text, and it should be easily understandable by those not directly involved in the health care professions. References are supplied at the end of the book for further reading. Illustrations and photographs are provided and are especially useful when discussing a disease whose potential transmission depends on detection of physical characteristics. Factual information is presented as such, and for the most part correctly. One exception is in the discussion of herpes infection in the pregnant patient; the new American College of Obstetrics and Gynecology (ACOG) recommendations suggest that weekly herpes cultures are no longer required and the only indication for cesarean section is that of the presence of prodromal symptoms or an active lesion. As Dr Sacks himself states, weekly cultures have not been shown to be effective and this small error results from the

ACOG change after the book had already been printed.

It appears that this book was written specifically for the lay public, and is one of the best texts written in such a manner to date. It is forthright and honest, yet is not intimidating or pessimistic in its discussion. Herpes is a continually growing problem all over the world, but there has been less and less discussion of it in the media lately as a result of the growing AIDS epidemic. Infection with the herpes virus is no less real to these patients, and Dr Sacks' book should provide an excellent source of information and comfort to them.

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HEART DISEASE IN INFANTS, CHILDREN, AND ADOLESCENTS

Fourth Edition. Edited by Forrest H. Adams, MD, George C. Emmanouilides, MD, and Thomas A. Riemenschneider, MD. 1,066 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1989. $149.95 (hardcover)

This voluminous book (1,066 pages) is the fourth edition of the leading textbook for the diagnosis and management of heart diseases in children. Since the first edition, published over 20 years ago, there have been many advances in pediatric cardiology, with perhaps the most significant advances in both diagnostic techniques and treatment made between the appearance of the third and fourth editions. Similar to the previous edition, this new volume covers the subject thoroughly with proper emphasis on new developments in the field.

More than 100 authors have contributed to this edition, most of them known authorities and leaders in their respective fields. Therefore, the specific chapters and statements made carry special weight. It is a timely and useful edition during this period of revolutionary strides in this field.

The general layout of the book has been preserved and includes sections on general cardiology, congenital defects, infectious diseases, connective tissue, metabolic and degenerative diseases, and general problems in pediatric cardiology. Many new chapters have been added or thoroughly reviewed.

In the section on general cardiology, the chapter on the development of the cardiovascular system has been reintroduced. The recent advances in Doppler ultrasound are properly presented with a new chapter by a leading authority in the field devoted to this emerging technique.

Similarly, a new chapter on magnetic resonance imaging with excellent pictures and brief sections on magnetic resonance flow studies and myocardial uptake techniques increases our appetite for the future applications of this new and promising technique. The invasive diagnostic section is excellent, with chapters on both diagnostic and therapeutic cardiac catheterization. The chapter devoted to invasive electrophysiologic studies is very brief. One is led to believe that the editors felt that expanded information on this subject is probably beyond the scope of this textbook and should be found in other books especially devoted to this topic. The brief section on myocardial biopsies is written by a pioneer in this field.

The second section, on congenital defects, has mostly remained without change. The introduction of a new chapter on the difficult subject of pulmonary atresia and ventricular septal defect is proper, and although the authors concede to the difficulties associated with managing such patients, the general impression still may be somewhat more optimistic and rosy than many of us feel when taking care of such patients. A welcome addition to this section is the expanded chapters devoted to the adult with congenital heart disease, either prior to heart surgery, or following it. The new chapter on pregnancy and congenital heart disease is also a welcome addition. The subject of the adult with a treated congenital heart disease is assuming a more significant role as more and more patients with treated congenital heart disease become adults.

Sections three and four underwent relatively minor upgrading and one wonders whether the discussion of Kawasaki's disease should not be significantly expanded. It seems that the current practitioner faces many more patients with Kawasaki's disease with cardiovascular complications than patients with acute rheumatic fever and rheumatic heart disease.

The last section on special problems of pediatric cardiology has been significantly expanded with excellent and timely chapters on prenatal diagnosis and fetal monitoring, persistent pulmonary hypertension in the newborn, and timely chapters on shock and transplantation. The role echocardiography has assumed in the diagnosis of congenital heart disease is probably best demonstrated in the chapter on cardiac surgery without cardiac catheterization. In this section, there is a brief discussion of ambulatory electrocardiography, which probably should be placed with other non-invasive diagnostic techniques presented in the general section on cardiology. It is of interest that this section is the only one where the issue of sudden infant death syndrome is mentioned. Sudden infant death syndrome accounts for the death of 2–5 out of 1,000 infants, perhaps more deaths than those resulting from congenital heart disease. Whereas many rare syndromes are cited in the index, no mention of sudden infant death syndrome, a topic of significant public and professional concern, can be found. A chapter devoted to the general discussion of sudden death in the pediatric population is missing in the volume; such a discussion belongs among the chapters on pulmonary hypertension, heart failure, and shock.

In general, the quality, accuracy and clarity of the book are outstanding. Many readers of previous editions will find the general organization, style, and technical features of this edition familiar. There is no question that the volume is an outstanding and timely addition in this field, and indeed with time has become the leading publication on this subject.

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BILIARY TRACT SURGERY: TACTICS AND TECHNIQUES

By Jacob A. Glassman, MD. 416 pp, illustrated. New York, Macmillan Publishing Co, 1989. $90.00 (hardcover)

This book deals with biliary tract surgery and the events preceding it. The author is a surgeon with a lifetime practice and interest in surgery. He is a gadgeteer who has fashioned many instrument models to aid in biliary surgery, some of which have found only limited acceptance among other surgeons. The book covers an enormous area, from liver transplantation to chemical dissolution of gallstones. While the parts are of interest, some entries seem to have been written quite some time before publication of the book. These have been updated by addendums by the authors. The editing of the book is somewhat deficient, ie, authors' initials are mixed and the legends do not always correspond to the illustrations. The author's prose is written from quite a personal viewpoint, and at times he adds case reports to his discussions. The value of these is sometimes debatable. There are many well written chapters in this book and the principal author's discussions are often a pleasure to read. They are obviously the thoughts of a man who has been deeply immersed in his undertaking. The book sells for $90 and is thus probably out of reach except for those with a special interest in the field. For these, the book makes interesting reading and will provide productive thoughts with respect to many of the problems facing the biliary tract surgeon.

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THE RIGHTS OF PATIENTS

Second Edition. By George J. Annas, JD, MPH. 312 pp. Carbondale, Ill, Southern Illinois University Press, 1989. $8.95 (paperback)

This book is part of a series of American Civil Liberties Union handbooks. It is meant to serve as a guide for hospital patients and their families so that they can be aware of both their ethical and legal rights in dealing with the health care system and particularly hospitals.

George Annas, a professor of health law and a frequent author of articles on medical ethics, is extremely well qualified to present this material.

I believe this book to be a valuable addition to the library of any practicing physician or health care administrator as it presents an excellent overview of major medical legal issues from the point of view of patient rights. A most difficult feature that is handled quite well is that laws differ from one state to another. This problem is overcome through citing certain examples and providing the ethical underpinning for the creation of legislation covering a subject. Also, the author discusses certain areas where the rights of patients are limited by the rights of the practitioner or hospital. For example, while a patient has the right to refuse specific treatment, if a patient consistently refuses to participate in any treatment the hospital might be justified in asking the patient to leave.

On the whole, I found this book to be a balanced approach to the subject of patients' rights with the exception of certain areas alluding to an adversarial relationship between the patient and the hospital. As a hospital administrator, I find that some statements made in this book contribute to this adversarial relationship, for example, "Even if you are unable (or unwilling) to read the book, people have reported that their care in hospitals has improved simply by having the book on their bedside table." This statement does not serve any positive purpose. The chapter on "The Patients' Rights Advocate" is also, in my mind, questionable.

I think this book makes excellent reading for the health care professional, but may be tedious and difficult for the average patient.

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BLOOD, SWEAT, AND CHEERS

By Sir Ian Fraser, 150 pp. London, British Medical Journal, 1989. $29.00 (hardcover)

This very pleasant partial autobiography by the late Sir Ian Fraser covers 60 years of surgical experience that began after the First World War. It also cover his active duty in the Second World War and ends in 1988. A successful practitioner, he was selected by his colleagues to serve as President of the Royal College of Surgeons in Ireland as well as President of the British Medical Association. Sir Ian writes very well. His choice of words and phrases reveals a keen sense of humor in a modest man who loved his profession and shared a happy home life with a loving wife and family. He tells his story succinctly and uses a continuous series of anecdotes to portray the changes that occurred in surgery during his lifetime. I liked his reference to the "three A's appearing in the 19th century—anaesthesia, antisepsis and asepsis" and their relationship to the rapid advances made in the 20th century. He pays special tribute to Von Bergmann (1880s) who introduced asepsis in surgery to "prevent the germ" in contradistinction to Lister's "kill the germ."

Sir Ian's story is familiar and his contemporaries, of whom I am one, will enjoy his reminiscences. We mourn the death of clinicians like the author. He was obviously influenced by his father and grandfather both of whom were general practitioners. The latter and a district nurse made a house call on a patient afflicted with typhoid fever. The patient recovered but Sir Ian's grandfather and nurse died of the disease.

The illustrations by Chrislaine Howard, sketched in black and white, are excellent.

I suspect that modesty has deprived us of many interesting, educational, and historical stories of the lives of prominent physicians who influenced most of us. I long for Ian Fraser types of memoirs written by my old chief, Dr William Dock, and by others. All of us have personal favorites; please forgive my self-serving selection. Read this gem and join me in thanking Sir Ian.

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Treatment of acute spinal-cord injury with methylprednisolone shows promise

Methylprednisolone is believed to have two major effects on injured spinal cords beyond its ability to activate corticosteroid receptors: enhancing the flow of blood; and blocking the lipid peroxidation and hydrolysis that results in breakdown of membrane. Findings from the National Acute Spinal Cord Injury Study 2 (NASCIS 2) demonstrate "a significant improvement in motor function and the sensations of pinprick and touch six weeks and six months after injury in the patients treated with methylprednisolone" (*N Engl J Med* 1990; 322:1405–1411).

Bracken et al compared the effects of methylprednisolone, naloxone, and a placebo on the motor and sensory outcomes of patients with spinal cord injuries. Patients, all of whom entered the study within 12 hours of their injuries, were randomly grouped according to three treatment protocols: methylprednisolone; naloxone; or placebo. Methylprednisolone was administered in a 30 mg/kg bolus dose over a 15-minute period, followed by a 45-minute period with no drug, and then a 23-hour maintenance infusion given at 5.4 mg/kg/hour. The naloxone bolus dose was 5.4 mg/kg, with a maintenance dose of 4.0 mg/kg/hr begun after the 45-minute waiting period.

Testing for neurologic function was performed on admission to the hospital and after six weeks and six months. Assessment of motor function was based on measurements of muscle contraction. Sensory studies included pinprick and light touch tests. The authors analyzed the data in light of the severity of injury and the time elapsed between the injury and the administration of a loading dose (greater than or less than eight hours).

Six weeks after injury, an evaluation of all the patients revealed greater improvement in pinprick and touch sensations for patients who received methylprednisolone, compared to placebo, with no differences in motor function. However, for patients who received methylprednisolone within eight hours of injury, there was significant improvement in motor function and touch compared to the placebo group. No significant changes in neurologic function were evident for the naxolone group. The greater improvement in motor function for patients who received methylprednisolone within eight hours of injury persisted at the six-month evaluation. These patients also had heightened sensory function at six months.

Methylprednisolone produced improved neurologic function in patients with more severe and less severe spinal cord injuries. "Clearly, any notion that patients seen in the emergency room with complete spinal-cord injuries will be unresponsive to therapy must be reconsidered," observe the authors. Prompt treatment is essential, as methylprednisolone exerted its beneficial effects in patients who received the drug within eight hours of sustaining their injury. The lipid peroxidation and hydrolysis believed to precipitate the breakdown of neurofilament in the spinal cord begin and peak within eight hours of an injury.

Methylprednisolone-induced improvements in neurologic activity "presumably reflect biologic changes in the spinal cord and should provide the basis for the patient's subsequent rehabilitation," according to the authors. "Previous work . . . showed that motor and sensory function have independent and additive effects on locomotor recovery and self-care after injury."

FDA supports generic drug use

Following testing of more than 2,500 samples of the 30 most prescribed generic drugs and inspection of 36 generic drug companies and 12 contract laboratories that evaluate the bioequivalence of generic drugs, the Food and Drug Administration (FDA) has recommended that physicians continue to prescribe generic drugs whenever appropriate (*FDA Drug Bulletin* 1990; 20(1):4–5).

The FDA's finding that about 1% of the sampled drugs did not meet the accepted standards for potency, dissolution, content uniformity, product identification, and purity was consistent with the usual findings for brand name and generic products. The FDA's inspections of drug manufacturing operations uncovered several cases of falsification of records. These findings led to the withdrawal of various approvals as a precautionary measure.

Hypercalcemia and carcinoma of the esophagus

Esophageal carcinoma usually presents with dysphagia or some local symptom, and sometimes with evidence of bone metastasis. Vaswani and Dhodhapkar reported a case of esophageal squamous cell carcinoma in which the sole presenting symptom was hypercalcemia (*Am J Gastroenterol* 1990; 85:621).

The 80-year-old male patient had no history of dysphagia when he was admitted to the hospital for treatment of altered mental status. He displayed no neck masses and no lymphadenopathy. His serum calcium level was 22.2 mg/dL, and the phosphorus level was 4.7 mg/dL. A bone scan revealed no metastases. Nodular lesions in the proximal esophagus were found during upper esophagogastroduodenoscopy for gastrostomy tube placement. Biopsy of the lesions led to a diagnosis of moderately well-differentiated squamous cell carcinoma of the esophagus.

The authors attribute the hypercalcemia in the absence of bone metastasis to "parathyroid hormone-like peptides synthesized by the tumor." Squamous cell carcinoma of the esophagus accounts for 1.1% of all cancers, 7% of cancers of the digestive organs, and 4% of all deaths from cancer. It is the fifth most common cancer affecting adult males. Early diagnosis is crucial for effective treatment or palliative therapy. Esophageal carcinoma can present without local symptoms of obstruction or dysphagia.

Nintendo-induced epilepsy

Photosensitive epilepsy is not uncommon, affecting about 2–3% of patients with epilepsy and 1 in 10,000 of the general population. Flickering light of various rates can precipitate generalized tonic-clonic seizures in sensitive persons. Sources can include television, sunlight flick-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

ering through trees while one is in a moving vehicle, and disco strobe lights.

Hart coined the term "Nintendo epilepsy" to describe the tonic-clonic seizure experienced by a 13-year-old girl while playing "Super Mario Brothers," a Nintendo video game (*N Engl J Med* 1990; 322:1473). "Electroencephalographic evaluation five days later revealed a photoconvulsive response consisting of polyspikes and slow waves during photic stimulation above frequencies of 8 Hz. At 16-Hz stimulation, she felt symptoms similar to those that preceded her seizure, and thus the test was terminated."

The patient and her parents opted for treatment with an anticonvulsant drug rather than avoiding video games. Similar epileptic seizures induced by another video game (as was described in England) led the author to conclude that "the phenomenon is almost certainly not game-specific."

Leukemia in men may be related to smoking

Two prospective studies administered by the American Cancer Society have demonstrated an association between smoking and the risk of myeloid leukemia in men (*Cancer* 1990; 65:2356–2360). The studies found no relationship between smoking and myeloid leukemia for women, and no association between smoking and lymphatic leukemia for either sex.

In animal studies, the chemicals urethane, nitrosamines, and benzene, as well as radioactive compounds—all of which are components of tobacco smoke—have been shown to cause leukemia. In two prospective studies initiated in 1959 and in 1982, the American Cancer Society compared the smoking habits of more than two million men and women aged 30 years and older with their risk of dying from total, lymphatic, and myeloid leukemia.

In both studies, men who smoked cigarettes had a higher leukemia mortality rate than men who did not smoke. Furthermore, standardized leukemia mortality ratios were greater for current smokers than for ex-smokers. The authors note that, "The positive association between tobacco smoking and myeloid leukemia risk in men may be due, in part, to confounding factors," such as occupational exposure to leukemogenic chemicals or radioactive compounds. There may, however, be a true biologic difference between men and women in the body's response to smoking. Studies in mice suggest that smoking may increase the risk of leukemia in male mice, while smoking may actually reduce the risk of leukemia in female mice.

The effects of prenatal alcohol exposure on child development

Day et al previously demonstrated a correlation between alcohol consumption during pregnancy and low birth weight—defined as less than 2,500 g—and an increased incidence of minor physical anomalies at birth. In the present study, conducted between 1983 and 1986 at an outpatient prenatal clinic, Day and coworkers demonstrated a significant relationship between prenatal alcohol exposure and the growth and morphology of children at eight months of age (*Pediatrics* 1990; 85:748–752).

At the beginning of the study, the 650 participants were all in their fourth month of pregnancy and were at least 18 years of age. The women were interviewed in their fourth and seventh prenatal months, after delivery, and at eight months postpartum. The follow-up rate at eight months was 78%. All of the children were examined at this time. Measurements obtained included weight, length, and head circumference. Morphologic anomalies were categorized as minor or major, with none of the offspring exhibiting fetal alcohol syndrome at birth.

Significantly lower average weights and lengths at eight months of age were correlated with maternal alcohol consumption of one or more drinks per day throughout pregnancy. Daily alcohol use was also associated with reduced head circumference, but with no statistical significance. There was also a significantly increased risk of minor physical anomalies and fetal alcohol effects at eight months of age for the offspring of mothers who consumed alcohol during any of the three trimesters.

By controlling for birth weight, the authors were able to demonstrate that "Not only were the alcohol-exposed offspring smaller . . . but the growth of the infants was slower . . . .Follow-up at additional points will allow us to determine whether this is a permanent reduction in growth rate or a lag in growth that is corrected."

Side effects of beta blockers in patients with migraines

Beta blockers such as propanolol, atenolol, and timolol are commonly prescribed for the prevention of migraine headaches. It is not clear how beta blockers inhibit the development of a migraine, however, the mechanism of action may be related to beta blockers' ability to reduce cerebral blood flow. This action may have a detrimental effect on some migraine patients.

Kumar and Cooney described a woman who experienced typical migraine associated visual symptoms in the absence of headache following treatment with atenolol (*Ann Intern Med* 1990; 112:712). The 37-year-old woman, who had had migraine headaches for 20 years, sought treatment when the frequency of headache increased to three to five times a month. Her headaches were accompanied by scotoma and scintillations only one or two times a year.

After three weeks of treatment with atenolol, 50 mg/day, the patient reported markedly fewer and less severe headaches. However, she also began experiencing scotoma and scintillations two to three times a week for 20–30-minute periods. These episodes were rarely accompanied by headaches. These visual symptoms ceased when the atenolol was withdrawn. She was subsequently maintained on time-release verapamil, 240 mg/day, with no further visual symptoms and good control of her headaches.

The authors propose the following hypothesis: "During the aura of a migraine attack, cerebral blood flow is reduced. By further reducing the cerebral blood flow during the aura, beta blockers could potentially produce cerebral ischemia or infarction." They support their hypothesis with evidence of patients with migraines who suffered strokes while taking propanolol.

When treating patients with migraine, the authors advise against using beta blockers in cases of "complicated migraine" and using them with caution in cases of "classic migraine." These recommendations would apply as well to ergotamines, which also exert a vasoconstrictive effect. Calcium channel blockers, which may increase cerebral blood flow, would be a good alternative for preventing migraines in these patients.

Maynard B. Badanes, MD, New York. Died January 31, 1990; age 77. Dr Badanes was a 1936 graduate of Duke University School of Medicine, Durham, North Carolina. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Cardiology. His memberships included the New York Cardiological Society, the New York State Society of Internal Medicine, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Emma Louise Bellows, MD, Hampton Bays. Died February 13, 1990; age 94. Dr Bellows was a 1918 graduate of the New York Medical College and Hospital for Women, New York. Her memberships included the International Academy of Proctology, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Lawrence John Biris, MD, New York. Died January 29, 1990; age 42. Dr Biris was a 1979 graduate of the Yale University School of Medicine, New Haven, Connecticut. He was a Diplomate of the American Board of Preventive Medicine, Inc. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Max Bleicher, MD, Hollywood, Florida. Died January 28, 1990; age 75. Dr Bleicher was a 1946 recipient of the Registrable Qualification Granted by the Scottish Conjoint Board, Scotland. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Eben Breed, MD, Garden City. Died March 11, 1990; age 91. Dr Breed was a 1928 graduate of the Cornell University Medical College, New York. He was a Fellow of the American College of Angiology. His memberships included the New York Academy of Medicine, the New York State Society of Internal Medicine, the American Geriatrics Society, the Nassau County Medical Society, and the Medical Society of the State of New York.

Matthew W. Burke, MD, Buffalo. Died March 14, 1990; age 73. Dr Burke was a 1950 graduate of the Georgetown University School of Medicine, Washington DC. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Glenna Garratt Caddy, MD, Carmel, California. Died January 26, 1990; age 92. Dr Caddy was a 1922 graduate of the University of Toronto Faculty of Medicine, Toronto, Ontario, Canada. Her memberships included the American Psychiatric Association, the American Medical Women's Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Carmen Cristo, MD, Rochester. Died February 4, 1990; age 58. Dr Cristo was a 1958 graduate of the State University of New York Upstate College of Medicine, Syracuse, New York. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the New York State Society of Internal Medicine, the American Society of Internal Medicine, the American Geriatrics Society, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Joseph Dalven, MD, Brooklyn. Died February 27, 1990; age 91. Dr Dalven was a 1929 graduate of the New York University School of Medicine. He was a Fellow of the American College of Allergists. His memberships included the American Academy of Allergy, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Charles John Derrico, MD, Bethpage. Died February 28, 1990; age 64. Dr Derrico was a 1954 graduate of the Georgetown University School of Medicine, Washington, DC. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Joseph George Falk, MD, Flushing. Died March 15, 1990; age 85. Dr Falk was a 1934 graduate of the Tufts University School of Medicine, Boston, Massachusetts. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Arnold Fischer, MD, Rockaway, New Jersey. Died September 15, 1989; age 76. Dr Fischer was a 1942 graduate of the Middlesex University School of Medicine, Waltham, Massachusetts. His memberships included the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Julius Gattner, MD, Endicott. Died March 13, 1990; age 97. Dr Gattner was a 1922 graduate of the Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Germany. He was a member of the Broome County Medical Society and the Medical Society of the State of New York.

Frederick Gordon, MD, Lauderhill, Florida. Died February 1, 1990; age 86. Dr Gordon was a 1929 graduate of the Tulane University School of Medicine, New Orleans, Louisiana. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Karl F. G. Hoffmann, MD, Cincinnati, Ohio. Died January 26, 1990; age 92. Dr Hoffman was a 1923 graduate of the Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Germany. His memberships included the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Emma I. Jacoby, MD, Miami Beach, Florida. Died January 14, 1990; age 93. Dr Jacoby was a 1922 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. She was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Edwin P. Kane, MD, Jackson Heights. Died February 10, 1990; age 75. Dr Kane was a 1941 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Medical Society of the

County of Queens and the Medical Society of the State of New York.

Louis Kaywin, MD, Bennington, Vermont. Died March 2, 1990; age 78. Dr Kaywin was a 1941 recipient of the Registrable Qualification Granted by the Scottish Conjoint Board, Scotland. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the Nassau County Medical Society, and the Medical Society of the State of New York.

Dimitrios G. Kotsilimbas, MD, Forest Hills. Died March 17, 1990; age 57. Dr Kotsilimbas was a 1957 graduate of the Faculty of Medicine, Aristotelian National University of Athens, Athens, Greece. He was a Diplomate of the American Board of Neurological Surgery, a Qualified Fellow of the International College of Surgeons, and a Fellow of the American College of Surgeons. His memberships included the New York Neurological Society, the American Geriatrics Society, the American Academy of Neurological Surgery, the New York County Medical Society, and the Medical Society of the State of New York.

Anthony La Russo, MD, Dunwoody, Georgia. Died March 1, 1990; age 85. Dr La Russo was a 1932 graduate of the Hahnemann Medical College of Philadelphia, Philadelphia, Pennsylvania. He was a member of the Broome County Medical Society and the Medical Society of the State of New York.

Alexander Leavitt, MD, Minneapolis, Minnesota. Died January 27, 1990; age 81. Dr Leavitt was a 1934 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Heinz Lichtenstein, MD, Grand Island. Died January 14, 1990; age 86. Dr Lichtenstein was a 1930 graduate of the Medizinische Fakultaet der Universitaet Heidelberg, Heidelberg, Baden-Wurttemberg, Germany. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the New York Academy of Medicine, the American Psychiatric Association, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

James Bernard Loftus, MD, Suffern. Died February 3, 1990; age 65. Dr Loftus was a 1948 graduate of the Jefferson Medical College of Thomas Jefferson University, Philadelphia, Pennsylvania. He was a Diplomate of the American Board of Obstetrics and Gynecology and a Fellow of the American College of Obstetricians and Gynecologists. He was a member of the Medical Society of the County of Rockland and the Medical Society of the State of New York.

Saul Lustig, MD, Rothsville, Pennsylvania. Died March 19, 1990; age 75. Dr Lustig was a 1942 graduate of the Faculte de Medecine de l'Universite de Lausanne, Lausanne, Switzerland. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Maurice M. Maltinsky, MD, Rochester. Died January 25, 1990; age 79. Dr Maltinsky was a 1944 graduate of the State University of New York at Buffalo School of Medicine. He was a Diplomate of the American Board of Family Practice and a Fellow of the American Academy of Family Practice. His memberships included the New York Academy of Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

John C. McCauley, MD, Cleveland, Ohio. Died December 29, 1989; age 88. Dr McCauley was a 1927 graduate of the University of Colorado School of Medicine, Denver, Colorado. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Henry Ernest Morelli, MD, Pelham Manor. Died March 14, 1990; age 79. Dr Morelli was a 1939 graduate of the New York University School of Medicine, New York. His memberships included the New York State Society of Internal Medicine, the Bronx County Medical Society, and the Medical Society of the State of New York.

James William Murphy, MD, Albany. Died February 27, 1990; age 54. Dr Murphy was a 1968 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna, Italy. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. His memberships included the American Thoracic Society, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

Aron Nagler, MD, Brooklyn. Died June 12, 1989; age 81. Dr Nagler was a 1934 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. His memberships included the American Society of Anesthesiologists, Inc, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Irving N. Rattner, MD, New York. Died December 19, 1989; age 90. Dr Rattner was a 1924 graduate of the Tulane University School of Medicine, New Orleans, Louisiana. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

R. Virginia F. Richter, MD, Lancaster. Died January 18, 1990; age 80. Dr Richter was a 1936 graduate of the State University of New York at Buffalo School of Medicine, Buffalo, New York. Her memberships included the New York Academy of Medicine, the American Public Health Association, the American School Health Association, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Edward J. Robinson, MD, Brooklyn. Died February 10, 1990; age 86. Dr Robinson was a 1933 graduate of the Lekarska Fakulta Univerzita Komenskeho, Bratislava, Czechoslovakia. His memberships included the American Society of Internal Medicine, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Benjamin B. Rubinstein, MD, Alexandria, Virginia. Died July 12, 1989; age 84. Dr Rubinstein was a 1936 graduate of the Laaketieteellinen Tiedekunta Helsingin Yliopisto, Helsinki, Finland. His memberships included the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Donald Barton Sanford, MD, Eustis, Florida. Died February 9, 1990; age 84. Dr Sanford was a 1933 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Fellow of the American College of Surgeons. He was a member of the Onondaga County Medical Society and the Medical Society of the State of New York.

Frederick G. Schnabel, MD, Henrietta. Died January 8, 1990; age 85. Dr Schnabel was a 1935 graduate of the Medizinische Fakultaet der Universitaet Erlangen, Erlangen, Bayern, Germany. He was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Arnold Gustav Schwyzer, MD, St Paul, Minnesota. Died January 9, 1990; age 80. Dr Schwyzer was a 1939 graduate of the Johns Hopkins University School of Medicine, Baltimore, Maryland. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Carrol Joslin Shaver, MD, West Seneca. Died February 25, 1990; age 73. Dr Shaver was a 1944 graduate of the State University of New York at Buffalo School of Medicine, Buffalo, New York. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Irving Siegel, MD, Brooklyn. Died January 4, 1990; age 79. Dr Siegel was a 1940 recipient of the Registrable Qualification granted by the Scottish Conjoint Board, Scotland. His memberships included the New York Cardiological Society, the New York State Society of Internal Medicine, the American Society of Internal Medicine, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

David Haskell Spotkov, MD, Boca Raton, Florida. Died February 21, 1990; age 84. Dr Spotkov was a 1930 graduate of the New York University School of Medicine, New York. His memberships included the American Geriatrics Society, the American Society of Abdominal Surgeons, the Bronx County Medical Society, and the Medical Society of the State of New York.

Felix Terner, MD, New York. Died January 25, 1990; age 91. Dr Terner was a 1926 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. His memberships included the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Arnold Joseph Trattler, MD, Great Neck. Died December 1, 1989; age 70. Dr Trattler was a 1943 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Edward C. Veprovsky, MD, Vero Beach, Florida. Died March 2, 1990; age 88. Dr Veprovsky was a 1925 graduate of the New York University School of Medicine, New York. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a Fellow of the American College of Obstetricians and Gynecologists and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the Pan American Medical Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Abraham Isak Weidman, MD, Brooklyn. Died November 26, 1989; age 78. Dr Weidman was a 1939 graduate of the Facolta di Medicina e Chirurgia dell' Universita di Bologna, Bologna, Italy. He was a Diplomate of the American Board of Dermatology, Inc, and a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the New York State Dermatological Society, the American Academy of Dermatology, the Society for Investigative Dermatology, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Hugo Weinberger, MD, Rockaway, New Jersey. Died November 11, 1989; age 90. Dr Weinberger was a 1926 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a Fellow of the American Academy of Family Practice. His memberships included the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Walter T. Heldmann, MD

1904–1990

Walter T. Heldmann, MD, a former president of the Medical Society of the State of New York (MSSNY), died on January 1, 1990, at the age of 85 years. Dr Heldmann, who graduated from Syracuse University School of Medicine in 1928, interned at St Vincent's Hospital in Staten Island. He later did a residency in otolaryngology at the New York Post-Graduate Hospital in 1931–1932. He served for many years as an attending physician at Staten Island Hospital and St Vincent's Hospital and at the time of his death was Director Emeritus of the Speech and Hearing Center, Staten Island Hospital.

Dr Heldmann was a member of MSSNY since 1929 and served as its president from 1969 to 1970. He also served as president of the Richmond County Medical Society from 1946 to 1947 and was president of the First District Branch from 1962 to 1964. In 1984 he was honored by MSSNY with the Henry I. Fineberg Award for Distinguished Service. He was also honored by the American Cancer Society for his more than 35 years of service. A Diplomate of the American Board of Otolaryngology, he was also a Fellow of the American College of Surgeons.

Dr Heldmann is survived by his wife, Evelyn, a son, Walter T., Jr, and three daughters, Marlene Ann Stone, Phyllis K. Georges, and Mary Lynne Heldmann.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1988; 88:456–462). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

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An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

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For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

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REFERENCES

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The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al*. Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

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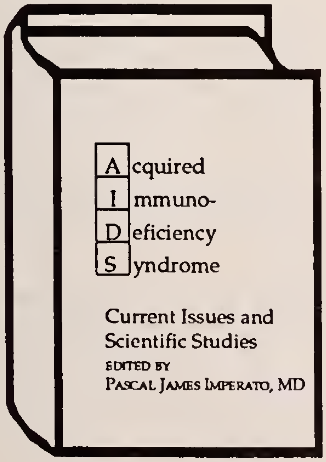

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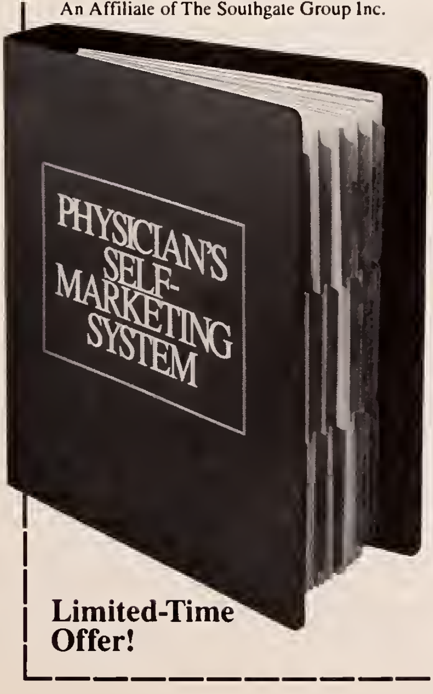

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AROUND THE WORLD

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Aug 22–24. **International Consensus on Supportive Care in Oncology.** Palais des Congres, Brussels. Contact: ICSCO, International Consensus on Supportive Care in Oncology c/o Symedco, Two Research Way, Princeton Forrestal Center, Princeton, NJ 08540. Tel: (800) 821-5678.

BERMUDA

Aug 5–11. **The 10th Annual Comprehensive Fine Needle Biopsy Course.** 25 Cat I Credits. The Princess Hotel, Hamilton. Contact: Extended Programs in Medical Education, School of Medicine, Room 124-C, University of California, San Francisco, CA 94143. Tel: (415) 476-4251.

BRAZIL

Aug 16–18. **1st Pan American Congress on Vascular Surgery.** 22 Cat I Credits. Rio Palace Hotel, Rio De Janeiro. Contact: Advanced Studies in Surgery, 50 South Beach Rd, The Landings #306, South Burlington, VT 05403. Tel: (800) 526-1782.

CANADA

Aug 16–18. **Advanced Ultrasound for the Office Practice: An Emphasis on Transvaginal Scanning and Newer Techniques.** 16 Cat I Credits. Toronto. Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024. Tel: (202) 638-5577. □

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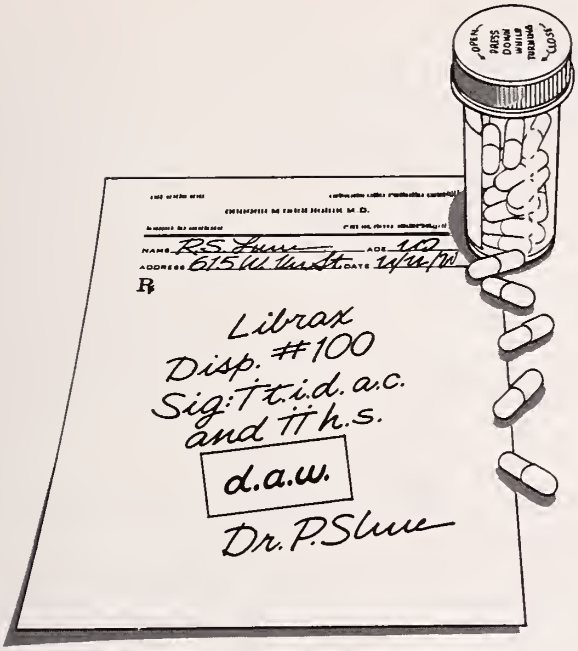

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Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

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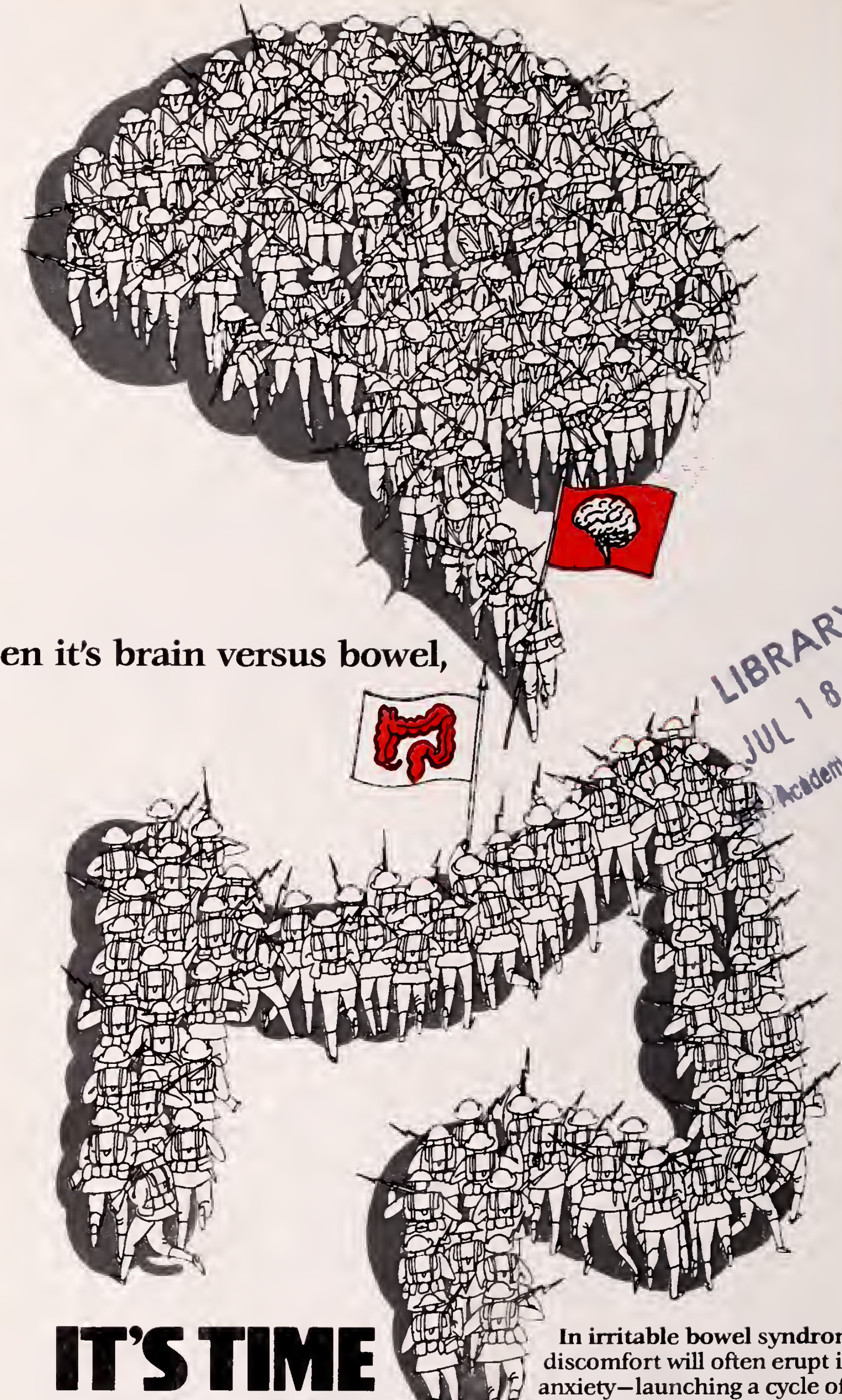

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Please see summary of prescribing information on adjacent page.

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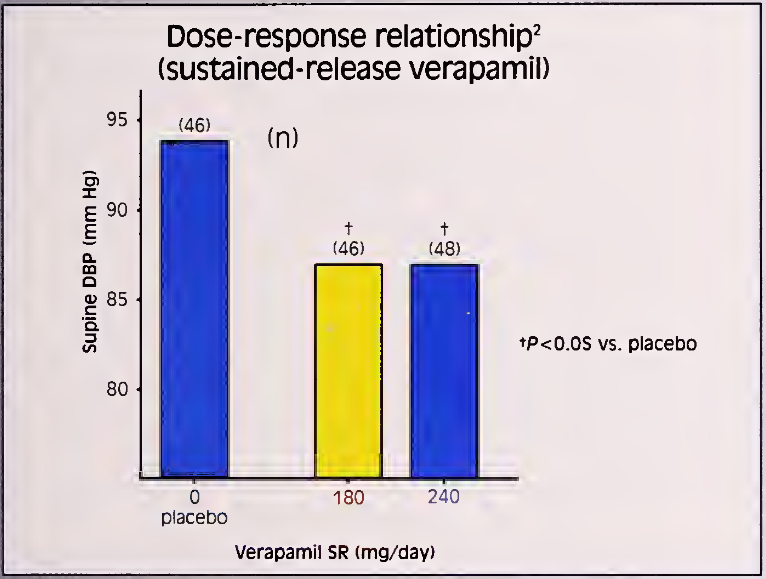

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BRIEF SUMMARY

Contraindications: Severe LV dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block (if no pacemaker is present), atrial flutter/fibrillation with an accessory bypass tract (eg, WPW or LGL syndromes), hypersensitivity to verapamil.

Warnings: Verapamil should be avoided in patients with severe LV dysfunction (eg, ejection fraction < 30%) or moderate to severe symptoms of cardiac failure and in patients with any degree of ventricular dysfunction if they are receiving a beta-blocker. Control milder heart failure with optimum digitalization and/or diuretics before Calan SR is used. Verapamil may occasionally produce hypotension. Elevations of liver enzymes have been reported. Several cases have been demonstrated to be produced by verapamil. Periodic monitoring of liver function in patients on verapamil is prudent. Some patients with paroxysmal and/or chronic atrial flutter/fibrillation and an accessory AV pathway (eg, WPW or LGL syndromes) have developed an increased antegrade conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving I.V. verapamil (or digitalis). Because of this risk, oral verapamil is contraindicated in such patients. AV block may occur (2nd- and 3rd-degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with verapamil.

Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol clearance may occur with combined use. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digitoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration. Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels or increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.

Adverse Reactions: Constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF, pulmonary edema (1.8%), fatigue (1.7%), dyspnea (1.4%), bradycardia: HR < 50/min (1.4%), AV block: total 1°,2°,3° (1.2%), 2° and 3° (0.8%), rash (1.2%), flushing (0.6%), elevated liver enzymes. The following reactions, reported in 1.0% or less of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitations, purpura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis or bruising, cerebrovascular accident, confusion, equilibrium disorders, insomnia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, arthralgia and rash, exanthema, hair loss, hyperkeratosis, macules, sweating, urticaria, Stevens-Johnson syndrome, erythema multiforme, blurred vision, gynecomastia, increased urination, spotty menstruation, impotence.

References:

1. 1988 Joint National Committee: The 1988 report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. *Arch Intern Med* 1988;148:1023-1038.
2. Data on file, G.D. Searle & Co.

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BRONX

Sept 26. **Forensic Aspects of Fire Investigation.** Lehman College campus. Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 511 Hempstead Ave, Suite 2, West Hempstead, NY 11552. Tel: (516) 292-2300.

COOPERSTOWN

Sept 12. **Geriatrics.** 3 Cat I Credits. Bassett Hall Conference Center. Contact: Charlotte L. Hoag, Medical Education Coordinator, Office of Medical Education, The Mary Imogene Bassett Hospital, One Atwell Rd, Cooperstown, NY 13326. Tel: (607) 547-3926.

MANHATTAN

Sept 5–Dec 21. **Basic Science Course in Ophthalmology.** 154 Cat I Credits. Bellevue Hospital, New York City. *Sept 15.* **Seminar on Parkinson's Disease & Related Movement.** 7 Cat I Credits. NYU Medical Center. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016.

Sept 6–7. **10th Annual Cosmetic Dermatology Symposium: New Developments in Topical & Surgical Therapies.** Podell Auditorium, Beth Israel Medical Center. Contact: Beverley R. Baptiste, Medical Education Office, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003. Tel: (212) 420-2000.

AROUND THE NATION

CALIFORNIA

Sept 6–8. **Sixth Annual Sonography Update Conference & Exhibit.** 18¼ Cat I Credits. *Sept 7–10.* **Sixth Annual Sonography Update for Physicians and Sonographers.** 18 Cat I Credits. *Sept 8–9.* **Sonography Registry Review.** 16 Cat I Credits. Hotel del Coronado, Coronado (San Diego). Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

Sept 14–16. **Uveitis and Retina Frontiers.** 12 Cat I Credits. Carmel Valley Ranch Resort, Carmel Valley. *Sept 17–18.* **Blepharoplasty Workshop: Principles and Technique.** 11 Cat I Credits. University of California, San Francisco. Contact: Extended Programs in Medical Education, University of California, San Francisco, Room C-124, San Francisco, CA 94143. Tel: (415) 476-4251.

FLORIDA

Sept 12–15. **Third Annual Applied Ultrasound: Accent on Doppler.** 18 Cat I Credits. Ritz Carlton Resort Hotel, Naples. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

LOUISIANA

Sept 21–22. **Pathology Update.** 7½ Cat I Credits. Westin Canal Place Hotel, New Orleans. Contact: Linda T. Reeves, Program Coordinator, Office of Continuing Education, Tulane Medical Center, 1430 Tulane Ave, New Orleans, LA 70112. Tel: (504) 588-5466.

NEBRASKA

Sept 24–29. **Emergency Medicine Review.** Lincoln. Contact: Continuing Education, University of Nebraska Medical Center, 42nd and Dewey Ave, Omaha, NE 68105. Tel: (402) 559-4152.

PENNSYLVANIA

Sept 15. **6th Special Regional Symposium on the Modern Treatment of Varicose Veins.** Sheraton Society Hill, Philadelphia, Pennsylvania. Contact: North American Society of Phlebology, 204 North El Camino Real #E234, Encinitas, CA 92024. Tel: (619) 942-8380.

(*continued on p 16A*)

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New York State Journal of Medicine

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Contents

COMMENTARIES

393 **Life, liberty, and the pursuit of happiness**
JOHN T. FLYNN, MD

394 **Organ donation**
FREDERICK K. MERKEL, MD

RESEARCH PAPERS

396 **The pattern of organ donation in a large urban center**
NABIL SUMRANI, MD; VERA DELANEY, MD; KHALID M. H. BUTT, MD; JOON H. HONG, MD

REVIEW ARTICLE

400 **A history of infectious waste legislation and regulation and their impact on hospitals in New York City**
MICHAEL T. TRONCONE, MPH

HISTORY

406 **A history of the Ray Brook State Tuberculosis Hospital**
KENNETH W. WRIGHT, MD; JAMES MONROE, MD; FREDERICK BECK, MD

SPECIAL REPORT

414 **Magazines that do not accept tobacco advertising**

CASE REPORTS

415 **Infected aortitis masquerading as bronchogenic carcinoma**
LINDA J. PARADOWSKI, MD; IVAN HAJDU, MD, PHD; LORRAINE COLI, MD; GREGORY LOEWEN, DO

(continued on p. 11A)

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Carcinogenesis, Mutagenesis, Impairment of Fertility—A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of varying durations included almost 5,000 patients. Among the more common adverse events in domestic placebo-controlled trials of over 1,900 nizatidine patients and over 1,300 on placebo, sweating (1% vs 0.2%), urticaria (0.5% vs <0.01%), and somnolence (2.4% vs 1.3%) were significantly more common with nizatidine. It was not possible to determine whether a variety of less common events was due to the drug.

Hepatic—Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to three times the upper limit of normal, however, did not significantly differ from that in placebo patients. Hepatitis and jaundice have been reported. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS—Rare cases of reversible mental confusion have been reported.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with equal frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic—Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental—Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity—As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Because cross-sensitivity among this class has been observed, H_2-receptor antagonists should not be administered to those with a history of hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance by approximately 84%.

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New York State
Journal of Medicine

CONTENTS, CONT'D

416 **Total pancreatectomy with celiac artery occlusion**
NOLAN KARP, MD; PATRICK J. LAMPARELLO, MD;
JOHN H. C. RANSON, MA, BM, BCH

418 **Spontaneous renal hemorrhage secondary to choriocarcinoma**
ROBERT WASHECKA, MD; WILLIAM MANN, MD; ROSEMARIE ZUNA, MD

LETTERS TO THE EDITOR

420 **The need for medical libraries in hospitals**
LILLIAN BURKE, MD

421 **Impotence: A single symptom with different causes**
JOHN F. O'CONNOR, MD, MONICA J. SALERNO, PHD

422 **Melville's "Bartleby the Scrivener"**
MILTON TARLAU, MD
JEROME M. SCHNECK, MD
EDWARD H. ROSENBERRY, PHD

424 **The physician as lobbyist**
GARY P. ANDELORA, ROBIN M. PELLEGRINO

426 LEADS FROM EPIDEMIOLOGY NOTES

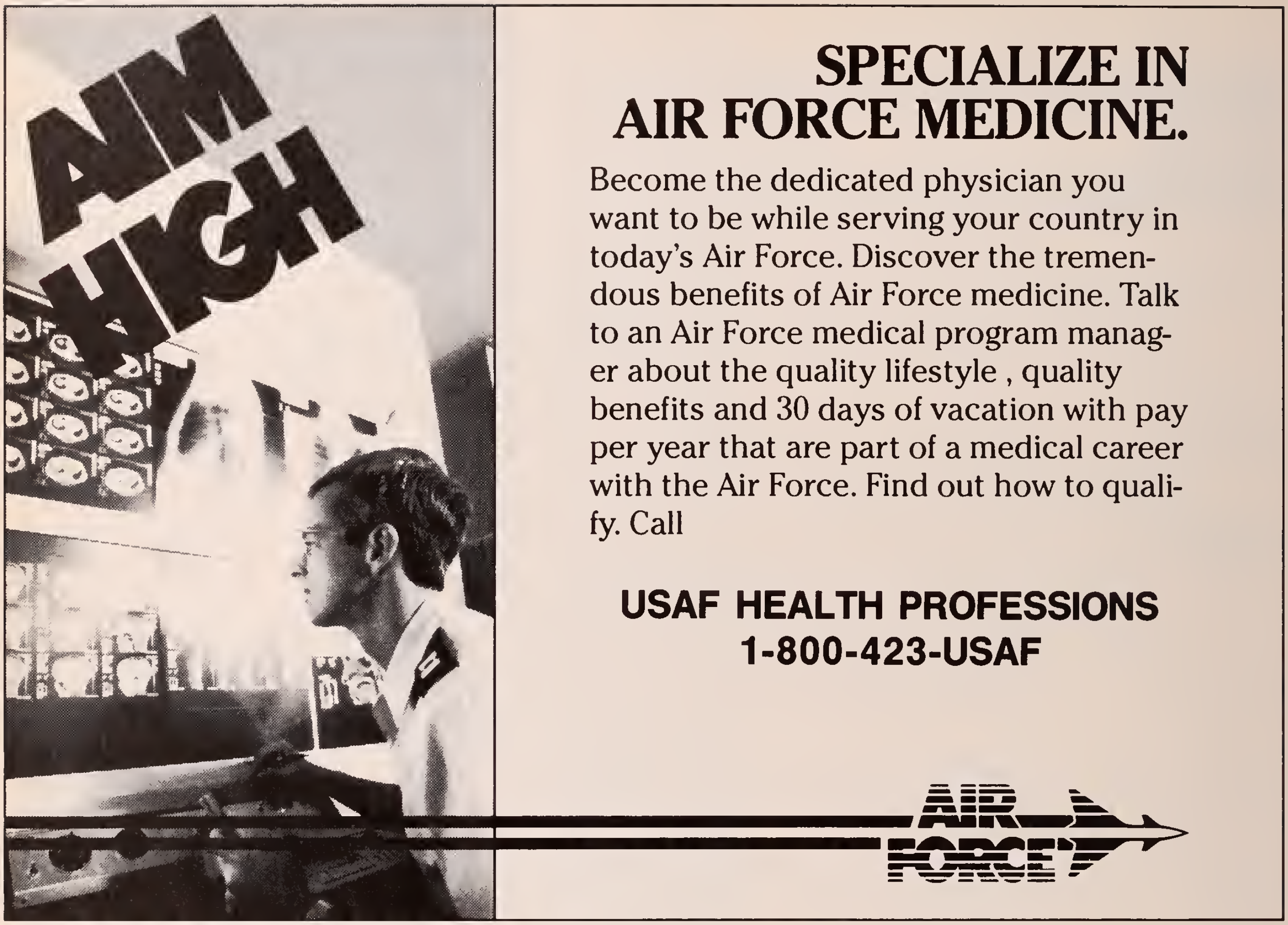
428 BOOK REVIEWS

432 NEWS BRIEFS

434 OBITUARIES

438 GUIDELINES FOR AUTHORS

6A MEDICAL MEETINGS AND LECTURES



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COMMENTARIES

Life, liberty, and the pursuit of happiness

> "We hold these truths to be self-evident, that all men . . . are endowed by their Creator with certain unalienable Rights, that among these are Life, Liberty and the pursuit of Happiness.—That to secure these rights, Governments are instituted among Men . . . "[1]

During the two centuries since Thomas Jefferson composed these words, life and liberty have been the major foci of social concern in Euro-American societies. Inspired by the Enlightenment, from Hutcheson and Montesquieu to Rousseau and later to Bentham, societies have slowly reshaped civil laws and mores, to safeguard the personal rights and liberties of individuals. In addition, during these two hundred years, civilized societies have encouraged the scientific advancements which have helped to improve human life through better functioning of the human body during an extended life span.

In the 20th century, these societies have reached a state in which the third of these inalienable rights, the pursuit of happiness, has gradually moved closer to center stage on a broad scale. The pursuit of happiness is the driving force behind a countless assembly of human activities, including the ownership of one's own home, the steady stream of fads and fashions, personal grooming, social events, vacationing, hobbies, the arts, games, sports, and sexual activities, to mention but a handful. The latter half of this century has witnessed a host of effects, both good and ill, which have arisen from broader opportunities in affluent societies for the individual to pursue happiness in his or her own fashion.

When the free and sometimes misguided pursuit of happiness suffers distortions or is inappropriately directed toward the relief of unhappiness, ill effects can result and now present a major challenge to governments and to the social order, placing an especially heavy burden upon both the health care system and the criminal justice system.

The pursuit of sexual pleasure can generate heterosexual and homosexual promiscuity, prostitution, sexual abuse, and the transmission of disease, including AIDS. Permissiveness toward sexual experience has encouraged teenage pregnancy and in turn its control, in part, by legalized abortion. The pleasures derived from food can proceed toward gluttony, obesity, and an increase in the hazards of ill health. The social pleasures derived from alcohol can escalate to chronic habits of alcohol abuse, with its multitude of disastrous complications to self and others. The enjoyment of tobacco can lead to an addiction, with often costly damage to health. Recreational use of chemical substances, centuries old, and their application to the relief of states of depression or unhappiness, have precipitated major social crises, national and international, and have further strengthened organized crime.

Other activities intended to procure happiness can lead to regrettable consequences. Betting can progress to a compulsion, with serious personal, family, social, and financial consequences. A natural and healthy interest in sports may evolve into a frantic insistence on winning at any cost—an urge which can lead to the unwise use of performance enhancing drugs, in addition to dishonesty on the playing field and illicit gambling. The urge to accumulate material goods as a route to happiness is too often reinforced by affluence in a society, abetted by a cornucopia of goods available for the satisfaction of every whim and taste, and fostered by media promotions which insist that more is better and happier.

The pursuit of individual happiness appears to be most strongly influenced by the entertainment industry, the manufacturers of commercial products and their advertising agencies, and, regrettably, by organized crime through the control of prostitution, gambling, loan sharking, and illicit drugs.

The public happiness factor absorbs the attention of elected officials—a proper awareness on their part. Too often, however, it can deter such officials from generating increased taxes for social necessities, and can interfere with needed governmental reforms or reallocation of resources or the provision of seriously needed government services.

Thus the ill effects of the pursuit of happiness impact upon many segments of an organized society.

Confronted with these difficulties, governments have chosen in many instances to exert coercive force to forestall the ill effects of the pursuit of happiness through restrictive and prohibitive responses by lawmakers and the criminal justice system. Their successes have been limited and marred by a spectrum of failures, the signal instance of which was the attempt to ban the manufacture, sale, and distribution of intoxicating beverages by our own 18th Amendment to the Constitution. Today, governments are still groping toward more positive steps in the appropriate support of individual happiness.

It may appear unusual, certainly unexpected, that Jefferson should have selected the pursuit of happiness for inclusion in the Declaration of Independence, instead of a number of other inalienable human rights, and thus involved governments in the protection of such a pursuit. One can argue that Jefferson was wrong in believing that governments should give more than peripheral attention to the individual's pursuit of happiness. Yet governments are expected by their constituents to give aid and to protect against the ill effects of the pursuit of happiness. When government fails, it is blamed and held accountable.

In fairness, how can one criticize a government's failure to deal effectively with such problems, when citizen and government have not begun to reach a consensus regarding their mutual responsibilities in these matters?

Jefferson left such a dialogue to later generations, but in the Declaration of Independence he committed our government, and indeed all governments, "To secure these rights." He could not have intended that governments assume the impossible task of determining the proper pursuit of happiness for each individual citizen. Nor could he have expected that governments would ensure the individual's right to pursue happiness, without regard to the consequences to self and to others, and no matter what the cost to society at large.

Major questions remain unanswered. Should government coopt and control some aspects of the pursuit of happiness, as has occurred with the diversion of gambling moneys into the public coffers? On the other hand, is the role of government in these matters more properly an educational one? To what extent should government and society offer assistance to those whose pursuits of happiness have contributed to destructive outcomes, both to self and to others?

Poorly structured family life, inadequate educational systems, unrealistic individual expectations, and immoderation in personal habits and activities can combine to thwart governmental efforts to deal effectively with the unfortunate consequences of the individual's pursuit of happiness.

A clear set of agreements, resulting from a dialogue between the people and their elected representatives, requires clearer public understanding of the individual's responsibility to self and others in his or her pursuit of happiness.

Such a clearer understanding requires carefully structured and faithfully completed research studies, free of rhetorical biases, in certain crucial areas. These should include the influence upon personal self-esteem and satisfaction of the individual's early family life, educational experiences, workplace, career satisfactions, marital family, and child-bearing and child-rearing experiences—the basic ingredients of most human lives and experiences.

We must pursue research into the neurophysiologic and pathologic bases of mental depression, which often stimulates the individual's recourse to activities and agents which can offer only momentary relief from depression and unhappiness. We need to understand more completely the influences that commit certain individuals to addictions that are patently self-destructive, as well as those conditions which create and perpetuate social despair.

These serious problems will not be resolved in our societies without extensive contributions to an effective public dialogue along these lines.

All of us must contribute.

JOHN T. FLYNN, MD
Consulting Editor

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1. In Congress, July 4, 1776, A Declaration by the Representatives of the United States of America, in General Congress assembled. Williamsburg. *The Virginia Gazette*, July 26, 1776, No. 78, p 1.

Organ donation

Organ transplantation is now a major therapy for the treatment of a wide range of human ills. Impressive advances in transplantation have occurred so rapidly during recent years that now almost every organ can be transplanted with some degree of success. Transplants are performed in university and community hospitals worldwide and even in Third World countries.

Cyclosporin A, the "wonder drug" of the 1980s, has so increased the success rate with transplants of the heart, liver, and kidneys that the availability of organs has not kept pace with need. Pharmacologic immunosuppression with newer drugs such as FK 506 will result in equally impressive improvement in results so that the demand for organs will be even greater. Since this organ deficit affects patients, physicians must try to discover the reasons for the lack of needed donors and find remedies. Because so many patients have received transplants, a large number of physicians have had some interest or patient care experience in this field.

The reasons for the gap between need and supply are numerous and varied. There may be different reasons for a lack of donors among various population groups. Indifference, lack of knowledge, suspicion of the ethics or motives of others, and religious deterrents may affect groups differently. Physicians caring for potential donors may not wish to become involved in the process for legal and psychological reasons, or because of discomfort in approaching the family. Further, physicians may wish to avoid the extra time commitments involved in the donor process, and the personnel of the donor hospital may be insufficiently prepared for approaching the family with a request for organs. Although there are probably enough potential donors available to provide the necessary organs, for a number of reasons, some poorly understood, only a small percentage of these donors are obtained in any racial group as shown by Sumrani et al in this issue of the *Journal.*[1] The authors' figures demonstrate that the rate of organ donation by the black and Asian populations in New York continues to be poor (24% and 21%, respectively), the rate of donation by whites has remained at a steady but unspectacular level (42%), and the only population group whose donation rate has improved has been the Hispanics (17% to 47%). Nevertheless, it is encouraging that Hispanic donation has improved and perhaps lessons learned from this group can be applied to the others.

The consolidation caused by the recent federal regulations eliminating competition has probably had a negative effect on the growth of donor programs. I personally believe that competition is an essential ingredient of the private practice of medicine and its elimination from organ retrieval is an unfortunate precedent for our system of medical care. The law also places the responsibility for donor requests on the donor hospitals rather than working through donor teams. This can result in poorly trained or disinterested personnel making the requests, hence undermining the objective of the legislation.

Clearly, these recent events have had a serious negative effect on the success rate of organ retrieval. In my opinion, and as clearly stated in the article by Sumrani et al,[1] education is the key to success. Above all, one must educate professional colleagues. Transplantation should be an important part of nursing and medical school curriculae and should be part of board and specialty examinations. Medical and nursing schools should not only teach students about transplants, but should also teach them how to speak with the families of the deceased in requesting donations. Articles such as the one by Sumrani et al[1] should regularly appear in medical journals as they serve an important educative function. As noted by the authors, the media should be educated as well, and then enlisted to educate the general public and public officials. Finally, patients should be educated as to how to obtain transplantation when needed and to lessen fears and concerns regarding both transplants and organ donation. The general public must also be enlisted as a major participant in this type of program. By these means, I am convinced that we will be able to care for the rapidly growing population requiring transplants.

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[1] Sumrani N, Delaney V, Butt KM, et al: The pattern of organ donation in a large urban center. *NY State J Med* 1990; 90:396–399.

The pattern of organ donation in a large urban center

Nabil Sumrani, md; Vera Delaney, md; Khalid M. Butt, md; Joon H. Hong, md

ABSTRACT. Despite increasing referrals for organ donation in metropolitan New York, procurement has remained essentially unchanged from 1983 through 1988 at 9 to 13 per million population, falling far short of increasing demand. This is not due to delay in the diagnosis of brain death, higher discard rates, or increased medical unsuitability, although exclusion because of human immunodeficiency virus (HIV) disease, or risk thereof, has increased and now accounts for 38% of exclusions. Consent for organ donation remains consistently low among blacks (24%), has increased among Hispanics from 17% in 1984 to 47% in 1988, and remains the highest, but without improvement, among whites. Causes for the observed stagnation and potential corrective factors include poorly focused educational efforts, lack of sensitivity to the grieving family by hospital personnel, physician frustration at the increasingly prominent role of government and its regulations in the practice of medicine, and eradication of competition between local organ procurement agencies.

(NY State J Med 1990; 90:000-000)

Major progress in immunosuppressive therapies, surgical techniques, and management of complications during the past 30 years has led to a new era in solid organ transplantation. Currently, one-year patient and graft survival rates approach 95% and 85%, respectively, for primary cadaveric renal transplants; 88% and 55%, respectively, for pancreas transplants; 80% for heart transplants, and 60% for liver transplants.[1] These improving statistics have led to a massive increase in the demand for organs. Data obtained from the United Network for Organ Sharing show that as of July 31, 1989, there were 15,271 patients in the United States awaiting renal transplantation, 1,199 awaiting cardiac transplantation, and 758 and 273 awaiting liver and pancreas transplants, respectively. Yet the number of kidneys procured in the United States has levelled off or decreased slightly from 1987 through the early part of 1989.[2] Speculation that only 15% to 20% of suitable organ donors are employed led to the introduction over the past five years of several federal and state laws aimed at promoting donation and effective utilization of organs.

In an attempt to assess the effect of these regulations on the pattern of organ donation in metropolitan New York, we reviewed our experience with organ procurement from 1983, the year cyclosporine was introduced into general use, through 1988.

Methods

Details of solid organ donor referrals and organ procurement were obtained from records derived from 1983 through April 1988 from the local organ procurement agency, named the "Gift of Life Program." This agency is located in metropolitan New York and serves a population of three million, with referrals from 26 hospitals. In April 1988, in response to federal demands for consolidation of organ procurement organizations, this agency became part of the Regional Transplant Procurement Program (RTP) which serves the entire metropolitan New York area with a population of 13 million and 186 hospitals. The procurement area serviced by our transplant team, however, diminished with this reorganization to 2.5 million and 23 hospitals. Donor referral and organ procurement data for the period April–December 1988 were obtained from the RTP and included in the 1988 data.

Donor screening and preliminary discussion with the donor family were performed, for the most part, by transplant coordinators, specialists trained in all aspects of organ procurement. The Required Request Law was implemented in January 1986. This law requires hospitals to request consent for organ/tissue donation from family members if the deceased is medically suitable. Medical suitability was determined by one of two transplant surgeons and was based on donor age, life style, concomitant systemic disease, absence of organ dysfunction, and lack of evidence of any transmissible disease. Brain death was established by the presence of coma and absence of brain stem reflexes without intoxication, primary hypothermia, metabolic or endocrine disturbances, or documentation of apnea, and/or electrocerebral silence by one or more electroencephalograms (EEG) and/or absence of cerebral perfusion by angiography. Individual neurologists, in conjunction with hospital policies, dictated which of these objective tests were used. The time period required to establish brain death ranged from 12 hours to seven days.

Results

Referrals of potential donors increased from 1983 to 1987, peaking at 180 in 1987. Irrespective of the number of referrals, the actual number of utilized donors remained relatively constant at 28–39 per year, or 9 to 13 per million population, with discard rates of 7–16% (Fig 1). Prospective donors excluded because of medical reasons remained approximately the same over the years of the study at 7–23% of referrals (Fig 2). Sepsis was the most common reason for exclusion, but HIV disease, carrier state, or life style associated with the risk of viral infection assumed greater importance in the last three years of the study, accounting for as many as 38% of exclusions in 1988. Other reasons for exclusion were donor age (donors less than two years old were not used throughout the entire study period) and renal disease, defined as an abnormally low creatinine clearance rate, often in association with abnormal urinary findings or anatomical defects of the urinary tract.

Potential donors who died prior to completion of a brain death work-up exceeded those whose organs were utilized in the first half of the study, with reversal of this trend in 1986 through 1988 (Fig 3). The major cause of death in prospective donors whose families consented to organ donation was intracranial hemorrhage, either spontaneous or traumatic (Fig 4), with no significant change over the

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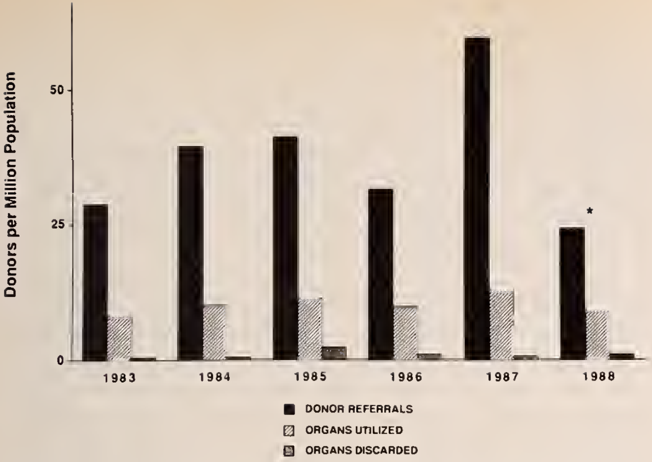

FIGURE 1. Donor referrals and organs procured, Gift of Life Program, New York City, 1983–1988 (smaller referral base; see methods).

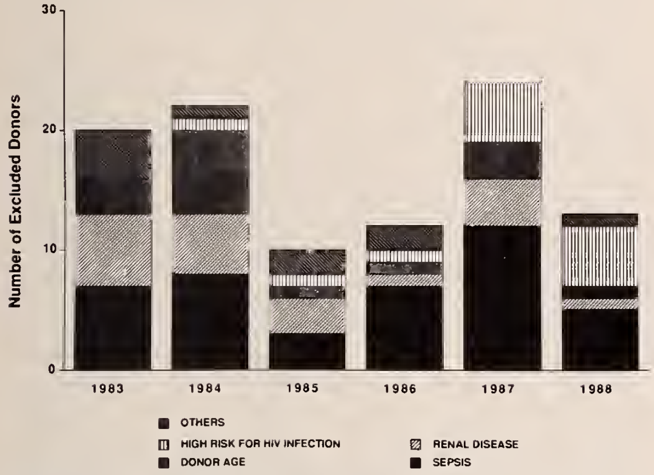

FIGURE 2. Causes of medical unsuitability for organ procurement, Gift of Life Program, New York City, 1983–1988.

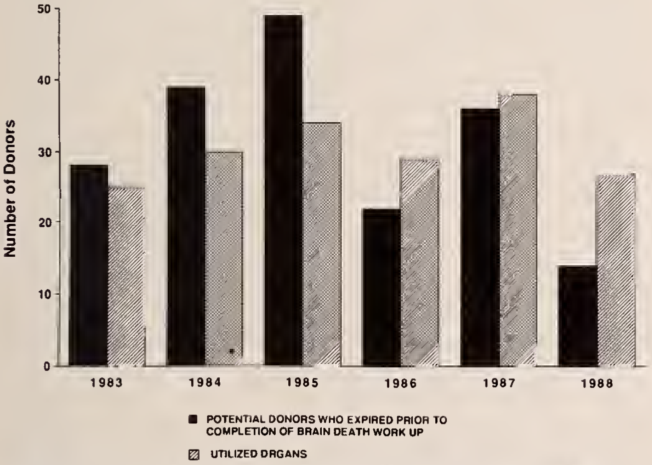

FIGURE 3. Comparison of numbers of potential donors who died prior to diagnosis of brain death with those utilized, Gift of Life Program, New York City, 1983–1988.

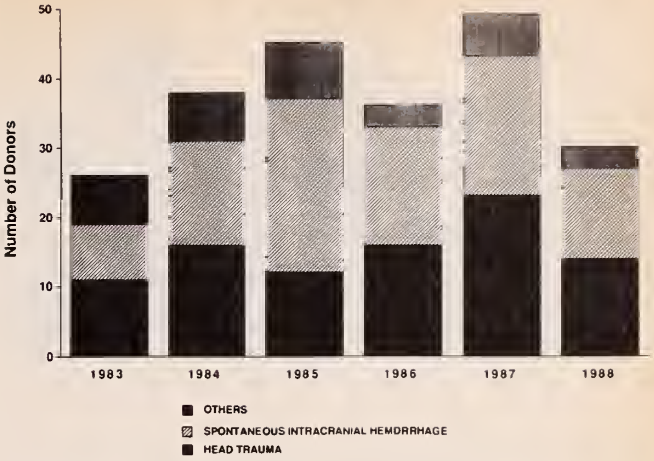

FIGURE 4. Causes of brain death among organ donors, Gift of Life Program, New York City, 1983–1988.

course of the study, but some variability in the approximately equal distribution within these two subcategories.

Whites remain the largest donor referral group, with about 50% consistently consenting to organ donation, compared with approximately 30% of blacks. In contrast, the percentage of Hispanics agreeing to organ donation increased from 17% in 1984 to 47% in 1988 (Fig 5). The Asian subgroup was too small for analysis. Utilized organs were predominantly from whites (50–75%), with an increase in the relative black and Hispanic contributions over the last two years (Fig 6). Cumulative data for the entire study period showed that, of the referrals received, 42% of whites consented to organ donation compared with 32% of Hispanics, 24% of blacks, and 21% of Asians (Table I).

DISCUSSION

Referrals for organ donation increased following passage of the Required Request Law in 1986, yet the number of actual donors has remained unchanged despite increasing demand (Fig 1). For example, 18 kidneys per million population were procured in New York City in 1988, far removed from the annual incidence of end-stage renal disease, which now approaches 150 patients per million. This unfortunately reflects the general trend in the United States over the last three years,[2] which is in contrast to Norway and Denmark, where yearly kidney procurement keeps pace with demand at 50 per million population.[3]

Progress has apparently been made in the rate of procurement of organs from patients dying of cardiovascular

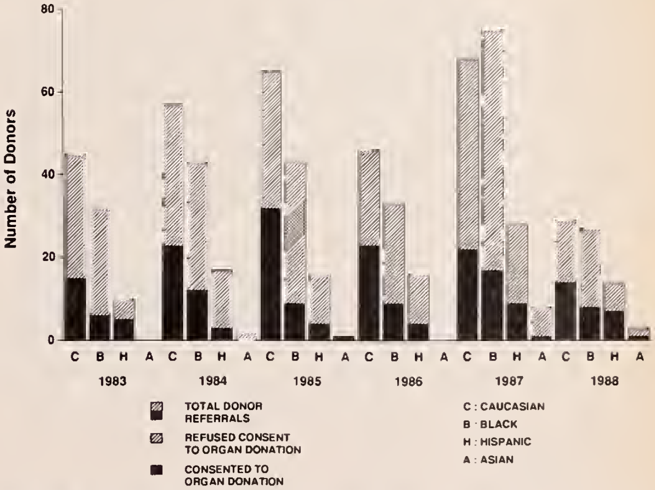

FIGURE 5. Influence of race on organ donation, Gift of Life Program, New York City, 1983–1988.

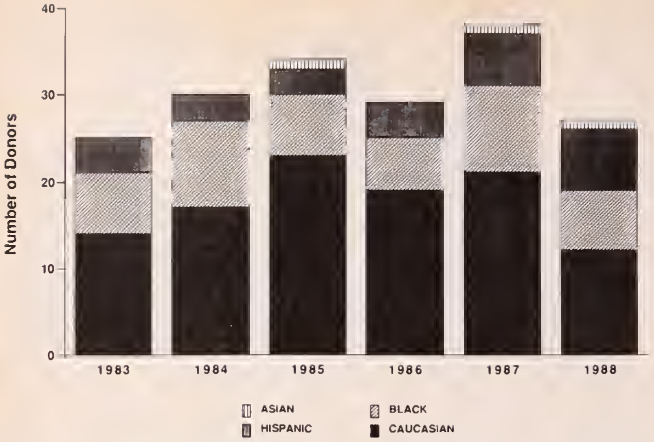

FIGURE 6. Racial distribution of procured donors, Gift of Life Program, New York City, 1983–1988.

or respiratory complications prior to declaration of brain death (Fig 3). This increase may be attributable to recent state laws (in 36 states) and local, hospital-based protocols that simplify and clarify the criteria necessary for establishment of brain death.[4] In seven states, including New York, the brain death standard was adopted by judicial decision. In the case of New York, a task force was set up specifically to examine and clarify the legal criteria to be used for the determination of death. Its report, entitled "The Determination of Death," was published in July 1986.[4]

Exclusion of prospective donors because of medical unsuitability has remained relatively stable over the past five years. The increasing contribution of HIV disease to medical unsuitability is apparent in this study (Fig 2) and reflects the inner city population with its large percentage of high risk groups.[3] Obviously, further attempts at increasing organ donation at the risk of harm to the potential recipient is untenable.

More stringently enforced seat belt and drunken driving laws in recent years may have diminished the number of donors who die as a result of head trauma, an effect masked, perhaps, by the increasing number of "gun-shot wounds to the head," also included in this category (Fig 4). This reflects the escalating violence in the inner cities.

Whites are more likely to consent to donation than is any other racial group (Fig 5), with Hispanics being the only subgroup to show improving trends in organ donation (Fig 6). This may be attributed to the willingness of Hispanics (70%) to discuss disposal of organs after death with their families. This number is in sharp contrast to the 25% figure among the black population, as shown by opinion surveys undertaken by the Gallup organization in 1984, 1986, and 1987. Lack of knowledge about transplantation, donation, and the need for kidneys among blacks in view of their higher incidence of renal failure, as well as religious concerns that the body must be kept intact for life after death, insecurity about the diagnosis of brain death, and distrust of medical personnel[5] are other causes contributing to the observed stagnation in organ donation rates among blacks.

TABLE I. Percentage of Referrals who Consented to Organ Donation According to Race, 1983–1988, Gift of Life Program, New York City

Race	Referrals	Percentage Consented
White	310	42%
Black	253	24%
Hispanic	101	32%
Asian	16	21%

According to the Task Force on Organ Transplantation, which submitted its report to Congress in August 1987, educational efforts have thus far been poorly focused and essentially ineffective. There has been no discernible change in the percentage (27%–30%) of Americans who consider themselves likely to donate during the years 1985 through 1987,[5] despite increased funding by both public and private concerns. Lack of specific, operationally defined goals and objectives, and the absence of efforts to evaluate the effectiveness of education programs, were serious shortcomings cited by the Task Force on Organ Transplantation in 1986.[5]

Corrective measures and increased funding have now been initiated, with more intensive and repeated exposure of the public to the concept of organ donation by use of the media, particularly television. Specific attention must be given to the fact that although 94% of Americans have heard about organ and tissue transplants, only 20% carry donor cards, and of the latter group, approximately half did not know that family consent is usually necessary. Prior discussion with family members as to the desire to donate is known to facilitate the subsequent decision by the grieving family[6] and should be encouraged. Education of religious teachers as to the benefits of transplantation with modification, if possible, of ancient, deeply held traditions would undoubtedly aid organ procurement. This has been achieved in some Muslim countries, where cadaveric transplantation is now taking place despite initial outrage at the concept.[7] Jewish religious leaders are still, unfortunately, reticent to accept the concept of brain death, essentially precluding organ retrieval, as evident from the proceedings of the 1985–1986 New York State Task Force on Life and Law, at which the determination of death was discussed in detail.[4]

Fostering a more sensitive approach to the bereaved family by requesting personnel, be it physician, surgeon, nurse, coordinator, or administrator, is imperative. Attending physicians are in a unique position to obtain permission for organ donation, having developed a close and often trusting relationship with family members throughout their ordeal. Unfortunately, these same physicians often do not feel that their duty extends beyond preservation of the life of the deceased. With the expansion of transplant programs throughout the US, most young doctors now gain experience in the field as part of their residency training and learn of the wonderful new leases on life extended to patients by organ donation. This added advantage may, unfortunately, be offset by the mounting frustration of doctors with the relentless increase in regulations imposed on them by federal, state, and other agencies.[8] The Required Request Law may become just another form to get signed quickly with as little explanation as possible. In this regard, transplant coordinators are, perhaps, better equipped at requesting organ donation. Unfortunately, state and federal regulations have also altered organ procurement by amalgamation of local programs into regional centers, with the consequent removal of competition and incentives, both of which may be essential to further growth of organ procurement.[9]

In conclusion, organ procurement within the United States, and particularly in New York City, is falling far short of demand. Goal oriented public education, designed to reach all socioeconomic groups, races, and religious creeds, is necessary for further growth. Additionally, physicians and hospital personnel must develop the right mix of compassion and enthusiasm necessary to encourage organ donation from grieving families.

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THE MEDICAL EDUCATION OF THE FUTURE

...The progress of preventive medicine has imposed on physicians a new class of duties for the discharge of which a high degree of disciplined intelligence is required. They are the only persons in the community who can thoroughly understand and explain the established principles and well-proved practices of preventive medicine; and they are, therefore, the most effective teachers of these principles and practices. The family physician should be responsible for the care of health even more than for the treatment of disease. It should be his function to give advice about the ways and means of healthy family life—about diet, sleep, fresh air, exercise, and habits of quietness and serene cheerfulness. Physicians must instruct the community in the new methods by which good public water-supplies are provided, tested, and preserved; and they must be equally familiar with the right methods of disposing of sewage, for the disposal of sewage is really a problem of pure water-supply. They must understand the restoration of polluted waters to a safe condition, through filtration, aëration, and dilution. Their judgment should be the final one in families concerning the safety of any given water-supply, and that judgment should be well founded on a general acquaintance with the subject and on all relevant local information. Physicians should also understand the general principles and most approved practices in ventilation; for ventilation is not only a means of promoting health, but also a means of defense, through dilution, against contagion and other noxious influences. Now, ventilation in both public and private buildings is in itself a very difficult subject, and one but recently developed in a practical way. As the mechanical construction of our buildings improves, they become tighter, and as heating contrivances become more economical as regards the proportion of utilized heat to wasted heat, they become less valuable as means of ventilation. The roaring fire in the wide chimney of our grandfathers, made a great draught; the quiet but effectual coal-stove is an inferior means of ventilation. The more indoor the life of the population, the more important ventilation becomes to the public health. Who but the physician and surgeon can teach disinfection and cleanliness in the treatment of contagious diseases, or impress the population with the need of separating healthy children or adults from those afflicted with tuberculosis or other chronic contagion? Who else is to object to damp cellars filled with organic rubbish, to bad cooking and ill-chosen diet, and to all the manifold interior decorations with which houses are made more unsanitary? Who else can instruct the community in school hygiene, in the imperative need of thorough cleanliness throughout school buildings, of effective ventilation, of good privies, clean books, strong light, and furniture adapted to the sizes of the pupils? Who else is to teach inexperienced mothers that nothing but the most painstaking cleanliness can prevent the nursing-bottle from becoming a regular culture-apparatus for micro-organisms?

There is an infinite amount of teaching to be done in regard to all these subjects, and the medical profession are in many communities the only available teachers. In order to teach effectively, the profession needs to be better trained than it now is in the ordinary methods of influence—trained, that is, to a better power of persuasive writing and speaking, and to the habitual exercise of that authority which should accompany recognized knowledge and disinterestedness. . . .

CHARLES W. ELIOT, LLD
(*Trans Med Soc State of NY* 1896; 87-104)

A history of infectious waste legislation and regulation and their impact on hospitals in New York City

MICHAEL T. TRONCONE, MPH

The Medical Waste Tracking Act of 1988 has mandated the federal Environmental Protection Agency (EPA) to establish a pilot program for medical waste tracking. Participants in the program are New York, New Jersey, Connecticut, Rhode Island, and Puerto Rico. The program will be evaluated in 1991, and the EPA may recommend that it be modified, allowed to expire, or extended to the entire nation at that time.

The impact of the changing requirements for handling medical waste on the nation's health care system can be judged from the experience of New York City's hospitals. The way in which these institutions have coped with the stringent requirements of New York City and New York State as well as the federal requirements is a signal measure that will help shape the EPA's recommendations to Congress.

PRE-1980 PERSPECTIVE

Specific, formal authority for the regulation of infectious waste at the federal level was given to the EPA with the passage of the Resource Conservation and Recovery Act of 1976 (RCRA). It required the EPA to assess "hazardous waste" in order to determine the impact of such waste on health and the environment. Regulations for hazardous waste control were to be promulgated and enforced by the EPA if and when there was a need. Infectious waste was included as a single facet of hazardous waste.

In 1978, the EPA proposed regulations under RCRA that were to regulate the disposal of infectious waste as a hazardous waste,[1] but deleted them from the regulations promulgated in final form in 1980.[2] The EPA based this action on its assessment that there was no evidence to show that infectious waste had the potential to "cause harm to human health or the environment."[3] Without that evidence, the EPA held that it was not authorized to act under the RCRA statute, and left the regulation of infectious waste to individual states.

The lack of a perceived hazard is evident by the vague regulations governing infectious waste disposal at all levels of government before 1980. Hill-Burton construction standards, the Centers for Disease Control, and the Joint Commission on the Accreditation of Hospitals (JCAH) all provided brief statements requiring only that hospital waste, infectious or not, should be disposed of "properly."[4]

The New York City Health Code required the incineration or burial by a licensed mortician of human remains or body parts and the "proper" disposal of waste from infectious cases. More guidelines existed for the sterilization of instruments and equipment used in infectious cases than for the waste generated in treatment.

At the state level, since 1966 Title X of the New York Code Rules and Regulations (NYCRR) chapter 5, Medical Facilities Code, section 702.2, paragraph (e), has stated in part, "dressings, surgical, biological and obstetrical wastes, infectious materials and disposable syringes and needles shall be destroyed on the premises by incineration, or as approved by the department." New York City health facilities were thus required to operate a hospital-based incinerator or find an alternate method for the disposal of infectious waste that was acceptable to the state department of health.

As air pollution control laws made it more difficult to operate an incinerator, the most common method of infectious waste disposal resorted to by hospitals became contracting with private carting companies. The carting company simply assured the hospital that its infectious materials were disposed of in accordance with the prevailing regulations. No record of generation and disposal was required, and no mechanism existed for ensuring that infectious materials were in fact disposed of properly after pickup.

1980–1985 DEVELOPMENTS

In 1982, the EPA published its guidance document, *Draft Manual for Infectious Waste Management* (US Environmental Protection Agency, SW 957, Sept 1982). It designated 13 categories of potentially infectious waste, based on specific waste types rather than source of the waste. These waste types were designated as infectious due to their likelihood of containing pathogens that "because of their type, concentration and quantity may cause disease in persons exposed to the waste."[5]

The draft was to be used by the states to develop local regulations for waste management and by individual institutions to develop in-house procedures. Rather than vague admonitions to "handle waste properly," a foundation for a uniform definition of infectious waste and appropriate handling techniques was developing. No formal regulations were planned by the EPA, as no solid evidence of

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harm to human health or the environment had been linked to infectious waste disposal.[6]

The State of New York Department of Health Memorandum series 84-12 of March 15, 1984, sought to clarify exactly what was defined as infectious waste, in order to ensure uniform handling and disposal practices under 10 NYCRR 405.3(b). Surgical waste, sharps, obstetrical waste, pathological waste, biological waste, isolation waste, blood and blood product waste, renal dialysis waste, laboratory waste, serums and vaccines, and body parts (all as defined in the memorandum) were required to be incinerated. Alternate means of disposal were allowable if approved in writing by the Department of Health after an application process.

This natural evolution of infectious waste regulation was interrupted by a number of factors. Throughout the 1970s and early 1980s, the number of disposable items in use in patient care areas steadily increased, thus increasing the volume of potentially infectious waste generated in patient care.[7]

Handling of infectious waste became a major concern of sanitation workers in New York City, and the mayoral campaign of 1985 focused on the issue. The result, Local Law No. 57, established harsh fines to force changes in New York City hospitals' waste disposal practices.

Local Law No. 57 was simple: all waste emanating from surgical procedures, patients in isolation, research and pathological laboratories, and the infectious patients of doctors and dentists would be barred from municipal landfills and incinerators. In addition, hospitals were to be charged by the City of New York for the routine collection of "regular" waste, all of which was to be incinerated.

Private haulers possessing a New York State Department of Conservation Waste Transporter permit, as required by the Environmental Conservation Law, Article 27, Title 6 NYCRR part 364, could transport infectious waste not burned on site to an appropriate treatment facility. The penalty for noncompliance was up to a $10,000 fine for each violation, and suspension of pickup of "regular" trash for up to six months. Since a "violation" was defined as "each container or bag of waste with a capacity of not greater than one cubic yard," the penalty amounts could escalate to the point of bankruptcy with one gross violation.

The result of the city's action was a radical decrease in the amount of improperly disposed of waste. Hospitals not equipped with incinerators capable of burning biological waste contracted with infectious waste haulers, which agreed to haul the hospitals' infectious waste to an approved incinerator for a fee of up to $0.335 per pound of waste. The members of the Joint Purchasing Corporation of New York expected to spend between $20 million and $30 million for such services in 1986.[8]

1986–1987 DEVELOPMENTS

While New York City hospitals were adapting to the new local regulations, the EPA released its *EPA Guide to Infectious Waste Management* (US Dept of Commerce National Technical Information Service EPA/530-SW-86-014, May 1986) in a finalized form in May 1986. Its purpose, as in 1982, was simply to "provide guidance on the subject of infectious waste management" to health facilities and state regulatory agencies.[6]

Although the EPA had once again declined to promulgate federal regulations, the guidance document provided a solid foundation for the future regulation, handling, and disposal of infectious waste. First, the EPA defined infectious waste as "a waste capable of producing an infectious disease," and noted that four factors are important in determining if a waste is in fact infectious: presence of a pathogen of sufficient virulence to cause a disease; dose; portal of entry; and resistance of the host. Health officials may make rational judgments as to what is to be segregated out of the routine waste stream as infectious based on the presence or absence of these factors.

The EPA recomended that six categories be designated as infectious: isolation wastes; cultures and stocks of infectious agents; human blood and blood products; pathological waste; contaminated sharps; contaminated animal carcasses, body parts, and animal bedding. Optional categories for evaluation by responsible persons in each institution included contaminated equipment, wastes from surgery and autopsy, laboratory wastes, and dialysis unit wastes.

The comprehensive recommendations made by the EPA also included the recommendation that each health care facility prepare an infectious waste management plan that would include the following: designation of infectious waste; segregation of infectious materials at the point of generation; packaging of infectious waste; storage of infectious waste prior to transport or destruction; on- and off-site transport; monitoring of treatment technologies; compliance with state and local regulations.

The EPA guidelines provided the tools that New York City hospitals needed to devise an effective program to cope with Local Law No. 57. In-house handling, containment, and storage of infectious waste was safe and effective using the guidelines provided.

At the hospital's doors, control of its infectious waste was consigned to a waste hauler licensed by the New York State Department of Environmental Conservation (NYSDEC). While the hauler was entrusted with the responsibility for proper disposal of the waste, under both RCRA and the Comprehensive Environmental Response, Compensation and Liability Act of 1980—the "Superfund Amendment"—the hospital retained its liability for the appropriate handling and disposal of the waste.[9]

The importance of this concept of "cradle to grave" liability cannot be overemphasized. Until absolute proof of the destruction of the infectious waste consigned to transport was received, the hospital remained fully responsible for its whereabouts. While the transporter could be held liable for its transport practices, the hospital was more easily targeted and penalized than the transporter.[10]

Since only one commercial incinerator was licensed to burn biohazardous waste in New York State, most infectious hospital waste was being transported across state lines to be destroyed. The expense of long distance transportation gave incentive to a few unscrupulous fly-by-night companies to cut corners. The resultant mishandling gave hospital administrators nightmares, as illegally dumped medical waste made the headlines in many parts of the country, but most frequently in the Northeast.

The horror stories were caused by a very few members of the transporter industry. Most were responsible corporations providing a necessary service to the hospitals. Licensed by the NYSDEC, these transporters hauled waste to biohazardous incinerator facilities in places as diverse as Quebec, Virginia, Pennsylvania, North Carolina, and South Carolina.

One important aspect of their service included the voluntary adoption of a four-part manifest system for the tracking of the client hospital's waste, and liability insurance policies for amounts of $1 million or more maintained by the transporters, so that funds would be available for cleanup in the event of injury, mishandling, or illegal dumping of infectious waste. These measures at least partially protected the client hospitals from transporter negligence.

The regulations subsequently promulgated in New York State adopted these features. In 1987, amendments to the State Public Health Law were passed governing the storage, treatment, and disposal of infectious waste generated in Article 28 facilities and clinical laboratories,[11] as well as amendments to the State Environmental Conservation Law governing treatment, storage, and disposal of infectious waste after it had left the generator's facility.[12] Both amendments were to take effect on April 1, 1988, as part of the regulations established by the New York State Department of Health and NYSDEC, respectively.

STATE REGULATION: 1988

The implementation of the 1987 legislation amending New York State's Health and Environmental Laws took place in 1988. Chapter 446 of the Laws of 1987, Article 13, Title XIII, Public Health Law of New York State, addressed the handling, storage, and treatment of infectious waste on site; Chapter 431 of the Laws of 1987, Title 15, Environmental Conservation Law of New York State, addressed the issue of infectious waste transporter requirements. New York City hospitals, coping with Local Law No. 57 since 1986, needed to make only minor changes to their waste handling procedures to comply with the new legislation, if they had in fact been in compliance with the local law.

But while these rules were being implemented by the health care industry, the infamous beach washups of 1988 had begun. By August 1988, more stringent emergency Part 70 and Part 364 regulations were promulgated as a direct response to the intense public pressure precipitated by the beach washups.[13]

Changes to the Part 70 and Part 364 emergency regulations directly affected the disposal procedures used by the city's hospitals. Labeling of transport containers, floatable tags in each red bag consigned to transport, and stringent requirements for timely completion of official NYSDEC manifests were included.

At the city level, Brendon Sexton, Commissioner of Sanitation, issued a Notice of Adoption of Emergency Regulations, effective July 13, 1988, which required all hospital waste collected by the New York City Sanitation Department, red and black bags alike, to display generator identification. Noncompliance would result in the loss of collection services. Hospitals using New York City Sanitation Services rather than a private carter for its "regular" black bag waste were thus required to identify even routine trash.

At both the state and local level, city hospitals were faced with new regulations prompted by the public outcry. Ironically, after investigation by state and federal authorities, it was found that hospitals were not a significant source of the medical items washing ashore.[14]

From the operations standpoint, the state regulations were certainly a success. Infectious waste was clearly defined by category, and straightforward rules existed for disposal practices. The stringent regulations faced by the transporters virtually eliminated the risk of illegal dumping if the hospital prudently checked the transporters' permits, etc.

Unfortunately, the cost of infectious waste disposal as defined by state and local laws is approximately $.40-$.60 per pound.[15] Supplies for the preparation of infectious waste for transport and administration and labor costs are additional. These costs are not reimbursable under present payment mechanisms, and must be considered a serious drawback of the regulations: New York City hospitals lost more than $120 million in 1988.

Interestingly, while administrative costs have increased with the more stringent regulations, disposal costs remained static in 1988, or actually decreased slightly. The competition among transporters and the increasing efficiency of those corporations surviving that competition contributed to this phenomenon. Since most reputable transporters had been meeting the major cost requirements of the new regulations before their implementation, simply to remain competitive (ie, liability insurance), there was little additional expense to pass on to the generator.

THE MEDICAL WASTE TRACKING ACT OF 1988

The nonpartisan nature of the Medical Waste Tracking Act and its overwhelming passage signaled the first step in the development of uniform national regulations for the handling and disposal of infectious waste.[16] Its purpose was to establish a tracking program under the aegis of the federal EPA to "immediately address and correct . . . improper disposal of medical waste."[17]

Significantly, Congress has found that "improper disposal of medical waste is presenting an imminent and substantial endangerment to public health and the environment in certain coastal areas."[15] Under RCRA, a new subtitle J was added, which required the EPA to regulate medical waste due to this imminent hazard.

The act directed the EPA to establish a two-year demonstration tracking program to gather information on the infectious waste issue while administering regulations to control the disposal of infectious waste.

Within three months of the expiration of the demonstration program, the EPA is to submit a comprehensive report to Congress indicating the number and types of generators of infectious waste in the United States, the types and amounts of waste generated, the "present or potential threat to human health and the environment" posed by infectious waste, the cost impact of infectious waste management, and the success/failure of the demonstration program. The EPA will also report on alternate methods for disposing of infectious waste, the effectiveness of state law, and the impact of the demonstration project on the handling, storage, and disposal practices. Interim reports due in nine months from the start of the project and in June 1990 are to serve as "progress reports" on the EPA's activities during the project.

The act included a list of ten types of medical waste, five mandatory categories to be included in the tracking program, and five optional categories that could be included if it was determined to be necessary by the EPA (due to the presence of a potential hazard to human health or the environment). The creation of a manifest system similar to the hazardous waste manifests already in use was to serve as the vehicle for the tracking of the waste from "cradle to grave."

The EPA is authorized to issue injunctions and to impose civil and criminal penalties from $50,000 per day and two years' imprisonment for knowing violations of the EPA's regulations, to $250,000 per day and 15 years' imprisonment for a knowing violation that places another individual at risk of serious injury or death. Authority to perform site inspections, examine records, and to collect and sample medical waste in order to obtain necessary data for EPA reports was also included.

EPA Regulations: June 22, 1989

On March 24, 1989, the EPA published Standards for the Tracking and Management of Medical Waste (40 Code of Federal Regulations (CFR) Part 259) in response to the congressional mandate imposed by the Medical Waste Tracking Act of 1988.[18] Effective June 22, 1989, these regulations set forth a program of medical waste tracking and control in a small number of states in order to gather the necessary information to establish a nationwide medical waste disposal program, if it is determined to be necessary.[19]

Under the Part 259 regulations, the EPA has defined medical waste as

> any solid waste generated in the diagnosis, treatment (e.g., provision of medical services), or immunization of human beings or animals, in research pertaining thereto, or in the production or testing of biologicals. The term does not include any hazardous waste identified or listed under part 261 of this chapter, or any household waste.[18]

A comprehensive definition of the waste to be regulated, by waste category, was included in the EPA regulations as "regulated medical waste." This term is used rather than the term "infectious waste," as the EPA itself notes that "Most medical experts and public health officials, including those from the Centers for Disease Control, believe that medical waste does not pose a public health threat, especially after it has been exposed to air and water."[20]

"Regulated medical wastes" include the following: cultures and stocks; pathological waste; human blood and blood products (in addition to bulk blood as defined at the state level, the EPA definition includes "items saturated with human blood, or items that were saturated and are now caked with dried blood," as well as intravenous bags); sharps; animal waste; isolation waste; and unused sharps.

These seven categories of regulated medical waste encompass those items that had been considered to be infectious by New York State, with one notable exception. The blood and blood products category has been greatly expanded, causing consternation among the state's hospitals.

The inclusion of blood-caked and blood-soaked items in this category creates a gray area in which a "judgment call" becomes necessary. In its comments on CFR Parts 22 and 259, the Greater New York Hospital Association (GNYHA) has noted that "Trying to discriminate between blood soaked, blood stained, and blood tinged can only result in massive oversorting in favor of 'blood soaked' to ensure compliance and avoid penalties."[22] Facilities laboring under tremendous amounts of adverse publicity will naturally err on the conservative side. New York City hospitals, faced with a Department of Sanitation compliance force assigned specifically to find violations, face tremendous difficulty in complying with this generalized definition.

It is easy to conceive the tremendous increase in the costs incurred for disposal when items previously disposed of at a cost of $.02 per pound now cost up to 30 times that amount. Compliance is expected to cost New York hospitals $60 million a year according to the GNYHA.[22]

It is also disconcerting to note that in addition to blood-caked items, intravenous bags are also to be regulated, as the EPA states in its preamble to the legislation, merely for aesthetic reasons.[22] The cost of disposal of intravenous bags alone will amount to more than $12,000 per year in a 200-bed facility.

As a study to determine overall infectious waste disposal practices in the United States, the EPA regulations may be fully supported. The EPA itself notes in its preamble to the regulations, however, that the aim of the Congress to control the washup of medically related waste on our shores may not be accomplished.[18] In fact, the EPA notes, "EPA has collected marine debris from harbors in Boston, New York, New Jersey, Baltimore, Miami, Seattle, and San Francisco. Preliminary information indicates that less than one per cent is medical waste or drug related."[20]

The EPA has estimated that compliance with the program would cost no more than $31 million per year if all ten states originally named in the Medical Waste Tracking Act had been included in the program. An individual hospital's cost was estimated to be $3,750. This is simply not the case. First year costs at a small facility will run closer to $20,000 including administrative costs. These costs are not reimbursable under present payment methodology, and must therefore have a direct negative effect on the quality of patient care. While this two-year demonstration program is to provide information to the Congress for permanent action on the infectious waste issue, the cost for this information is being borne by the hospital patient.

Conclusions

New York City hospitals are operating under a three-tiered system for infectious waste disposal. Local Law No. 57 and the "watchful eyes" of the New York City Sanitation Department are the first tier of the system. Next, the NYSDEC enforces the Health and Environmental Regulations included in Part 70 and Part 364 of their respective state codes. Finally, the EPA's Standards for the Tracking of Medical Waste, CFR 259, mandate specific disposal

TABLE I. A Comparison of Requirements—The Three-tiered System

Requirement	New York City	New York State	Federal Government
Regulated category—definitions			
Animal waste/animal carcasses (from laboratory animals)	Yes	Yes	Yes
Blood/blood products (from patients in isolation)	Yes	Yes	Yes
(including items caked with dried blood and intravenous bags)	No	Yes	Yes
Cultures and stocks	Yes	Yes	Yes
Isolation waste	Yes	Yes	Yes
Laboratory waste ("including all disposable matter eminating from laboratory areas")	Yes	No	No
("that were in contact with infectious agents")	Yes	Yes	Yes
Pathological waste	Yes	Yes	Yes
Sharps	Yes	Yes	Yes
Surgical waste ("including all items emanating from OR, ie, gowns, shoe covers, masks")	Yes	No	No
Packaging and handling requirements			
In-house			
Red bags	Yes	Yes	No
Sharps containers	Yes	Yes	Yes
Labelling of inner containers	Yes	Yes	No
Labelling of sharps containers	Yes	Yes	Yes
Floatable tags	No	No	No
Rigid, waterproof outer containers	Yes	Yes	Yes
Outer containers			
Date of pickup	No	Yes	Yes
Generator's name and address	Yes	Yes	Yes
Transporter's name and address	No	Yes	Yes
Infectious or biohazard symbol	Yes	Yes	Yes
Transport requirements—facility			
Record keeping (four-part manifest)	No	Yes	Yes
Transport by licensed transporters only	No	Yes	Yes
Transport requirements—transporter			
Record keeping (four-part manifest)	No	Yes	Yes
(log)	No	Yes	Yes
Liability insurance	No	Yes	Yes
"Dedicated" vehicles for waste transport	No	Yes	Yes
State and federal permits	No	Yes	Yes

practices at the federal level.

On June 5, 1989, the New York State Senate enacted S.6188-A, a bill that directed the State Department of Health and NYSDEC to amend the existing regulations to better conform to the Medical Waste Tracking Act.

In response to this legislation, the NYSDEC issued revised Part 364 regulations on July 10, 1989. The term "potentially infectious waste" has been deleted from the state regulations, and "regulated medical waste" has taken its place. The state definitions of "medical waste" and "regulated medical waste" have been made to conform to the EPA definitions. Of special concern, the state's blood and blood products category has also been expanded to include "blood saturated items" and intravenous bags.

In addition to the EPA's categories, the state includes surgical and autopsy wastes from patients in isolation and dialysis wastes as regulated medical wastes. The use of floatable tags was required at the state level as well, but this provision was deleted from the regulations on January 12, 1990.[21]

While these revisions were issued on an emergency basis and are effective immediately, a public comment period is planned, as are public hearings. This initial step toward conformity is a welcome movement toward the simplification of administration of an infectious waste disposal program. An important part of the present EPA program is to determine if infectious waste does in fact warrant specialized (and costly) disposal practices. It has not been proved that infectious waste poses a hazard to health or the environment. The recent change in terminology to the more correct terms "medical waste" and "regulated medical waste" rather than the term "infectious" is a reflection of the learning process of our legislators, who are becoming aware of the questionable nature of the infectious waste threat.

Despite the lack of concrete evidence of a hazard to the public, special handling and disposal procedures are a necessity to protect those handling the waste. These procedures must be based on sound scientific evidence as to safety, efficiency, and cost-effectiveness. Public hysteria cannot be allowed to dictate disposal practices in light of the limited funding available.

A national program for medical waste disposal will be welcomed by New York City hospitals as an end to the ever increasing regulatory net surrounding waste disposal. An immediate result will be the stabilization of disposal costs.

Currently, New York City generates 300 million pounds of medical waste each year. Almost 55 million pounds of this waste was consigned to infectious waste transporters in 1988, at a cost of approximately $.50 per pound.[22] Transporter costs amounted to almost $27.5 million in 1988.

In-house costs have also increased as handling proce-

TABLE II. An Outline of Possible Recommendations for Effective Medical Waste Disposal

I. Creation of a national solid waste disposal program containing uniform requirements for medical waste disposal. Requirements must include the following:
   - definition of infectious waste by category based on current epidemiologic evidence
   - handling and packaging requirements
   - manifesting requirements for off-site disposal
   - transporter licensing requirements
   - disposal facility requirements
   - requirements for small quantity generators

II. Creation of a mechanism for reimbursement of disposal costs

III. Continued public education regarding the lack of a public health threat by hospital waste

IV. Capital investment in regionalized incinerator sites to handle medical waste from all generators

V. Funding for research for alternate disposal options dures and administration of the compliance program have grown more complex. These costs include supplies (red bags, disposable gloves, carts, storage area, etc); labor (segregation and collection); and administration (tracking forms, surveys, confirmation of procedures in light of changing regulations).

In-house costs are more difficult to estimate, as they may vary from institution to institution. A good estimate is approximately 25% of the cost for transporter services at the institution. Using this amount, 1988 disposal costs for regulated medical waste disposal were almost $34.5 million in New York City alone.

The next two years will decide the future of regulated waste disposal in the United States. A concise description of regulated medical waste must be devised. The blood and blood products category must be altered to reflect the need for regulation rather than including a broad array of items for aesthetic reasons. This alteration alone will save millions of dollars for patient care. In addition, epidemiologic evidence proving the need for regulating any part of the waste stream is a necessity for cost effective operation. Payment methodology must take into account new operations requirements and technologies in order to avoid short changing patient care.

For the foreseeable future, incineration is the most cost effective and complete method of infectious waste disposal. Volume reduction is a major advantage over autoclaving and other treatment methods, all of which require the landfilling of treated medical waste.[23] Incinerators reduce the volume of material to be landfilled by up to 8 to 1. Incinerators may also be equipped as co-generation facilities, where electricity and steam are generated and sold to a public utility, partially offsetting operating costs. Regional incinerators have gained support as a cost effective method of waste disposal, reducing the investment necessary for any one facility and encouraging shared operations costs. A regional incinerator can be manned by highly trained personnel and equipped with computerized pollution control and monitoring systems that a single facility simply cannot afford. Transportation costs are also decreased as waste is no longer trucked across state lines for disposal.

The problem of regulated medical waste disposal is a part of the larger solid waste disposal dilemma. A national effort is needed to develop an effective waste disposal system for all types of solid waste. As the public begins to perceive medical waste disposal as part of the broader disposal issue, regulation of a sound nature, based on scientific evidence, may take place.

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A history of the Ray Brook State Tuberculosis Hospital

KENNETH W. WRIGHT, MD; JAMES MONROE, MD; FREDERICK BECK, MD

In 1889, Biggs, Prudden, and Loomis prepared a paper in New York City entitled, "Report of the Prevention of Pulmonary Tuberculosis."[1] Under a regulation passed in New York City in 1893, the medical institutions were required to report tuberculosis. Four years later, Biggs secured passage by the New York City Board of Health of an ordinance declaring tuberculosis to be infectious and communicable and instituting mandatory reporting by physicians.[2] As is often the case in innovations of this kind, conservative members of the medical profession and of the public opposed an attempt to register consumptives for fear it would add to their hardship.

Governor Theodore Roosevelt signed chapter 416 of the New York State Laws of 1900 that established authority for a State Hospital for the Treatment of Incipient Pulmonary Tuberculosis and appointed a board of trustees to carry out recommendations of the legislature. The first meeting of the board was held in Albany on May 31, 1900. Present were Frank E. Kendall, John H. Pryor, and Howard Townsend; W. G. MacDonald and Walter Jennings were absent.[3]

In the ensuing months, 22 sites were evaluated by Pryor, Kendall, and Edward Baldwin of Saranac Lake. All but Lake Clear, Dannemora, Lake Kushaqua, and Ray Brook were rejected. On September 6, 1900, at a public hearing, citizens from Saranac Lake and Lake Placid were divided in opinion over the question of appropriation of land by the state in general, and for building a state hospital in particular. It was less than a decade since enactment of chapter 707 of the Laws of 1892, and the state constitution, Article XIV of 1894, which established the 2.8-million-acre Adirondack Park; many citizens within the area were fearful of state takeover. The outcome of the hearing was not recorded, but an announcement was forthcoming on January 21, 1902, that 516 acres of property at Ray Brook were to be used for the construction of a new hospital.[4]

In 1903, a state appropriation was made for dormitory pavilions, connecting corridors and solaria, furnishings, and laundry and kitchen equipment totalling $115,000, and architects were authorized to prepare the necessary drawings. Gallo[5] reviewed the development of architectural styles that prevailed in the Saranac Lake community. William Scopes and Maurice Feustmann influenced styles in building cure cottages and sanatoria.[5,6]

During 67 years of operation, Ray Brook had nine directors: John H. Pryor, 1904–1905; Melvin P. Burnham, 1905-1907; Albert H. Garvin, 1907-1917; Harry A. Bray, 1917-1950; Frederick Beck, 1950-1959; James Monroe, 1959-1965; F. Clark White, 1965-1968; and Bela O. Duboczy, 1968-1971.

Pryor's tenure was brief, but he was a significant figure in the early days of the hospital, as he was a member of its board of trustees and a member of the site selection committee. He also superintended the initial construction and admitted the first patient on July 1, 1904. Pryor wrote on empyema and on immobility of the diaghragm.[7,8] He stated in the first annual report, "the number of patients admitted the first three months was limited to 44 because only the tent camp could be utilized. The pavilions constructed .... are still not completed"[9] (Fig 1). Examining physicians for applicants for admission to Ray Brook were H. M. Biggs for New York City, S. B. Ward for Albany, H. L. Elsner for Syracuse, and H. R. Hopkins for Buffalo. During the first six months, 82 patients were admitted. The following year the capacity was listed as 150.

Melvin P. Burnham succeeded Pryor. During this time, the payroll listed monthly salaries as follows: director, $200; domestics and waitresses, $14–$16; laborers, $20–$30; coachman, $35; three nurses, each $35. Albert H. Garvin followed Burnham on July 1, 1907, and was joined by a young Canadian, Harry A. Bray, and by Harry J. Brayton of Syracuse; in 1909, John M. Wise of Buffalo joined them. In 1917, Garvin went to France with a civilian tuberculosis commission, and Bray (Fig 2) became acting director until appointed director in 1919 when Garvin entered practice in Buffalo.

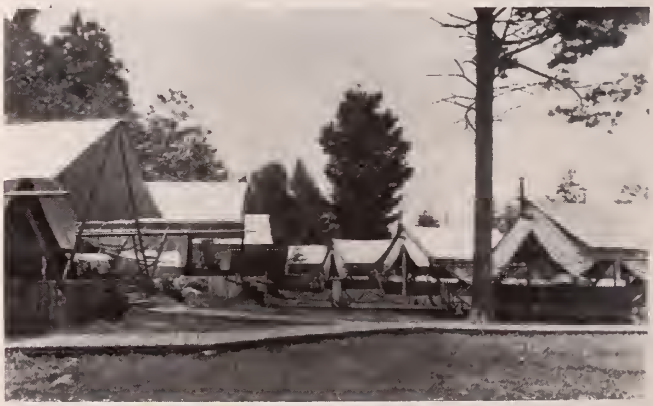

FIGURE 1. "The tent colonies reappeared on the hill each summer until 1920" (Kurung JM: *Ray Brook 1904–1954*, unpublished manuscript, photograph in a collection presented to James Monroe, MD, by Joseph M. Kurung).

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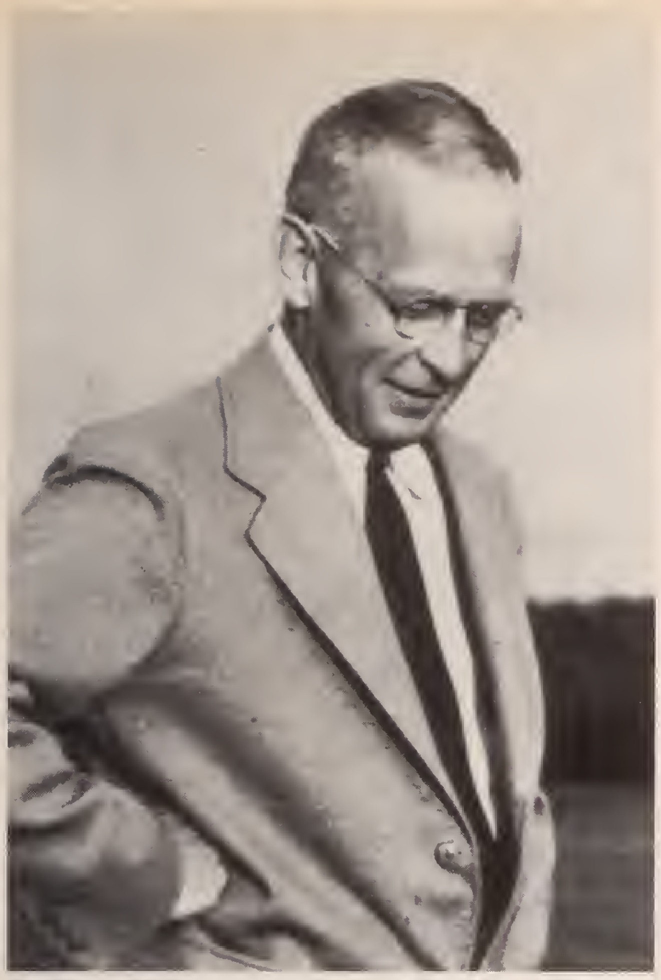

FIGURE 2. Harry A. Bray, MD, Director of Ray Brook, 1917–1950 (photographed by Joseph M. Kurung, Collection of James Monroe, MD).

Of all the directors, Bray served the longest, more than 30 years. Born in the Province of Ontario, Canada, into a physician's family, he received his medical education at Trinity College in Toronto and did postgraduate work at the University of Edinburgh. He trained at Lakeside Hospital in Cleveland, Ohio. Bray suffered from a recurring affliction of his eyes, and in later years was assisted by Miss Schubel, secretary, and Miss Magdalene Nemeth, a nurse originally from Cleveland, who read to him when he had to protect his eyes (Nemeth M, Saranac Lake, NY, private correspondence, 1988).

Under his tenure, forest and swamp surrounding the hospital gave way to attractive parks with woodland brooks and ponds, retaining walls and steps, walks, and lawns (Fig 3). By 1928, Bray arranged to build a new laboratory. In 1937, he managed construction of a nine-hole miniature golf course for use by the patients. In 1939, a 99-bed infirmary was constructed with a surgical unit, new x-ray department, laboratory facilities, pharmacy, outpatient clinic facilities, and new classrooms for adult education.

According to Kurung,[4] the homelike atmosphere of Ray Brook in large part resulted from planning done by Bray, who sought the advice and help of friends of the hospital. The furnishings were attractive, and the dining area was conducive to congeniality.

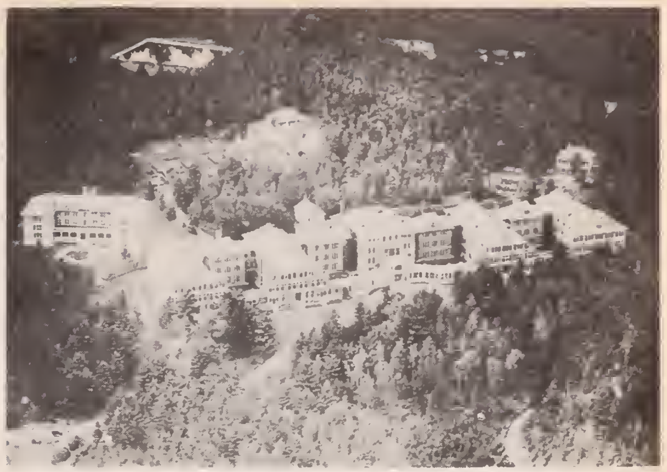

FIGURE 3. Aerial view of Ray Brook ca. 1926 (photograph in a collection presented to James Monroe, MD, by Joseph M. Kurung).

Ray Brook became a self-contained community with a post office, and a railroad station served by the New York Central and Delaware and Hudson spur from Lake Clear. Patient Henry Levy's memoirs of 1931 recorded the following:

> After an eleven hours train trip, my glimpse of a small cubicle of a station buried in heavy snow, stuck seemingly in a cold wilderness, was far from comforting. . . . I was ushered in and instead of cold foreboding walls, was amazed at the warm reception.[10]

Ray Brook had a "piggery" and butcher shop dating to 1908, an orchard, store rooms, a bakery, and a laundry; a coal burning steam plant provided heat and generated DC current until the 1950s, when an electric power line connected the hospital to the Paul Smith hydroelectric plant. Many employees lived on the grounds. The "village" had its own chlorinated water supply and sewer system. In the early days, ice was harvested from a nearby pond. Fire control measures were enforced by the hospital's own marshall; except for exterior walls, the hospital was built of wood and was considered a fire trap. Said one who recalled the stringent fire rules, "Smoking was verboten everywhere but in 'rest rooms' under careful supervision." The same patient recalled Pullman Porter, PHD: "Dr. Porter was Professor of Ornamental Floral culture (generally known as landscape architecture) at Cornell. He came to Ray Brook summers and gave Dr Bray and Clyde Perry directions for plantings. Cornell students were assigned summers' jobs with pay to work with the grounds crew."

THE WHITE PLAGUE

The situation in 1904, when the New York State Hospital for Treatment of Incipient Tuberculosis officially opened, was graphically described by Rene J. Dubos:[11]

> The cough of the tubercular was heard every where in our communities. A feverish glow betrayed the advanced stage of consumption in two out of every one hundred persons and foretold of death within a few years. Tuberculosis was the White Plague. . . .

There had been little if any change since Austin Flint (1812–1886), in his first edition of *A Treatise on the Prin-*

ciples and Practice of Medicine,[12] stated, "of the diseases which afflict the human family, this (tuberculosis) is one of the most important, prevailing, more or less, in almost every quarter of the globe, and occasioning, exclusive of those which prevail epidemically or endemically, larger proportion of deaths, than any other disease." In a further passage he went on to ask, "Is the tuberculous cachexia removable by any known remedy? The question is to be answered distinctly in the negative."

Osler's biographer tells of Johns Hopkins students and house officers who developed tuberculosis.[13] "In the class of 1899 alone four (of nineteen) developed consumption"; a fifth member, "possibly the most brilliant" died. The death rate from tuberculosis then exceeded 250/100,000,[14] compared with 1.2/100,000 today.[15]

Monroe[16] (Johns Hopkins class of 1934) detailed his experiences:

> It was accepted that ten to fifteen percent of medical students developed active tuberculosis. Seven of the seventy in my class developed active disease. We were exposed to infection during training on the wards, in outpatient clinics, in the autopsy room. As a third year student I was assigned to examine a man who had been coming to the clinic for "asthma". During my work-up I collected his sputum, made a slide, stained it with Ziehl's carbolfuchsin solution. This unconcentrated specimen contained so many tubercle bacilli that it remained visibly red even after applying acid-alcohol. The man died before a bed became available for him in the tuberculosis hospital.

Monroe's own history of tuberculous pleurisy with effusion is not atypical:

> I developed pleurisy with effusion, 'cured' one year at Trudeau, and returned to school in Baltimore. After finishing my fourth year in medical school, I had a relapse and developed cavitary disease, so instead of entering pathology training at the Mallory Institute in Boston, I had to return to Trudeau as a patient. There I was soon allowed to work with the staff, but my disease became progressive and I had a "phrenic" (temporary phrenic nerve interruption). Pneumothorax was unsuccessful because of pleural adhesions. I was transferred to Ray Brook and after some time underwent three-stages of 10-rib thoracoplasty at the Saranac Lake General Hospital by Dr Warriner Woodruff. I was assigned to take histories and do sputum examinations as soon as deemed ready by my floor physician for my "cure task." Patients arrived from metropolitan New York, rural villages and communities, and some came from Trudeau.[17]
>
> Despite an almost uniform fear of tuberculosis, there was a state of euphoria, "spes phthisica," in the patients at Ray Brook and around Saranac Lake. Almost everybody at Ray Brook had tuberculosis, some persistently active, others inactive, but there was rarely an expression of dread or caution.[16]

The situation of tuberculosis infection among medical students was studied in 1938–1944 and reported by Gordon M. Meade in 1948.[18] The medical school class at the University of Rochester varied in size from 40 to 60. In 1938, among newly admitted students, 42.6% had positive Mantoux tests. Intensive follow-up skin testing was inaugurated. By the end of the first year, 54.2% were positive reactors, but at the end of their second year, 92% were positive reactors. Contact with tuberculosis tissue in pathology and in the autopsy room was considered the most likely source of infection for the second year students. Beginning in 1943 (class of 1945), attendance by students in the autopsy room at Strong Memorial Hospital for known tuberculosis patients and at the Monroe County Tuberculosis Hospital was eliminated. Immediately the rate of second year students testing positive at the end of the year dropped to 51.9%, as compared with 94% in the previous class.

THE RAY BROOK CURE TASK SYSTEM

The new state tuberculosis hospital was located in the Adirondacks in an environment advocated by Dr Brehmer (1826–1889) in Germany.[23,24] William Osler (1849–1919) in *The Principles and Practice of Medicine*[25] noted,

> The question of environment is of first importance in treatment of tuberculosis. It is illustrated in an interesting and practical way by an experiment by Trudeau, showing that inoculated rabbits (with tubercle bacilli), confined in a dark, damp place, rapidly succumb, while others, allowed to run wild, either recover or show slight lesions. . . .

In the first annual report of Ray Brook, Pryor stated, "The tent camps have proved a great success and have won many words of approval from experts." By the 1920s, there was a population in excess of 4,000 patients in Saranac Lake. A small industry grew up in the manufacture of a "cure chair" modelled after one designed by Dr Dietweiller. This was produced by A. Fortune & Co, undertaking and furniture, and George L. Starks & Co, hardware, as well as others in and around Saranac Lake.

A "cure" industry had grown up around Saranac Lake. Besides Trudeau Sanatorium there were "cure cottages." Stoney Wold Sanatorium at Lake Kushaqua was established in 1903 for the treatment of working women and children by a group of philanthropic women, including Mrs Herman M. Biggs. Biggs stated the following:

> In general the cases of tuberculosis in the laboring classes should be divided into two groups, both of which should be as far as possible treated in institutions. First, the incipient, early and moderately advanced but hopeful cases, which should be treated in sanatoria situated in favorable locations in the country; and, second, the advanced and unfavorable cases, which should be treated in special hospitals, where they can be so guarded that they will not communicate the disease. . . . It does not seem to me desirable that the advanced cases should be rendered uncomfortable by being forced to live and sleep out-of-doors at all seasons of the year . . .[1(p196)]

Harry A. Bray advocated the "cure task" system. His idea of "rest cure" incorporated out-of-door "curing" and graduated physical activity. Patients slept in the open in tents or on covered unheated porches even during the coldest weather. Patient Levy's memoirs of 1931 state, "You piled on sweaters over long undies and donned a long overcoat assigned to each patient, and armed with mittens and skating cap covering the ears, you ventured forth." Another patient remembers from 1936, "We got cold from below up through the mattress, so we used 6″ layers of newspaper for insulation underneath the mattress, soap stone or crockery filled with hot water at our feet, so hot when we crawled in that it had to be wrapped in a blanket or two. The latter was advertised in *The Journal of Outdoor Life* as 'pigs'."

Dr William Halstead, a Johns Hopkins surgeon, was an advocate of sunlight and open air therapy and read a paper before the first annual meeting of the National Association for Study and Prevention of Tuberculosis.[20] Dr Vincent Y. Bowditch, not to be confused with his uncle Henry I. Bowditch, both prominent physicians in Boston, advocated life in the open.[21]

A graduate of the cure task system reported that "those able for assignment were checked daily by doctor and nurse. Patients entered into this system enthusiastically because of its manifest results. I never saw nor heard of any complaints from them." One young woman went to Ray Brook because of a tuberculous pleurisy with effusion and later developed a parasternal draining tuberculous sinus. Under this cure program, she regained her health and remained to work with Dr Bray, serving as superintendent of nurses, head of housekeeping, and dietician.

Walter L. Rathbun (Yale, 1903), former Ray Brook patient, and author of the first article published in the *American Review of Tuberculosis*, which was entitled "Classification of Pulmonary Tuberculosis,"[22] said, "the 'cure task' worked well at Otisville New York City Municipal Sanatorium as long as we were supplied with the type of patient, i.e., [those] with 'incipient' tuberculosis."

Rest as a part of the "cure" program was evaluated in one of Bray's last published articles entitled "Bed Rest Appraised."[19]

TUBERCULOSIS ENTERS THE SCIENTIFIC ERA

Until the microscope was invented, diagnosis had depended on what could be seen, felt, heard, and smelled. With the coming of the age of bacteriologic discovery and modern tools for examining the living body, the era of clinical medicine, as we know it today, was ushered in. When Herman M. Biggs returned in March 1885 from an apprenticeship in Germany to become instructor at the Carnegie Laboratory, he found William H. Welch already staining the tubercle bacilli. The first health department laboratory in this country resulted from the efforts of Biggs and his colleagues. Soon the new methods of staining bacteria with aniline dyes and techniques for culture and animal inoculation became standardized.

Bray developed the laboratory at Ray Brook to perform work for the district covered by the outpatient clinics as well as the hospital. Kurung[4] described the new laboratory as "a separate building on the hill in the rear of the main building, dedicated in 1928 to Dr Frank Erdwurm, who was a member of the Board of Trustees and its chairman for many years" (Fig 4).

David T. Smith, director of the laboratory before going to Duke as Professor of Bacteriology and Medicine, published his work done there with fuso-spirochaetal lung disease.[26] He told Monroe that his attention was first attracted by patients at Ray Brook who had pulmonary cavities but were sputum negative for tubercle bacilli. Monroe stated that "This was common enough a problem that on my ward there might be 6 or 8 such patients with pulmonary cavities but 'negative sputum'. This got us all interested in non-tuberculous cavitary disease."

Hugh E. Burke, a former director of the laboratory, published several papers about work done there on silicosis and involving animal experiments.[27–29] (Another of the Ray Brook staff, Harry E. Rose, went on to Columbia as Chairman of the Department of Microbiology.) Yegian and Kurung published articles concerning the Ray Brook laboratory work (Fig 4).[30–36]

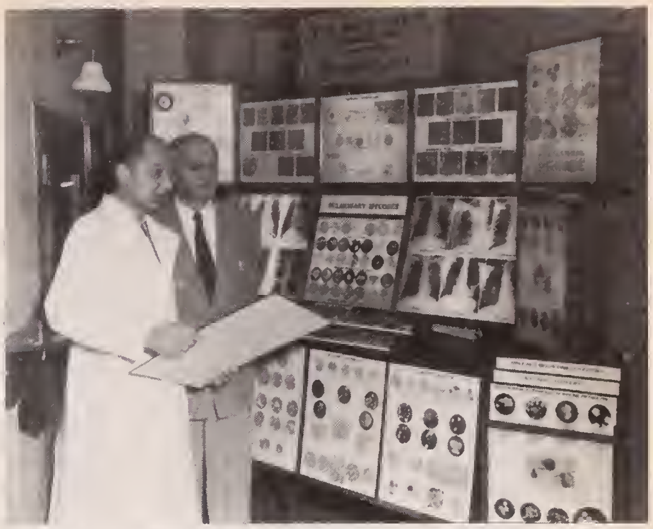

FIGURE 4. Joseph M. Kurung (left) and Diran Yegian (right) at an exhibit demonstrating sputum examination methods and techniques (photograph is one of the collection presented to James Monroe, MD, by Joseph M. Kurung.)

Since there was no morgue at Ray Brook, autopsies were performed by Ray Brook physicians in undertakers' rooms. A special metal box fitted into a suitcase and containing necropsy instruments and jars was in readiness for Joseph Kurung's preparation for pathologic study. Bray taught a healthy skepticism and quoted Morgagni (1682–1771) from the *Five Books on the Seats and Causes of Diseases Investigated by Anatomy* (trans. London by A. Miller & T. Cadell 1769): "Those who dissected or inspected many (autopsies) have at least learned to doubt when others, who are ignorant of anatomy and do not take the trouble to attend to it, are in no doubt at all."

One staff member recalled the frequent visits of Rene J. Dubos, PhD, of Rockefeller Institute, when his first wife, Marie Bonnet Dubos, was a Ray Brook patient:

> Dr Dubos spent many hours in the laboratory and evenings after supper talking about his work. He was a fluent speaker with a slight French accent. He had been with Waksman at Rutgers in the 1930's, and he verified the theory that soil bacteria were a source for therapeutic agents. He isolated Bacillus brevis from which he derived Tyrothricin.

Mrs Dubos never recovered from tuberculosis. Dr Dubos took her home, and she died shortly afterwards, in April 1942. (He and his second wife, the former Letha Porter, wrote the non-technical account of tuberculosis entitled "White Plague.")

Kurung[4] stated that x-ray equipment at Ray Brook was not installed until 1914. With the original Crooks tube, voltage was measured by length of spark gap. Recalling a conversation with the x-ray technician, Ann Sanlow, Monroe[16] reports the following:

> The old tube was around for many years. Miss Sanlow showed it to me and told how frightening to see the spark gap jump 12″–20″, smell the ozone and hear the crackling noise. Ann Sanlow came from the Ukraine and had been a patient. Dr Bray observed her fine handiwork, her knitting and crocheting and tatting, evaluated her personality and chose her to become the x-ray techni-

cian. She took English lessons of a former high school teacher and spoke fluently.

By World War I, the original glass plates were superseded by nitrocellulose film, which turned out to be highly flammable and gave off nitrous oxide gases—highly toxic fumes that can cause pulmonary edema. This was not generally realized until a tragic fire at Cleveland Clinic on May 15, 1929.[37] The code for storing old film was revised, and safety film was introduced.

Fluoroscopy and radiographs taken in different projections to supplement posterior-anterior views and grids to produce better detail all enhanced the study of thoracic shadows. Bray's review of "shadow mutations on the chest roentgenogram in tuberculosis when x-ray exposures were made on the same patient, first at the height of costal then at the height of diaphragmatic inspiration" was accompanied by 12 plates.[38] His study of the etiology of pleural pain[39] is referred to in MacCallum's *A Textbook of Pathology*.[40] Stereoscopic roentgenography had been introduced by 1919. Principles of sectional radiography, known as planigraphy, were outlined in Europe and soon adopted here.[41]

In 1938, at Plattsburg, NY, where Ray Brook held one of its monthly field clinics, a Works Project Administration (WPA) force of 23 men was engaged in clearing an old school house belfry of pigeon droppings. The laborers became ill with flu-like symptoms. This "epidemic" was reported in 1944 at the annual meeting of the American Public Health Association by Nauen of the Ray Brook staff and Korn of the New York State Health Department as "A localized epidemic of acute miliary pneumonitis associated with handling pigeon manure." A decade after the episode, when ten of the 23 were questioned, eight stated that they still had pulmonary symptoms. Repeat chest films revealed calcification not unlike that which was generally interpreted as post-tuberculous. A follow-up was presented by the Ray Brook group in 1949, including x-rays and laboratory analyses of sera saved from the time of the "Plattsburg epidemic."[42] In 1953, Furcolow and Grayson[43] included the "epidemic" in their report entitled "Occurrence of histoplasmosis in epidemics." Monroe and Kurung[44] reported the case of a 56-year-old man, a life-long resident of northern New York State, who was admitted with bilateral apical pulmonary infiltration and cavities. His sputum contained *Histoplasma capsulatum*. Unpublished reports of other fungal diseases of the lungs included actinomycosis, aspergillosis, blastomycosis, coccidioidomycosis, and cryptococcosis, in addition to histoplasmosis.

Within a radius of 40 miles from Ray Brook were iron mines at Crown Point, Lyon Mountain, Mineville, and Witherbee. The ore known as magnetite was in a matrix of granite. At Graphite near Ticonderoga, graphite was mined for use in the pencil-making industry. To make the "lead" for lead pencils of certain hardness, fine sand crushed into fine powder in a ball mill was mixed with graphite. Before industrial hygiene regulations took effect, there was no cover or dust-removing equipment for protection against exposure to the fine sand dust, silicon dioxide. Cases of silicosis came from the iron mines and the graphite industry. Studies were reported by staff members.[45,46]

George W. Thorn (Buffalo, 1929) as a medical student had elected a semester at Ray Brook. In the summer of 1940, he returned. Monroe[16] recalled the following:

> When I was first on the Ray Brook staff, Dr. Thorn and wife and son, along with R. Palmer Howard, a nurse and dietician and laboratory technician, spent two months with us, living on the hospital grounds. Dr. Thorn made clinical rounds with us. With one of our staff members, Howard Dayman [Buffalo, 1934], they did some work on electrolytes with the tuberculosis patients. That summer Ray Brook became a mecca for patients with Addison's Disease because of the presence of Dr. Thorn and his group. Alec T., an outpatient, was one being treated with implanted desoxycorticosterone acetate (DOCA), the first synthetic adrenal cortical hormone, which we received from Dr. Thorn and his group.[47] [George W. Thorn, R. Palmer Howard, and Warfield M. Firor had worked out the principles of DOCA pellet implants at Johns Hopkins University.[48,49]]

Monroe's notes continue:

> Addison's disease in the days when I was in medical school was almost always associated with tuberculosis. Before DOCA was available, Addison's disease was fatal, life expectancy limited to one-and-one half years. With DOCA it was expanded to five or ten years. When cortisone and other steroids came along, these patients had a normal life.

Monroe also preserved the following note:

> Mary G., an Irish national who was a patient at Ray Brook, had Addison's disease. She became one of my best teachers. (I stood up with her when she became an American citizen at ceremonies at the County Seat of Essex County at Elizabethtown.) With George Thorn's teaching, and our patients, that bit of tissue, the Adrenal gland, became of *no little* importance to us at Ray Brook and endocrinology became a fascination.

THE RAY BROOK COCKTAIL

Before the discovery of a specific anti-tuberculosis drug, there were alleged palliative procedures and measures. Cod liver oil was an old remedy going back as far as the 1770s to Brompton Hospital in London. In America, Austin Flint[12] espoused it in his *A Treatise on the Principles and Practice of Medicine*. William Osler wrote,

> In glandular and bone tuberculosis this remedy [cod liver oil] is undoubtedly beneficial in improving the nutrition. In pulmonary tuberculosis its action is less certain. . . . For the diarrhea [from intestinal tuberculosis] large doses of bismuth combined with Dover's powder [Thomas Dover, 1742, powder of ipecac and opium] and small starch enemata, with or without opium, may be given. The acetate of lead and opium pill often acts promptly, and the acid diarrhea mixture, dilute acid, morphia and acetate of lead may be tried.[25(p256)]

Mack McConkey (Fig 5) (Harvard, 1925) developed what became known as the "Ray Brook cocktail": cod liver oil (CLO) floated on tomato juice (TJ). He reported 50 cases treated with this cocktail, 50 with heliotherapy, and 28 controls.[50] Other articles record his experience in the treatment of laryngeal and intestinal tuberculosis.[51,52] Flint advocated judicious use of alcohol as well as cod liver oil, but use of alcohol at Ray Brook was restricted as reported by a former staff member: "Miss Cleland told me of her early days as a nurse. She was placed in charge of the whiskey supply and kept it locked up. Sometimes whiskey was used as an 'appetizer', 'stimulant', and 'cough remedy'. I never once ordered it." Reporting on the

FIGURE 5. Mack McConkey, MD, assistant director of Ray Brook (photographed by Joseph M. Kurung, collection of James Monroe, MD.)

effectiveness of cod liver oil, one former patient stated, "I had a Gaffkey X (the highest rating for positive sputum). I never missed taking C L O and T J. It was the old fashion C L O, horrible testing, with tomato juice to help it go down, served in a wine glass inscribed with my initials, placed in a pantry next the dining room, and records kept. I'm still thanking Mack McConkey."

McConkey[53] studied bronchial stenosis, for which he coined the aphorism, "all that wheezes is not asthma." At least one Ray Brook patient with tuberculous bronchial stenosis is still living.

Part of McConkey's work was done at Ray Brook House, a "cure cottage" half a mile away. Patients with advanced tuberculosis could be admitted, and their care was paid for by their county of residence. Some of the patients had intestinal tuberculosis. Ulcerative ileocecal tuberculosis seldom occurred except in a patient with a highly positive sputum. There was as yet no effective treatment for anyone suffering with the severe cramps, painful diarrhea, and anorexia except palliative morphine, Dover's powders, warm compresses, and soothing enemata. Here McConkey found his cocktail helpful for some patients with laryngeal and intestinal tuberculosis. (Eventually, McConkey's health failed due to recurrent pulmonary tuberculosis.)

SURGERY AND SPECIFIC ANTI-TUBERCULOSIS THERAPY

The first Ray Brook budget with specific allocations for surgery was in 1937. Saranac Lake surgeons were consultants at Ray Brook beginning in 1926, according to the annual report of that year. When J. Maxwell Chamberlain, who had trained under John Alexander of Ann Arbor, Michigan, joined the staff in 1937 as the first resident surgeon, he divided his time between Ray Brook and Homer Folks Tuberculosis hospitals. Ethan Flag Butler was the thoracic surgeon for Biggs Memorial and Mt Morris tuberculosis hospitals. Joseph Gordon and David Pecora followed Chamberlain at Ray Brook. Anesthesia was administered by physicians from Saranac Lake or Sunmount Veterans Hospital at Tupper Lake, unless a recuperating doctor at Ray Brook was capable.

Bronchoscopy done to determine sites of bronchial stenosis was reported by McConkey and Gordon.[54] Bronchoscopy done to localize sources of positive sputum was reported by Pecora and Yegian.[55] Differential bronchospirometry was done prior to resectional pulmonary surgery. White, Beck, and Pecora[56] reported 15 cases with coexisting lung cancer and tuberculosis.

The long search for an effective anti-tuberculosis drug culminated in 1944 with the discovery of streptomycin. The capacity of certain saprophytic organisms to inhibit growth of *Mycobacterium tuberculosis* had long been recognized. Experiments with numerous chemicals and biological extracts had been reported, among which was sodium-gold-thiosulfate (Sancrysin).[57,58] Wallace and Bray[59] at Ray Brook experimented with *Lactobacillus pentaceticus* and *Bacillus macerans* and found the free and combined carbohydrates of virulent tubercle bacilli (strain H 37) to be destroyed by a product obtained from these naturally occurring bacteria. Sulfapyridine,[60] sulfanilamide,[61] and promizole, the latter used by Edith Lincoln at Bellevue Hospital on the pediatrics service for tuberculous meningitis,[62] were studied.

In April 1944, Feldman and Hinshaw in Minnesota were furnished with a small supply of streptomycin by Selman A. Waksman of Rutgers University.[63,64] Collaborative studies by Yegian (Ray Brook) and Middlebrook (Rockefeller Institute) and reported jointly by them, confirmed the bacteriostatic effect of streptomycin.[65] Resistance studies were done by Yegian and Vanderlinde at Ray Brook.[66] The next anti-tuberculosis drug was dihydrostreptomycin.[67] The five-year follow-up was reported by the New York State Tuberculosis Hospitals in 1955.[68] One Ray Brook patient included in the study suffered "marked dysphagia due to pharyngeal tuberculosis complicating far advanced pulmonary disease. Less than 24 hours after the first dose of streptomycin injection he stated he 'could swallow without the marked pain.' He made a successful recovery." Monroe, senior author of the five-year follow-up report, achieved almost 100% follow-up for Ray Brook patients.

RAY BROOK STATE TUBERCULOSIS HOSPITAL CLOSES

On June 30, 1971, 67 years after its opening, Ray Brook Hospital closed and was partly turned into a noncriminal women's drug rehabilitation center. The task of discharging the last patients to their homes or transferring them to Homer Folks or Mt Morris State Tuberculosis hospital or to general hospitals fell to Dr Bela O. Duboczy and the remaining staff. Field clinics for Clinton, Essex, Franklin, Hamilton, and St Lawrence counties were continued. Duboczy established the central clinic at New York State Sunmount Developmental Center, formerly the Sunmount Veterans Hospital at Tupper Lake, and he and the staff operated field clinics at Ausable Forks, Canton, Gouverneur, Indian Lake, Keeseville, Malone, Massena, Ogdensburg, Plattsburg, Potsdam, Port Henry, Saranac Lake, Star Lake, and Ticonderoga.

It became possible for the New York State Health Department to set in motion a program to close the remaining state tuberculosis clinics, and to shift the tuberculosis control program to county health departments. This was not to say that tuberculosis had been entirely eradicated. During 1974, in the Tupper Lake area, 30 new cases were found, among them three school teachers and eight students with active tuberculosis. During the same year, more than 300 patients were being treated in the field clinics. The last clinic session was held June 20, and the official closing date was June 30, 1975, 71 years after the opening of the New York Hospital for the Treatment of Incipient Tuberculosis.[69]

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FROM THE LIBRARY

OBSERVATIONS ON AMERICAN CLIMATES AND LOCALITIES IN THE TREATMENT OF PULMONARY TUBERCULOSIS

There is reason to believe that the scourge of pulmonary tuberculosis will eventually be mastered by the science of man. Being a specific infectious disease, the nature and causes of which are adequately understood, it requires no great strain of the imagination to foresee a time not many generations hence when this most virulent enemy to human life, will to a great extent, be eliminated as an important factor in mortality statistics. It is well known that the disease is preventable, and morbidity tables, in not a few localities, have already been perceptibly influenced by the sanitary measures put in operation as a result of knowledge acquired within a few years. For a very considerable period yet to come, however, consumption will continue to rank as the most effectual weapon of the Grim Destroyer, and the ratio of about one death from this disease to seven who are removed from earthly joys and sorrows by all causes will not be materially altered until we can bring entire batteries to bear where as yet only occasional random shots are heard. If it became known that we were about to engage in a war with a great foreign enemy which would probably result in the sacrifice of the lives of 100,000, 50,000, or even 25,000, of our fellow-citizens, how quickly would our country spring to the call to arms. The great patriotic breast of America would thrill with heroic determination and resolve, and not a hamlet in our broad land that would not yield up its best and bravest in the national defense. Yet we are engaged in a perpetual war with an enemy far more deadly and implacable than any foe which may come against us with ships and soldiers and guns. At the current death-rate from pulmonary consumption, more than 10,500,000 of the present population of the United States must succumb to that disease. The paramount importance of this subject is, therefore, apparent to all. We know the enemy. We are acquainted with his origin, with his methods, and with his manifestations, and we are also fully cognizant of a well-tested and successful, yet withal, expensive means of operating against him. The important question is, do we possess sufficient good sense, and sufficient love for our fellow creatures to employ our knowledge to the best advantage? Prevention is our chief reliance for the future, but let us here consider the great problem; How can we best combat the progress of consumption after it has actually manifested its symptoms and signs in the patient's economy?

It is the writer's absolute conviction that some form of climatic treatment, with its accessory circumstances, offers far more hope than all other methods of treatment combined. In an experience of more than twenty years the writer has not known of a single recovery from undoubted tuberculosis in which a change of surroundings did not constitute an important factor. The patient should, as speedily as possible, be removed from the locality in which the disease was inaugurated. The appearance of phthisis is in itself an evidence that there is something wrong about that particular atmosphere for the patient in question, and if he remains under the same environment he is almost sure to get worse. . . .

JAMES K. CROOK, MD

(*Trans Med Soc State of NY* 1903; 173–183)

Magazines that do not accept tobacco advertising

In December 1983, the Journal published a listing of magazines that did not accept cigarette advertising (Richards JW Jr: A positive health strategy for the office waiting room. NY State J Med *1983; 83:1358–1360). At the House of Delegates of the Medical Society of the State of New York in March 1990, a resolution (90-24) was passed requesting that a list of magazines that have voluntarily chosen to decline tobacco ads be published in the Journal. The resolution also encouraged physicians to substitute magazines without tobacco ads in their office reception areas. The following list was compiled by the American Medical Association and published in* JAMA *(1989; 262:1290–1291, 1295), and is reprinted with permission of the AMA.*

Adirondack Life
Air & Space
Alaska Magazine
American Baby
American Health
American Heritage
American History Illustrated
Animal Kingdom
Arizona Highways
Audubon
Aviation Week & Space Technology
Backpacker
Bicycling
Boy's Life
Business Week
Consumer Reports
Cyclist
Dance Magazine
Down East Magazine
Farm Journal
Fishing Facts
The Futurist
Golf Illustrated
Good Housekeeping
Hadassah Magazine
Harvard Business Review
Harvard Medical School Health Letter
Health
Highlights for Children
Historic Preservation
Horticulture
Humpty Dumpty's Magazine
International Travel News
Isaac Asimov's Science Fiction
Jack and Jill
MAD Magazine
Maine Fish & Wildlife
Maine Life Magazine
Mayo Clinic Health Letter
Model Railroader
Modern Maturity
Montana Magazine
Mother Earth News
Mother Jones
Nation
National Geographic
National Parks Journal
Nation's Business
Natural History
The New Yorker
North American Review
Nutrition Action Healthletter
Oceans
Old House Journal
Organic Gardening
Parenting
Parents Magazine
Personal Computing
Petersen's Hunting
Popular Communications
Prevention
Railfan and Railroad
Ranger Rick
Reader's Digest
Runner's World
Sail
Saturday Evening Post
Science
Science News
The Sciences
Scientific American
Sesame Street
Seventeen
Sierra
Smithsonian
Sports Afield
Stork
Sunset Magazine
Theatre Crafts
Travel Holiday
Utah Holiday
Vegetarian Times
Venture Magazine
Vermont Life
The Washington Monthly
Western Outdoors
Writer's Digest
Yankee

Infected aortitis masquerading as bronchogenic carcinoma

Linda J. Paradowski, md; Ivan Hajdu, md, PhD; Lorraine Coli, md; Gregory Loewen, do

Aortobronchial fistula is an uncommon and usually fatal complication of thoracic aortic aneurysm, often diagnosed on postmortem examination. A case of an infected aortitis with thinning of the descending thoracic aorta complicated by aortobronchial fistula and rupture is described, with a brief review of the literature.

Case Report

A 72-year-old man was admitted to the Buffalo Veterans' Administration Medical Center in December 1987 with nonspecific complaints including generalized malaise and occasional night sweats. His medical history was remarkable for a remote right hemispheric stroke. He was a smoker. On physical examination, the patient was febrile (102°F/38.8°C) and was normotensive. He produced blood-streaked sputum but did not appear to be in respiratory distress. A mild central left facial droop was present, with paresis of the left arm. The lungs revealed diminished breath sounds in the left base. The cardiac examination was normal and peripheral pulses were not diminished. The blood pressure was equal in both arms. The rest of the physical examination was noncontributory.

Laboratory studies disclosed a leukocyte count of 13K without a left shift; the patient was not anemic. A chest radiograph revealed a left hilar mass with volume loss of the left lower lobe (Fig 1). The electrocardiogram (EKG) was normal.

Bronchoscopy revealed a blunted carina, patent left upper lobe, and a large amount of blood obscuring the left lower lobe. No biopsies were performed, and bronchial washes revealed scant inflammatory cells. Gram stain and culture of the washes were negative for bacterial growth. Subsequent computed tomography (CT) of the chest revealed a posterior mediastinal soft tissue mass contiguous with the descending thoracic aorta. Aortography (Fig 2) confirmed the presence of a large saccular aneurysm of the descending thoracic aorta.

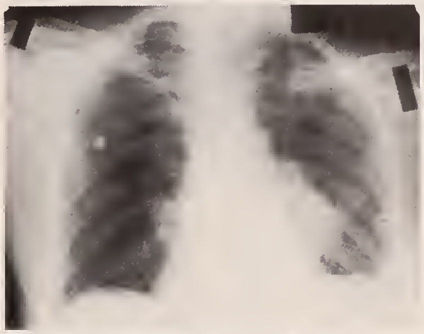

FIGURE 1. Posteroanterior chest film showing large left lingular mass with volume lost.

The patient was taken to the operating room, and the aneurysm was resected under cardiac bypass. During the surgery, the aneurysm was found to have eroded into the parenchyma of the left lower lobe of the lung. A left lower lobe lobectomy was performed, and a dacron graft was interpositioned in the line of resection.

Histologic studies revealed a focally thinned aortic wall with mild to moderate atherosclerosis and focal acute aortitis with purulent inflammation of overlying plaques (Fig 3). The site of rupture occurred in one of these areas, with hemorrhagic necrosis of adjacent lung tissue. Small aggregates of gram-positive cocci were scattered throughout the superficial and deep media (Fig 4). No signs of cystic medial necrosis or luetic aortitis were identified. Cultures of corresponding tissues did not grow any bacteria.

After surgery, the patient's condition gradually improved; he received a 14-day course of broad-spectrum antibiotics. Eventually he was discharged from the hospital.

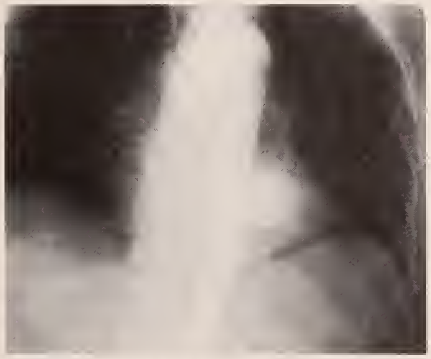

FIGURE 2. Aortography demonstrating a saccular aneurysm of the thoracic aorta.

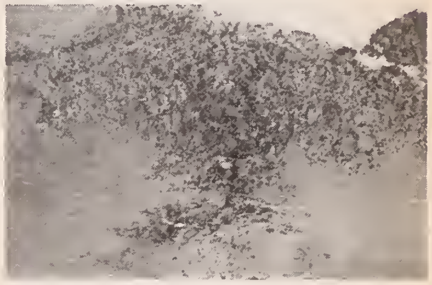

FIGURE 3. Histologic sections of the aortic wall demonstrating focal acute aortitis with atherosclerotic plaques.

He returned to the hospital four months later with fever and hemoptysis. He suffered massive hemoptysis and died on the day of admission. Permission for a postmortem examination was refused.

Discussion

Suppurative infections of the aorta may arise from septic microemboli (eg, infectious endocarditis), local spread of adjacent infected structures (eg, vertebral osteomyelitis), hematogenous seeding during bacteremia, and from trauma or direct iatrogenic contamination.[1]

Worrell et al[2] suggested that a secondarily infected, established aneurysm, the "mycotic aneurysm," should be differentiated from an infected aortitis which may subsequently progress to a pseudoaneurysm with eventual rupture. In the latter case, a normal or atherosclerotic blood vessel is infected during bacteremia. Interestingly, Worrell et al[2] observed that the aortic media may provide protection to the bacteria from the inflammatory response. We have corroborated this finding in the pathologic specimen of this patient, which showed obvious organisms between the elastic lamellae and a paucity of neutrophils (Fig 4). Although permission for an autopsy was refused, we can conclude from the surgical specimen that the patient's aneurysm arose from an infectious aortitis and was not a true "mycotic" aneurysm. Unfortunately, most of the literature fails to

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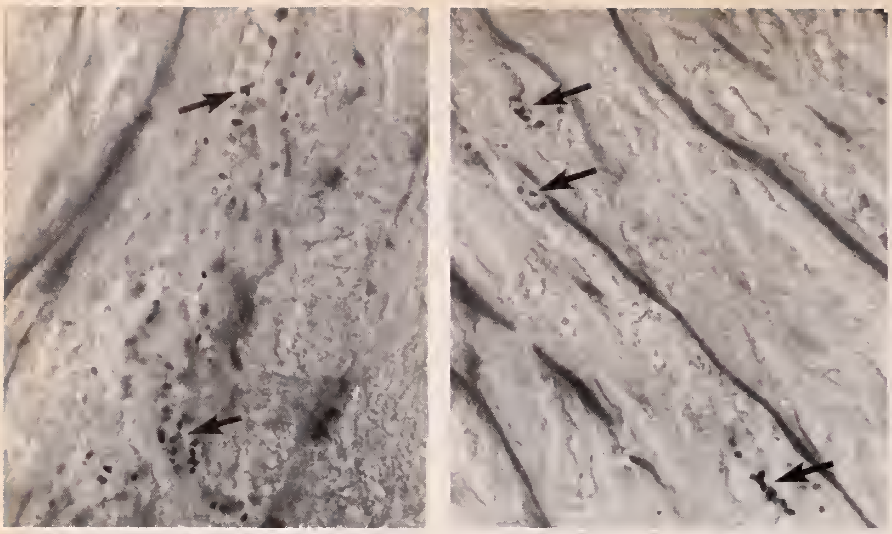

FIGURE 4. Aggregates of gram-positive cocci were scattered throughout the aortic media.

make this pathologic distinction.

Most patients with an infected aneurysm have an insidious course which delays diagnosis until catastrophic rupture of the aneurysm.[3] Aneurysmal involvement of the lung is not particularly common. In two reviews of a total of 20 thoracic aneurysms (infected and bland atherosclerotic) with aortobronchial fistula, only seven patients survived the operative repair of this complication. Two of these patients died of massive hemoptysis following aortic resection. The aortobronchial communication almost always occurs with the left lung and most commonly with the left lower lobe.[4,5] It is not clear if an infected aneurysm predisposes more to this complication than does a bland atherosclerotic, thoracic aortic aneurysm. McNamara and Pressler[6] do mention the low incidence of airway compression in their review of patients with atherosclerotic thoracic aneurysms that arise distal to the left subclavian artery.

The bacterial agents that cause infected aortitis differ markedly according to the cause of the infection.[7-9] Postoperative survival is significantly improved if gram-positive cocci are found, as compared to gram-negative bacilli.[1,8] Patel and Johnston[9] suggest that infected aneurysms from a variety of sources should be treated with preoperative, intraoperative, and at least six weeks of postoperative antibiotics. This patient received antibiotics for two weeks after surgery, and his initial hospital course suggested that this treatment was adequate. However, four months later he died of massive hemoptysis, most likely from a catastrophic rupture of an aortic suture line into a bronchus.

Successful outcomes in cases of infected aortitis are predicated on both adequate surgical resection of all aneurysmal inflammatory tissue and sterilization with a prolonged course of bacterial antibiotics.[8] Since autopsy was not obtained, it is impossible to comment on the viability of the graft site, but we must consider that this patient's course of antibiotics may have been too brief.

This case illustrates the high index of suspicion necessary to make the diagnosis of infectious aortitis, since the presentation can be quite insidious. It also emphasizes the aggressive surgical and medical treatment necessary to avoid catastrophic consequences.

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Total pancreatectomy with celiac artery occlusion

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Interruption of the hepatic arterial blood supply during the course of pancreatic resection in a jaundiced patient with hepatic insufficiency usually results in fatal hepatic necrosis. Hepatic arterial anatomy is "normal" in only 55% of all people[1,2] and identification of variant vasculature by preoperative angiography is often recommended in patients for whom major pancreatic resection is planned.[3] Information concerning hepatic vascular anatomy is not, however, always available to the surgeon. This report describes a case of total pancreatectomy for carcinoma of the pancreatic head performed with no knowledge of the hepatic vascular anatomy. An unanticipated arteriosclerotic occlusion of the celiac axis was identified and successfully managed.

Case Report

A 71-year-old man was transferred to New York University Hospital. He gave a two-week history of jaundice. The patient had had insulin dependent diabetes for 14 years and hypertension for ten years. The

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only significant finding on physical examination was the presence of deep jaundice.

Computed tomography (CT) of the abdomen (Fig 1) demonstrated a large, 4-cm mass in the head of the pancreas with high grade obstruction of the common bile duct and pancreatic duct. The lesion deviated and distorted adjacent major vascular structures (superior mesenteric artery and portal vein). Preoperative visceral angiography was not obtained, because it was thought that, on the basis of the CT findings, there was a low probability that the lesion was resectable.

At laparotomy, a 4-cm mass was confirmed in the head of the pancreas with dilation of the biliary and pancreatic ductal systems. There was no evidence of nodal or hepatic metastatic disease, and the superior mesenteric and portal veins were uninvolved. Several smooth, 1–1.5-cm cysts were present in the body and tail of the pancreas. A decision was made to proceed with pancreatic resection. Because of the cysts in the pancreatic tail and the preexisting diabetes, a total pancreatectomy was performed. The splenic vessels and spleen were sacrificed due to uncertainty about the nature of the pancreatic cysts.

Since a preoperative angiogram had not been obtained, the anatomy of the hepatic artery was examined; the artery appeared to be arising "normally" from the celiac axis. Furthermore, the hepatic artery was palpated during test occlusion of the gastroduodenal artery, and there was no apparent diminution of the bounding hepatic arterial pulse. During the course of the resection, a large, 3–4-mm artery was noted entering the body of the pancreas from the superior mesenteric artery. On completion of the resection, no pulse was present in the hepatic, splenic, or left gastric vessels. Further examination revealed an arteriosclerotic plaque in the celiac axis. A nonreversed, saphenous vein graft with valves excised was placed from the infrarenal aorta to the common hepatic artery. Excellent pulses were restored to the hepatic, left gastric, and splenic arterial stump, and the gastrointestinal anastomoses were then performed.

The patient's postoperative course was uncomplicated. A postoperative arteriogram (Fig 2) demonstrated a patent graft.

DISCUSSION

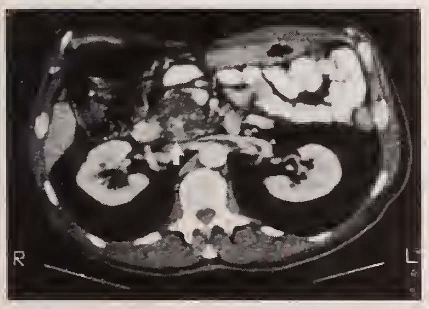

FIGURE 1. Computerized tomography of the abdomen demonstrating large mass (arrow) in the head of the pancreas.

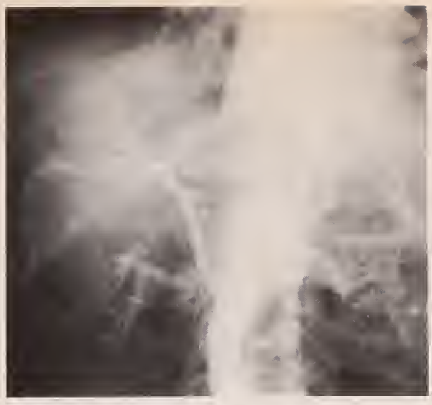

FIGURE 2. Postoperative aortogram demonstrating patent aorto-hepatic artery bypass graft.

Interruption of the hepatic arterial inflow is a known and often lethal complication of pancreatoduodenectomy. Two factors that may contribute to this complication are the variability of the hepatic arterial anatomy and the occurrence of occlusive vascular disease.

The entire principal hepatic arterial supply derives from a common hepatic artery that arises from the celiac axis in only 55% of people.[2] In several variations, such as a left hepatic artery arising from the left gastric artery, there is little risk of hepatic devascularization during pancreatic resection. However, the major right hepatic artery or an accessory right hepatic artery arises from the superior mesenteric artery in 18% of individuals, and the common hepatic artery arises from the superior mesenteric artery in 4.5%.[2] These vessels usually run posterior to the pancreas and they can, in most instances, be preserved during pancreatoduodenectomy. Unless their presence is recognized, however, they may be inadvertently occluded.

The frequency of arteriosclerotic occlusive disease affecting the hepatic arterial supply has been reviewed via angiographic studies.[4,5] Sclerotic lesions of the celiac vessels were found in 12% to 44% of patients examined, with 0.7% having celiac occlusion.[4] In most cases, collateral blood flow to the celiac vessels is maintained though the pancreatoduodenal arcades (Fig 3). Clearly, these vessels are sacrificed during pancreatoduodenectomy, and hepatic ischemia may result. Occasionally, as in the present patient, arterial inflow to the celiac vessels is maintained by collateral arteries from the superior mesenteric artery to the splenic artery. Such vessels include the transverse pancreatic artery, the superior pancreatic artery, and the great pancreatic artery.

When major pancreatic resection is anticipated, preoperative angiography to identify variations in arterial anatomy facilitates planning of the operative procedure. Knowledge of arterial anatomy is not, however, always available in patients undergoing resection. This may be because resection is not anticipated, as in the present patient, or because of hypersensitivity to contrast media, or other factors.

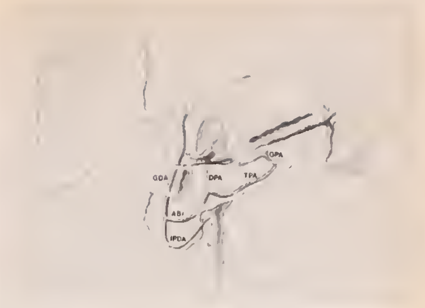

FIGURE 3. Diagramatic illustration of the collateral blood supply between the superior mesenteric artery and the splanchic artery. (ABr, anastomotic branch; DPA, dorsal pancreatic artery; GPA, great pancreatic artery; TPA, transverse pancreatic artery; GDA, gastroduodenal artery; IPDA, inferior pancreaticoduodenal artery).

When pancreatic resection is performed without preoperative angiography, the anatomy of the celiac and superior mesenteric arterial vessels must be determined. Congenital anatomic variants, such as a right hepatic artery arising from the superior mesenteric artery, usually are easily identified. Obstruction of the hepatic or celiac vessels by arteriosclerotic disease or a median arcuate ligament may be more difficult to establish.[6] Since collateral arterial flow to the celiac vessels is usually maintained through the pancreatoduodenal arcades and gastroduodenal artery, the integrity of the hepatic arterial pulsation should be evaluated after temporary occlusion of the gastroduodenal artery and before deciding on pancreatic resection. In the present patient, hepatic arterial pulsation was well maintained during gastroduodenal artery occlusion. During pancreatic resection, a large vessel arising from the superior mesenteric artery was noted. This was probably the transverse pancreatic artery. After pancreatic resection, including sacrifice of the splenic artery, pulsations were absent in the hepatic, left gastric, and celiac vessels. Arterial inflow to these vessels was restored using a vein graft from the aorta to the hepatic artery. The patient's recovery was uncomplicated.

A small number of cases have been previously reported in which celiac revascularization has been undertaken in conjunction with pancreatic resection. In these cases, the need for vascular reconstruction was always identified by preoperative angiography.

Miyata et al[7] reported on a patient in whom celiac occlusive disease was associated with collateral flow to the liver through the gastroduodenal artery. Pancreatoduodenectomy was undertaken with celiac arterial revascularization using a vein graft from the aorta to the hepatic artery. Two similar cases were reported by Thompson et al,[8] who restored celiac arterial inflow by implanting the proximal splenic artery into the superior mesenteric artery. Fortner et al[6] described two patients in whom celiac axis occlusion was identified by preoperative angiography and relieved by division of the median arcuate ligament.

The present report differs from the previously described cases in that angiography was not obtained prior to surgery. In this circumstance, if arterial anatomy appears normal and celiac occlusion is suspected, test occlusion of the gastroduodenal artery and splenic artery will identify most patients in whom celiac axis inflow is arising by collateral pathways from the superior mesenteric artery. These cases, however, clearly emphasize the importance of determining the integrity of hepatic and other arterial flow at the conclusion of every major pancreatic resection. Failure to identify and correct interruption of arterial flow to the celiac vessels may result in postoperative hepatic necrosis.

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Spontaneous renal hemorrhage secondary to choriocarcinoma

ROBERT M. WASHECKA, MD; WILLIAM MANN, MD; ROSEMARIE ZUNA, MD

Spontaneous renal hemorrhage (SRH) has diverse causes, including occult malignancy. Tumor is the underlying cause of spontaneous renal hemorrhage in about 60% of cases; a malignant neoplasm is the cause in a third of all patients.[1–3] Choriocarcinoma, a manifestation of gestational trophoblastic disease, is well recognized by its hemorrhagic complications at metastatic sites (choriocarcinoma syndrome).[4] Renal metastasis can be an initial presentation of the disease. A patient with SRH secondary to metastatic trophoblastic disease is presented with a review of treatment options.

CASE REPORT

A 27-year-old woman presented with severe pain in the left flank, gross hematuria, and diaphoresis for three days. Physical examination revealed a tender, boggy mass in the left flank associated with guarding. The patient became hypotensive and was given six units of packed cells. A computed tomographic (CT) scan of the chest and abdomen revealed a left pulmonary nodule as well as a large left perinephric hemorrhage (Fig 1).

At exploration, there was a large perinephric hematoma and a 3 × 4-cm defect in the anterior wall of the left kidney. Left nephrectomy was performed due to the massive amount of bleeding. Histologic examination revealed choriocarcinoma (Fig 2). The serum beta human chorionic gonadotropin (HCG) level was 20,270 units after surgery. Uterine curettage revealed an old implantation site without evidence of malignancy. Metastatic evaluation showed multiple pulmonary nodules bilaterally consistent with disseminated tumor. The patient had an excellent response to chemotherapy, with clearing of pulmonary metastases and return of beta HCG titer to normal. However, compliance was poor and the disease ultimately recurred; two and a half years after the diagnosis, the beta HCG titer was elevated and the patient died, despite intensive chemotherapy, thoracotomy with wedge resections, and hysterectomy.

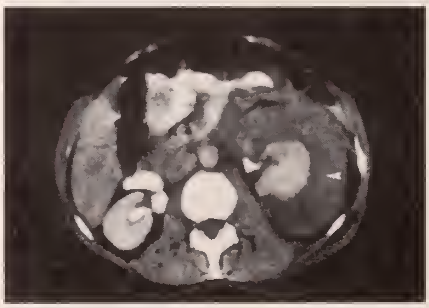

FIGURE 1. Computed tomography of abdomen reveals a large amount of irregular heterogenous opacity in the perinephric space consistent with hemorrhage; the superior pole of the left kidney is ruptured—consistent with a ruptured kidney with hemorrhage and dissection about the kidney and spleen.

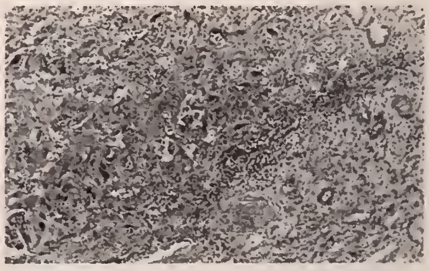

FIGURE 2. Microscopic section of kidney revealing a focus of syncytotrophoblasts and cytotrophoblasts in the renal parenchyma surrounded by several sclerotic glomeruli (hematoxylin-eosin stain, original magnification × 200).

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DISCUSSION

Renal metastases have been found in up to 48% of patients with choriocarcinoma at autopsy.[5] Since the development of effective chemotherapy, the apparent incidence of such metastases has declined to less than 5%, ranging from 1% to 20%. In one series, renal metastases were identified in 1.3% of 154 women presenting for primary therapy and 14% of 42 referred for salvage treatment for metastatic gestational disease.[6]

Renal manifestations including a mass, flank pain, hematuria, and spontaneous hemorrhage have been observed in patients in several series.[5,6] It is important to note that all cases of renal metastases demonstrated coexistent or antecedent radiographic evidence of pulmonary metastases.[6-8] Patients with high risk gestational trophoblastic disease require aggressive treatment involving chemotherapy with multiple agents, selective surgery, and radiotherapy, since approximately 60% of these patients can be effectively treated. Accordingly, nephrectomy is indicated in patients with unilateral renal involvement and limited pulmonary metastases. However, it may not affect survival in patients with extensive metastatic disease. The role of radiation in renal metastasis is unclear, but has been unsuccessful in combination with chemotherapy in one patient.[6]

In summary, female patients who present with spontaneous renal hemorrhage and pulmonary nodules may have metastatic gestational trophoblastic disease. Such patients require careful evaluation during chemotherapy, including computed tomography of the abdomen with attention directed at the kidneys to monitor for possible severe hemorrhagic complications of the disease. Patients with renal metastases who are resistant to chemotherapy may require selective use of surgery, such as nephrectomy, to achieve remission.

Acknowledgment. The authors thank Dr Gerald Ryan of Patchogue, NY, who provided histologic sections for review during the course of this patient's management.

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The need for medical libraries in hospitals

TO THE EDITOR: I was somewhat distressed to read the recent commentary by Dr Peter Farnsworth[1] regarding the state's recent proposal to eliminate funding for library services. As I understand it, the state's contention is that there is no documented evidence that library services have a beneficial effect on the care of patients. I would, therefore, like to provide some documentation from my personal experience.

Case 1: Thrombotic thrombocytopenic purpura. While working in a rural hospital, I had the opportunity to see a patient who had post-transfusion purpura. In reading about this diagnosis through the assistance of the library, I obtained articles on other unusual causes of thrombocytopenia. Among these were several articles on thrombotic thrombocytopenic purpura. Four years later, while at a major university hospital in another state, I saw a patient on hematology rounds with the classic symptoms of thrombocytopenic purpura. At that point, knowing that this was a fatal disease, I checked my diagnosis and asked the librarian to do an emergency literature search for me. Articles and abstracts were obtained showing that within the last year the life saving treatment of plasmapheresis or plasma infusion had been described. The patient was given plasma infusions and six months later was still alive. Subsequently, several other patients with that syndrome were seen at that same institution.

Case 2: Diagnosis of hemachromatosis. While at a major university medical center, I had the opportunity to see and read about several patients with hemachromatosis. Five years later, in private practice in upstate New York, I saw a patient with continued elevations of liver function studies without any other obvious abnormality. His family history was significant for heart failure and diabetes. Therefore, serum ferritin was ordered and the diagnosis of hemachromatosis was made. Subsequently, several other patients in our town were diagnosed with hemachromatosis, and are presently receiving appropriate therapy. This includes routine therapeutic phlebotomies which should decrease their risk of hepatoma, heart failure, and diabetes.

Case 3: Diagnosis of pheochromocytoma. While in a university hospital, I had the opportunity to see a patient with pheochromocytoma. After reading several articles about this, I determined that patients with hypertension and significant postural drops were probably at increased risk for pheochromocytoma. Approximately three years later, I saw a patient in my private practice who had hypertension and a significant postural drop, as well as a 20-lb weight loss. She had no other symptoms; no night sweats or tachycardia. However, urine catecholamines were ordered. A diagnosis of pheochromocytoma was made and the patient was appropriately treated with operative intervention.

Case 4: Diagnosis of traumatic injury. Due to my interest in automotive safety, in 1985 I did extensive reading through the services of Auburn Hospital on the subject of seat belts. Following this, I saw a young woman in my office who told me that she did not believe in seat belts. Using the evidence I had accrued from library sources, I convinced this young woman that she should wear her seat belt. One week later, she was in a car accident which by verbal report exactly duplicated a car accident which had occurred to another woman one week prior to that time. In the earlier accident, the young woman was ejected and died. My patient had decided to wear her seat belt and suffered a broken arm but was otherwise unharmed.

Case 5: A problem of differential diagnosis. This year, while taking care of a patient with a very complicated, undiagnosed neurodegenerative disorder, we found ourselves having great difficulty controlling her seizures due to multiple drug side effects or poor efficacy of several other agents, including phenobarbital, valproic acid, and carbamazepine. The patient's seizures were finally controlled with phenytoin (Dilantin). However, liver function tests were abnormal, and there was a question as to whether or not the phenytoin had caused the patient's hepatotoxicity. A literature search was performed, and after review of the appropriate papers, I concluded that phenytoin hepatotoxicity was unlikely. Therefore, due to the great risk which recurrent grand mal seizures caused to this patient, she was placed back on phenytoin. Liver biopsy has been delayed due to coagulopathy, but an appropriate workup is now in progress, and the patient is neurologically stable.

Case 6: Diagnosis of phenylketonuria (PKU) in pregnancy. While I was in my upstate practice, a patient came in as one of the first patients diagnosed with PKU. She had made the decision, based on her diagnosis, that she could not have children. Through a literature search, we were able to determine that with proper diet she would be able to have a child safely and this information was relayed to her. In addition, she was referred to appropriate agencies for follow-up.

Other literature searches which have been important in my practice and which have significantly affected patient care included the effects of high dose vitamin D, the side effects of lithium, the proper treatment of panic disorder, the diagnosis and treatment of hypotension secondary to right ventricular myocardial infarction, the use of fats during hyperalimentation in a patient with acute pancreatitis, the proper use of electrocardiograms (EKGs) prior to surgery, and proper treatment of decubiti ulcers.

In addition, I find that physicians frequently share their conclusions from the results of their literature searches with one another and with other health professionals. I have also obtained copies of articles from other health professionals which have assisted me in my practice. For instance, recently a respiratory therapist gave me a copy of an article describing the use of positive pressure support in weaning a patient from chronic use of a ventilator.

Other services which the library has provided include access to books, through inter-library loan as well as our own collections. I have used these books to prepare educational EKG conferences, to review issues related to malpractice, and to review the proper evaluation and treatment of reno-vascular hypertension, and I am presently reading books on multiple personality disorder.

Several conclusions may be drawn. The availability of library services has been important in one physician's care of patients. It is unlikely that my experience is significantly different from that of other physicians and health personnel who make use of library facilities. If a study is to be done documenting the effect of library services on improvements in patient care, the study would have to evaluate long term effects. This should include the effects of information obtained in one literature search and subsequently used to make a significant diagnosis in another patient, possibly several years later.

There is no funding mechanism presently available for providing these services. I do not charge my patients for any of these services, including use of my own computer time in some instances, or use of my time (which may be several hours) to review the appropriate literature. The total cost of these services for each patient saved from a life threatening illness seems intuitively likely to be within range of other medical interventions such as vaccines, although this hypothesis could be tested.

Given the very obvious improvement of my care to patients and the decrease in morbidity due to the availability of library services, it seems likely that the services could be documented to be of importance to other patients. It may be worthwhile for individual physicians to report their experiences to provide preliminary documentation prior to the time that an adequately designed and controlled study can be undertaken.

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1. Farnsworth PB: The elimination of the requirement for medical libraries in hospitals. *NY State J Med* 1989; 89:601-602.

Impotence: A single symptom with different causes

TO THE EDITOR: Although Dr Zorgniotti's article, "Vascular insufficiency: A significant factor in impotence,"[1] was a clear presentation of his conceptualization and treatment of impotence, there are a few points that need to be clarified in an overall approach to diagnosis and treatment.

The patient population described in the Zorgniotti article is similar to the patient population 45 years of age and older seen at the Columbia Presbyterian Medical Center, some of whom were beginning to show the effects of the aging process, ie, vascular complications, diabetes, neuropathy, etc. However, more than 60% of our patients were younger and after a complete medical workup showed no evidence of physical impairment.[2] Some of this group suffered from stress-related impotence due to divorce, death, financial loss, etc. These patients generally recovered with time as the stressful situation resolved itself. Another separate group included males who had a history of transitory impotence or premature ejaculation. They were generally referred for psychological treatment.[3]

It is doubtful that any understanding of psychogenic impotence was ever entirely based on the "Oedipal complex." There is little question that impotence and anxiety and or depression occur together with a great degree of frequency in all age groups. The concept of psychogenic symptoms today has to include not only a previous experience, which can be symbolic, but also the knowledge of central and peripheral neural mechanisms which are chemically mediated. The role of an intact endocrine system in both sexual development and expression is universally accepted, but it is only recently, with increased knowledge and more precise measurements, that we are beginning to understand the underlying reactions. The more we learn about these factors, the less of a need to speculate on causation.

In 1972 we reported on the effects of psychotherapy and psychoanalysis in the treatment of sexual disorders.[4] The overall improvement rate was 66% and, in impotency, an improvement rate of 57% was seen. The short term behavioral treatment of Masters and Johnson[5] in St Louis resulted in an initial failure rate of 29% and 40% failure rate in impotency. Masters and Johnson chose to report the results of treatment in terms of a failure rate rather than improvement or success rate for reasons still unknown. Other investigators in the field reported the results of treatment as either success or failure. Columbia Presbyterian Medical Center[6] and Johns Hopkins University[7] reported improvement rates between 60% and 80% in their short term therapy programs.

In the male population, there is a gradual increase in the occurrence of impotence as age increases. At 30 years there is a 0.8% expectancy of impotence while at 60 years the expectancy rate rises to 18.4% of the population.[8] Since the study was demographic, the authors did not attempt to ascertain etiologic factors.

Most of the articles in the medical literature on male impotence come from clinical specialty settings or private practices where patients previously cleared by generalists or internists are referred to endocrinologists, psychiatrists, or urologists. This presents us with a screened and preselected group. While the articles are important, too often they reflect the disciplines of the authors, be they endocrinologists, psychiatrists, or urologists. With this in mind, Dr Zorgniotti's statement that "The etiology of impotence long thought to be psychogenic is now believed to be mainly vascular"[1] is no more valid than to say it is "mainly psychogenic." Suffice it to say the integrity of the central and peripheral nervous systems, the endocrine and vascular systems dictates function, and a failure in one or more of these systems can result in impotence.

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In reply. Dr O'Connor makes a point when he says that articles "often reflect the discipline of the author." A review of the psychiatric literature in the 20th century and conversations with psychiatrists over the years clearly show that the prevailing belief has been that impotence is mainly a psychiatric problem. This belief persists in the absence of any strong evidence to the contrary. My article was intended to show that, since the 1970s, evidence has accumulated, almost geometrically, that impotence is a vascular problem in many instances.

The current vascular treatment for impotence is intended to restore lost ability for vaginal penetration and is frequently successful. As a practitioner, I see the failures of psychiatric therapy but not the successes. "Improvement" is not enough. I can only reiterate the complaint that many of these patients are usually treated without physical or laboratory evaluation.

The concept that impotence is a vascular problem has been gaining acceptance. Patients can have both vasculogenic and psychogenic impotence and patient treatment on a cooperative basis is already a reality.

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Melville's "Bartleby the Scrivener"

TO THE EDITOR: It is always gratifying when medicine can and does make a contribution to the understanding and appreciation of a literary work of art. It was with pleasure, therefore, that I read the paper of Drs Whitehead, Liese, and O'Dell on Melville's Bartleby the Scrivener.[1] But I must take issue with their diagnosis and interpretation of the syndrome described. Their opinion is that "it can be concluded that Bartleby's Axis I diagnosis was major depression." However, the individual with major depression does not retain the emotional drive deliberately and calmly to take a course of action which can only infuriate his employer, the figure of authority. The depressed person who can no longer function will leave his work, not remaining in a situation which he is still competent to know will lead to conflict. He will not "prefer not"—but will either admit he cannot function or will leave the scene of his failure, not having the energy openly to resist. Bartleby's behavior can only be interpreted as hostility. With this in mind, it is apparent how successful is his campaign. He is taking vengeance on his employer as a representation of society. Subsequently, when he is in prison, he starves himself, implying that society is now persecuting him to the point of death, similar to the hunger strikes indulged in by some political prisoners, not because of depression but to publicize the world's cruelty. Ultimately he does starve to death, but at the end he has assumed a fetal position, a subconscious last protest against having been forced into life, which he hates.

The description of Bartleby's illness is a pure culture of a passive-aggressive personality disorder. The diagnostic manual quoted by the authors of the paper describes this personality disorder as a "pervasive pattern of passive resistance to demands for adequate social and occupational performance."[2] What better summary is there to describe Bartleby's character and behavior? That depression may accompany this disorder does not negate the diagnosis, but I doubt Bartleby's major emotional tone was depressive; rather it was hostility so intense that it dictated death rather than to permit society to succeed in integrating him into the accepted pattern of behavior in the current culture.

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1. Whitehead FA, Liese BS, O'Dell ML: Melville's "Bartleby the Scrivener": A case study. *NY State J Med* 1990; 90:17-22.
2. *Diagnostic and Statistical Manual of Mental Disorders*, ed 3. Washington, DC, American Psychiatric Association, 1987.

TO THE EDITOR: I read with interest "Melville's 'Bartleby the Scrivener': A case study" by Whitehead, Liese, and O'Dell in the January issue of the *Journal*.[1] I was especially concerned with the section pertaining to past psychological perspectives on the story.

Karl Kahlbaum (1828–1899) described catatonia in his monograph, *Die Katatonie odor das Spannungsirresein*, published in 1874.[2,3] The authors of the article on Melville believe the diagnosis of catatonia would not be tenable in the case of Bartleby. They say that while Bartleby had a number of symptoms of catatonia in common with Kahlbaum's original description, "he shows no evidence of delusions, hallucinations, or other severe thought impairments which are practically a *sine qua non* of schizophrenia today."[1] Apparently they lump together all the subtypes of schizophrenia when making this claim. Their assertion that there is an absence of thought impairment is open to question, and is not discounted by their brief reference to Bartleby's statements: "I know where I am" and "I know who you are." Furthermore, delusions and hallucinations are not necessary components of catatonia, especially the state of catatonic stupor.

A description and diagnosis of catatonia, although published by Kahlbaum more than a century ago, is still relevant today. It is important to note that in their discussion, the authors stress DSM III-R criteria.[4,5] Bartleby's illness fits the diagnostic description of catatonia in the earlier DSM-III. In view of ever changing classifications, who can predict what will be seen in the DSM VIII or IX, should they ever be published? Diagnoses in the future may revivify diagnoses of the past.

Bartleby's illness, as described by Melville, is an excellent representation of catatonic behavior leading to stupor and, in this case, to eventual death. This can and does happen. The diagnosis and occurrence of catatonia are apparently rare now in Europe and North America (DSM-III).[5] The authors of the Bartleby article mention a decrease in the frequency with which schizophrenia is diagnosed. Changes in the frequency of occurrence of some illnesses and symptoms are not unusual. There are also decreasing evidences of "La belle indifference" today, as compared with the era of Charcot. At the time the story was written, catatonia was common.

Two of the authors of the Bartleby article hold PhD degrees and one is a physician, affiliated not with a department of psychiatry, but with the Department of Family Practice at the University of Kansas Medical Center. I mention this because I have the impression that their observations are not based on their own clinical experience with psychiatric patients. I am reminded, consequently, of an observation made by William Osler in 1901. "To study the phenomena of disease without books is to sail an uncharted sea, while to study books without patients is not to go to sea at all."[6]

Whitehead, Liese, and O'Dell present Melville and his literary work well. I believe it is unfortunate and regrettable that their opinions and conjectures about the psychiatric theme in "Bartleby" mar their otherwise meritorious contribution.

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TO THE EDITOR: The fact that Melville's enigmatic character is plainly pathological has always driven readers to hunt for some scientific or at least logical key to his behavior.[1] What may be forgotten in that intriguing exercise is that Bartleby is a purely literary creation, not intended to be a case study or even an amateur psychiatric puzzle. It was in the nature of Melville's art, like that of his contemporary and friend Nathaniel Hawthorne, to point his reader past the descriptive markers in his story to some ulterior pattern of figurative meaning. If Bartleby is psychotic, it is a metaphoric psychosis intended to speak, not of mental illness, but of the spiritual tragedy of alienation and the eternal mystery of anomie. Seen in that light, the author's point is precisely *not* to be understood in any analytical sense: it is to confront the reader with unease rather than disease.

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In reply. We are delighted to respond to the letters concerning our paper.[1]

Dr Tarlau suggests that Bartleby has a passive-aggressive personality disorder. This idea is paralleled by literary critics who have suggested that he is variously: a pacifist[2]; a Buddhist contemplative[3]; a Christian martyr[4]; based on Henry David Thoreau, the well-known advocate of passive resistance in Melville's own era[5]; or even a Taoist holy man.[6] These critics have pointed out that several of Melville's other fictional characters such as Benito Cereno and Billy Budd similarly exhibit passive resistance traits, and that through them Melville may have favored some form of passive resistance to the injustices of established society.

Interestingly enough, it is Bartleby's fellow scriveners who at once become most incensed against him for what they consider slacking, which is indeed characteristic of passive-aggressive personalities. In the early part of the story, the attorney is equally indignant, referring to his "mulish vagary."[7] Yet as the story unfolds, the attorney is forced through Bartleby's patient, quiet refusals to confront and question all his own fundamental values, and thereby to become a more compassionate man.

Bartleby does not really display the aggressive side one might expect if he were passive-aggressive. He does not complain, "forget" where he put assigned work, or even resort to a sardonic smile. His overall emotional tone is of pervasive sadness, or as the narrator describes it, "melancholy."

Rather surprisingly, the passive-aggressive personality disorder has attracted little attention in the recent medical literature. Small et al[8] even subtitled their important paper "A Search for a Syndrome." Within the last few years, Perry[9] has tried to establish a clinical typology, and in a current review, Goulet[10] has urged a systematic study of the disorder and its diagnosis and treatment.

In our experience, patients with this disorder usually have a contentious side to their personalities, eventually manifesting itself underneath or alongside the passive traits. Furthermore, while it is not unknown for these patients to commit suicide, cachexia, as in Bartleby's case, is quite rare, and much more common in depression.

As we pointed out in our paper, the story well demonstrates the intricate and complex interaction between environment and character. From a medical standpoint, we are faced with a similar choice: Is Bartleby's illness exogenous or endogenous? Since Leo Marx,[11] the literary understanding of the story has tended to concentrate on the environment: the dark and bleak cistern-like office, the grim walls of Wall Street, etc. The other two scriveners have serious health problems. Turkey is an alcoholic, and Nippers has chronic indigestion. They are so impaired that each is functional for only half of any given working day. Would we say it just so happens that each of the three brings his sickness to the office, or that the office, with its unending, dull, and tedious copying, has caused their problems? In this regard the story remains problematic and challenging, but we think that those who incline to a purely endogenous view of Bartleby miss the many clues that Melville has given us, that the environment is of vital importance.

In response to our paper, Dr Schneck basically restates the argument of his 1972 article, that Bartleby suffers from catatonic schizophrenia.[12] On the surface, this is plausible, and has long been accepted by literary scholars. It is not utterly impossible that Bartleby is a schizophrenic as we pointed out in our paper, but we feel it is unlikely. Schneck cites Kahlbaum's classic study of catatonia,[13] but fails to note that most of Kahlbaum's cases evidenced hallucinations and delusions, such as that the Devil was attacking them, that their bodies were machines, and the like. We find evidence of these gross cognitive disorders absent in the description of Bartleby.

We have reviewed the details of Kahlbaum's 25 case histories, and find that 15 had hallucinations and similar severe thought disorders. Another group were victims of syphilis, epilepsy, likely meningitis, and an episode of attempted suicide by hanging which resulted in temporary paralysis. This diverse group, which included patients suffering from organic disease, were obviously neither schizophrenic nor affective. Two cases were megalomanics, and for one Kahlbaum gives only a brief autopsy report with no previous clinical information. The three remaining cases (Kahlbaum's case nos. 1, 18, and 19) were apathetic and melancholy patients quite like Bartleby. Using current diagnostic criteria, the five patients with hallucinations were likely or at least possible schizophrenics, while the three melancholics were probably victims of depression.

As early as 1913, Kirby[14] suggested that some psychotic and seriously ill patients were manic-depressive rather than schizophrenic. A number of recent studies, most published since Schneck's original article in 1972, have raised doubts about the diagnostic validity of catatonia, and have called for more detailed clinical analysis and evaluation of the term.[15–20] Some articles have concentrated on organic disease manifesting itself as catatonia (a possibility acknowledged by Kahlbaum himself, as we have seen).[21-24] Some articles particularly suggested that many patients diagnosed as schizophrenic were in fact suffering from affective illness,[25-26] which is exactly the point we are making.

Of course, it is possible that Bartleby had some form of organic disease, but if this is so, definitive evidence is lacking. The attorney who narrates the story comes to believe strongly that Bartleby's malaise is spiritual, and even philosophical or metaphysical in nature.

It is striking that Dr Schneck does not evaluate Bartleby's repetition of the phrase "I prefer not to." Kahlbaum frequently met with the phenomenon of his patients repeating meaningless words or phrases, which he defined as verbigeration, a term which has continued in clinical use.[27] We could view Bartleby's phrase as verbigeration, but to thus reduce it to something irrational and incoherent is surely to miss the point that Bartleby just does not want to do the kind of work which everyone else in the office does. And it is this work which interferes with the morale and health of his fellow scriveners as well.

As a final note, we were surprised that an eminent historian of medicine like Dr Schneck would resort to derogatory comments about the specialty of family practice. Here, as elsewhere, the use of an ad hominem fallacy may serve to conceal an insufficient evaluation of clinical evidence.

Dr Rosenberry argues that the story must remain, in the end, elusive and mysterious, and he takes a largely existential view. We agree with him to a certain extent, insofar as we acknowledge in our article that it is well for all of us to remember that Bartleby is a fictional character rather than a living patient. That said, we believe that it is precisely because the story of Bartleby is told with such a remarkable wealth of careful detail that readers have naturally felt challenged to explain the source and nature of his illness or malaise. We do not agree with Rosenberry that trying to understand this complex and fascinating story with the tools of modern clinical diagnosis is "murdering to dissect." Instead, we hold that understanding can deepen and enrich our response to literature. Thus, while we reaffirm our diagnosis that Bartleby is suffering from a major depression, the final word on this matter can never be spoken. Scholarly and scientific readers will no doubt produce new interpretations for a long time to come.

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The physician as lobbyist

TO THE EDITOR: The current regulatory and reimbursement climate is having an unprecedented effect on the practice of medicine in New York State. An ever-increasing volume of federal and state mandates now affects virtually all aspects of health care. Such restraints discourage promising young minds from entering the medical field, and make the options of early retirement or career changes more attractive to established practitioners. Many feel "that is just the way it is. There is nothing I can do." This is not true. Physicians can have a say in the changes which lie in the future. But to do so, they must learn how to become effective lobbyists. Only through advocacy, centralized lobbying efforts, involvement through their Political Action Committee, and grassroots lobbying will physicians succeed in affecting public policy and shaping the future health care system.

But what is lobbying? It is not a subject included in standard medical school curricula, nor is it a natural aptitude. Rather, it is a process one learns: first, by understanding the basics of the legislative system and second, by applying proven methods to influence the input and end products of that system.

Lobbying entails bringing to the attention of an elected official items of concern which currently or potentially impact on the lobbyist's clients. It is an educational process. The lobbyist provides information on issues to lawmakers that may not otherwise be available to them and, through logical argument, attempts to persuade the official to support a particular stand. Lobbyists are integral to the decision making process and oftentimes provide expertise. Lawmakers often rely on lobbyists for specific insights as they grapple with deciphering thousands of bills each year.

Recognizing the powerful role the New York State Legislature plays in governing the delivery of health services, the Medical Society of the State of New York (MSSNY) has placed a high priority on its lobbying function. The Governmental Affairs Division, housed a few yards from the State Capitol building, projects a strong voice and presence for organized medicine.

Though staff devotes the vast majority of its time to lobbying efforts, an essential element of lobbying and the key to ultimate success lies in broad physician member involvement. Today's health care delivery environment demands that physicians become intimately involved in lobbying activities, both in the home district and in the state capital. To integrate physicians into MSSNY's lobbying function, the

Division of Governmental Affairs organizes an annual legislative visitation program. Each week throughout the legislative session, a different county medical and specialty society is assigned a day for staff to travel to Albany with a contingent of their physician membership. Staff and physician members are briefed on the society's positions on key bills. They are escorted on visits with their local legislators. If the schedule permits, physicians and staff are encouraged to observe legislative committee meetings and/or session debates in order better to understand the state lawmaking process and to see their legislators in action.

The MSSNY has also organized a Key Contact Program in which physicians are called upon to contact personally their elected representatives on a specific item. This program provides physicians with an ideal opportunity for one-on-one lobbying. The Key Contact Program develops and enhances personal relationships between physicians and state policymakers.

The third large scale lobbying initiative of the Division of Governmental Affairs is the organization of two statewide legislative days, scheduled at the start of the legislative session in early spring and at the session's crucial climax in the summer. Representatives from all county medical and specialty societies congregate in the state capital for the purpose of launching a major educational campaign for the legislature. Through this intensive lobbying effort, the physician/lobbyist attempts to ensure that health care issues in the legislative arena get full and fair focus from informed legislators.

All three programs have been successful in facilitating physician/legislator exchanges and leading to a clearer understanding of each others' positions.

Regardless of which avenue physicians choose, the fundamental value and success of the encounter(s) is in the interface and dialogue. With this point in mind, the Division of Governmental Affairs offers some specific procedures that have a proven track record of success.

An excellent approach for physicians to take when lobbying is to present their arguments from the patient's standpoint: How will this proposed bill or regulation negatively or positively affect the patient? This is information that physicians have personal knowledge of and that legislators need to know to make good decisions. Once the case has been presented from the patient's standpoint, the stage is then set to present the argument from the viewpoint of the physician and organized medicine.

When physicians have the opportunity to meet with a legislator or staff member they must remember that in the great majority of cases, the senator or assemblyperson has been furnished with background materials. Position papers or memoranda in support or opposition are developed by the MSSNY for every major health related bill to be considered by the legislature, and are circulated among the members. Physicians should obtain copies of these materials and familiarize themselves with the MSSNY's position and rationale. They should also learn the arguments of the "other side" in order to prepare rebuttal. In this way they are in a position to answer questions or provide clarification.

Individual legislators will display varying levels of understanding on the same bill or issue. If it becomes apparent that the legislator has reviewed materials sent to him or her and clearly demonstrates a knowledge of the issue, the physician can then summarize the materials and reemphasize key arguments. On the other hand, if the legislator has not developed a good understanding of the issue, the physician/lobbyist should use the meeting as an educational session.

Thus, key points to remember are that physicians should explain the issue in lay terms, and relate the issue to the office setting and its effect on the patient. One should avoid the use of jargon or complex clinical language. When a physician speaks about actual office experience, this information is readily absorbed by the legislator both as a policymaker and as a health care consumer.

There is another unique avenue of lobbying which physicians may utilize. That is using the one-on-one doctor/patient office visit to educate patients about particular pieces of legislation which affect them. This opportunity can be used in shaping public opinion and perhaps ultimately to persuade patients in turn to lobby their elected representatives.

Each physician must make a judgment as to the appropriateness of this lobbying vehicle based on his or her relationship with patients. But those who have used their offices for lobbying purposes have usually come to view their patients as important and powerful allies. Many encouraging success stories have been reported to the Division of Governmental Affairs.

The current health care environment demands that physicians become more actively involved in formulating public policy. The need for physician input and involvement will only grow. The medical community has great potential influence to wield in shaping the practice of medicine in New York State. But this reservoir of power will remain only theoretical unless physicians unleash it. It is the goal of the physician/lobbyist to work toward this aim.

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Bicycle injuries

Traveling by bicycle is common throughout the world. There are an estimated 85 million bicycles in the United States today,[1] and the New York State Department of Economic Development reported that more than 750,000 bicycles were sold in the state during 1989.

Whether used for transportation or recreation, bicycling is a healthy activity. However, if cyclists ride unprotected and unaware of safety rules, they can be seriously injured.

From 1984–1988, New York State death certificate data show approximately 300 people died from injuries suffered in bike mishaps, and more than 69% of these people suffered head injuries. Bike injuries are the leading cause of head injuries in school-age children.[2]

As Figure 1 indicates, more than 77% of bicycle-related deaths in New York State involved collision with a motor vehicle, 21% were nonmotor vehicle related (eg, collision with a fixed object or fall from bike) and 1.5% involved cyclists killed after hitting a pedestrian. One third of the cyclists killed were between ages 5 and 14 and an additional 30% were between ages 15 and 24.

Nonfatal bike-related injury data are limited to police reports of bicycle/motor vehicle collisions. Between 1984 and 1988, an average of 10,000 individuals per year were reported injured in bicycle/motor vehicle collisions in New York State. Of those injured, 38% were between ages 15 and 24, and 33% were between ages 5 and 14.

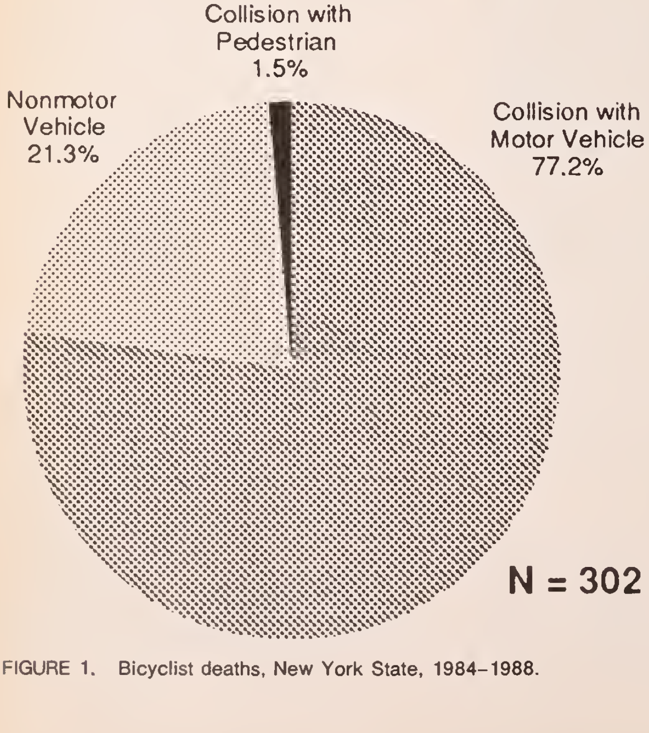

FIGURE 1. Bicyclist deaths, New York State, 1984–1988.

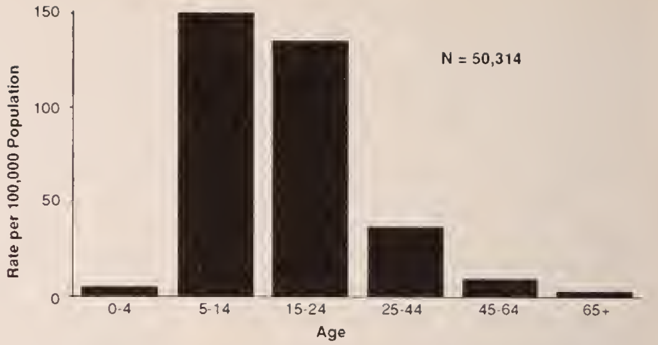

FIGURE 2. Rate of injury from bicycle-motor vehicle collisions (including all fatal and nonfatal injuries), New York State, 1984–1988.

Figure 2 shows the relative rates of age-specific injuries from bicycle/motor vehicle collisions during 1984–1988. The overall rate was 52.9 injuries per 100,000 population, representing more than 50,000 fatal and nonfatal injuries. The highest rates were for the ages 5 through 14 (149.9/100,000) and for ages 15 through 24 (134.9/100,000).

Police accident reports represent only a portion of nonfatal bike-related injuries. Only one-fifth of bicycle-related injuries treated in North Carolina emergency rooms resulted from motor vehicle collisions, and only 10% of those injuries were reported to the police.[2] Thus, the actual number of bicycle-related injuries might be ten times greater than the numbers represented by available data.

Public Health Role

Through public health programs, bicycle-related injuries can be prevented or reduced by:

- educating bike riders;
- providing a safe riding environment;
- promoting public information campaigns, bike rodeos and school-based programs.

Bicyclists share the road with motor vehicles and pedestrians, and must follow many of the same laws intended to ensure safe traffic flow. However, unlike motorists, license and formal training or testing are not required to operate a bicycle on public roadways.

Few roadways provide separate or adequate space for bicycle traffic. Public health agencies can promote bicycle lanes in reconstructed and newly constructed roadways and bicycle paths in public parks and recreational areas.

Bicycle safety helmets prevent or reduce head injuries. A one year case-control study conducted at five major hospitals in the Seattle area[3] concluded that wearing bike helmets reduced head injury risk by 85%. During the year, 776 cyclists were treated in studied hospitals, of which 42.1% suffered brain injury (concussion or worse); 43.4%

suffered scalp injury; 47.2% suffered forehead injury; 8.1% suffered ear injury; and 1.3% died. Approximately 65% of the brain injured were children.

To reduce bicycle-related head injuries in children, the New York State Legislature enacted a bicycle helmet law in 1989. The law requires a bicycle passenger under age five to wear a helmet and ride in a separate seat attached to the bicycle. In addition, a child under age one is prohibited from being a passenger.

Department of Health Program

The State Department of Health Injury Control Program directs a statewide bicycle helmet initiative to prevent disabilities from bicycle-related head injuries. The bicycle helmet project, centered around the theme, "Head Out with a Helmet," focuses on three strategies and includes an evaluation component.

Helmet Distribution

The department funds programs that distribute bike helmets to children from low-income families in counties with the highest rates of bicycle injuries from bicycle/motor vehicle collisions reported to the New York State Department of Motor Vehicles. Currently, the distribution has begun in New York City, and Niagara, Monroe, Erie, Onondaga, Chemung, Schenectady, Albany, Cortland, Genesee and Warren counties. Other counties that have received technical assistance in developing bicycle helmet education programs are Westchester, Clinton, Essex and Saratoga.

Evaluation will focus on public awareness, including the number of educational sessions conducted and the number of children receiving and wearing helmets. The Injury Control Program has observational study protocols available to measure project effectiveness regarding increased helmet use.

Professional Education

Weiss reported that physicians have a unique opportunity to educate families and the community about the importance of helmet use.[4] The Injury Control Program sent an information packet to all New York State family practice physicians and pediatricians affiliated with the New York State American Academy of Pediatrics encouraging involvement with community bicycle helmet initiatives. The packet contained additional information for use in their medical practices. Requests for materials are monitored to assess the packet's success.

Public Education Campaign

The Injury Control Program oversees Safe Kids activities in New York State as part of the National Coalition to Prevent Childhood Injury (Safe Kids).

To increase bicycle helmet use, the Safe Kids Campaign distributed a public service announcement to 33 television stations statewide and additional materials during 1989. Currently, a statewide campaign is being developed to regularly include the helmet as bicycle equipment.

A 1984 Burlington, Vermont, study of bicyclists over age 10 (nearly 42% were ages 10 to 19) reported the most common reasons for not wearing a bicycle helmet were: rider on a short trip, 28%; uncomfortable, 27% and negligence, 13%.[5]

School-based Survey

To determine the level of helmet ownership and usage and to identify target groups, a survey of schoolchildren was developed. A questionnaire, designed for easy use by a classroom teacher, asked schoolchildren about bicycle ownership, frequency of bicycle use, helmet ownership, availability and frequency of use, the number of students riding a bicycle to school that day with and without helmets and the number who would wear a helmet if available. The classroom teacher read each question to the class and recorded the number of male and female students responding to each. With cooperation from the New York State Congress of Parents and Teachers (PTA) and local PTA presidents, a random sample was drawn, stratified by geographically defined PTA regions. Nearly 28,000 students in grades K-12 were surveyed.

Results showed that 82.6% ride bikes, only 14.5% of those own helmets and only 20.3% of those ever wear their helmets. Only 3.6% of those students owning a helmet reported always using it. Males were twice as likely as females to own a helmet and nearly twice as likely to always or sometimes use a helmet. More than three-quarters of the kindergarten students indicated they would wear a helmet if given one. This rate decreased by grade to less than 5% for high school students. These results are consistent with other studies documenting low use of bicycle helmets.[1,3,5–7] New York City schools are currently being surveyed.

The survey form and instructions are available for any agency or group interested in encouraging helmet use in its community.

The New York State Department of Health has an educational brochure for adults, entitled *Head Out With a Helmet*, available in limited quantities, fact sheets and physician promotion materials.

For copies or more information, contact the Injury Control Program, New York State Department of Health, Empire State Plaza, Corning Tower Room 621, Albany, NY 12237.

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MEDICARE'S NEW HOSPITAL PAYMENT SYSTEM: IS IT WORKING?

By Louise B. Russell. 114 pp, illustrated. Washington, DC, The Brookings Institution, 1989. $22.95 (clothcover)

This reviewer's initial reaction to this book may send traditionalists into emotional shock. I suggest that you read the last page of the last chapter first. In so doing, basic concepts about Medicare's new hospital payment system can be crystallized and kept foremost in your mind as you read the rest of this volume. Detective mystery enthusiasts are urged to forgive this serious breach of etiquette.

On page 91, the author comments that the underlying issue "about prospective payment is not solely, or even primarily, technical. It comes down to: how much does the nation want to spend on the medical care of the elderly? And for what standard of care?" Thus, the response depends upon American attitudes toward and values about the elderly. Significantly, few medical schools offer many geriatric care courses in the curriculum.

Congressional intent aimed "to pay for necessary care efficiently provided" with quality maintained. However, the amount of dollars expended and the individual's resultant health status are not clearly correlated or easily defined. Other nations do spend far less and achieve far better health status results for their citizens than the United States.

In addition to starting from the back of the book, I would also suggest that readers remember that the prospective payment reimbursement concept is well on its way into ambulatory care via ambulatory visit groups (AVGs); into long term care via resource utilization groups (RUGs); into payments for physicians via resource based relative value scales (RBRVS); into psychiatric care via alternate DRGs (ADRG); and into payments for nursing care via relative intensity measures (RIMs). It is apparent that there are enough reimbursement schemes already being tested to eventually encompass the entire health care delivery system.

On the dust jacket, the title asks if the prospective payment system is working, with 25 question marks in a row. That question is answered by Bruce K. MacLaury, President of The Brookings Institution, in the foreword, as he notes author Louise B. Russell's findings: " ... prospective payment has accomplished its primary goal by achieving substantial savings without shifting costs to other payers." Commenting on the immediate associated issue, he points out that " . . . the available evidence does not show any clear deterioration in quality."

With this question answered, what follows, according to those who debated the laws, mores, and customs, is "merely commentary." This concise, well-documented book of about 125 pages provides that commentary within six chapters, 18 tables, three figures, an index, and 205 references. Technical rationales are easy to understand and follow.

Medicare hospital expenditures rose alarmingly from $5 billion in 1970 to $37 billion in 1982. Legislative actions evolved from the threatened bankruptcy of the Medicare Hospital Insurance Trust Fund. That threat resulted in the passage of the Tax Equity and Fiscal Responsibility Act of 1982 (TEFRA). TEFRA established the prospective payment system (PPS) that used diagnosis related groups (DRGs) as the reimbursement mechanism for inpatient hospital care provided to people covered by Medicare. About 28 million elderly plus three million disabled individuals under 65 are in the PPS/DRG program.

Importantly, the author asks: What's known about PPS? What needs to be learned? What is the tradeoff between costs and care? How much quality would the nation be willing to give up for greater savings?

For those unfamiliar with the PPS/DRG system, chapter two explains the specific details. Five variables interact in the reimbursement calculations: urban or rural area, hospital wage rates, teaching hospital designation, and the proportion of low-income patients in the case-mix. In essence, if care is provided by the hospital at less cost than the fixed payment for each of the current 475 DRGs, the hospital profits. If the care costs more, the hospital absorbs the loss.

Changes occurred in the pattern of care as hospitals reacted to the cost containment incentives. For the first time since 1967, Medicare hospital admissions dropped sharply to the lowest occupancy rate in four decades, about 65%. There was a significant shift to outpatient alternatives, such as ambulatory surgery and home health care. "... [W]ithout question, elderly patients are being cared for differently now, with shorter hospital stays and more post-hospital care."

Concerns about the quality of care caused legislatively mandated Peer Review Organizations (PROs) and others to look at mortality rates, readmissions, transfers, and premature discharges. There is "no clear cut signal that prospective payment has brought ill effects. So far . . . the loss of good health does not appear to be large although the evidence is sketchy and there are worrisome signs."

Revenue from Medicare accounts for about 30% of a hospital's income. It is estimated that the PPS/DRG scheme will save 20%, reducing projected 1990 expenditures by $18 billion. How will hospitals fare? "Early concerns that hospitals might not be able to cope with the new system have turned out to be unwarranted; most have done well and gradual adjustments in the rate system are helping many of the rest." Third party payers seem to be benefiting from changed "practice patterns" of providers adopting cost containment procedures. Out-of-pocket expenses of patients are not increasing.

Since the PPS/DRG reimbursement concept is complex, this book provides an excellent quick and ready reference guide for facts and figures. If you disagree with the payment scheme, this book is still highly recommended. At one sitting, readers can gain insights from the accumulated evidence, the issues still to be resolved, and the portents for the future. One must remember that the basic prospective payment framework has remained unchanged for the past six years and is accepted by even the critical professional provider associations. It is vital that practitioners become familiar with the ins and outs of this reimbursement scheme. Frequently suggested changes, such as severity of illness adjustments, may be achieved more quickly from the inside than from outside of the mainstream in the health care delivery system. PPS/DRG appears to be alive and well in the United States.

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MORPHINE, ICE CREAM, TEARS: TALES OF A CITY HOSPITAL

By Joseph Sacco, MD. 264 pp. New York, William Morrow & Co, Inc, 1989. $17.95 (clothcover)

For most physicians, internship evokes many memories. It is a one-year period that entails long hours, hard work, and an emotional roller coaster ride through the various medical specialties. In my city hospital training days, the term "days of the giants" was the byword for our housestaff. For Joseph Sacco, MD, "Morphine, ice cream, and tears" became his battlecry.

In this book Dr Sacco describes his internship at the private "Manhattan Hospital" and its sister city hospital, "New York General." In its 18 chapters the reader is educated about the basic process of becoming a physician and the various patients and rotations the author has experienced. Numerous case histories and personal experiences are shared with readers by the author in his attempt to demonstrate the horror, humor, greed, and pathos that surround medical training in a big city hospital environment. From such chapters as "The Patient Even Bellevue Didn't Want" to "Successes" and "Death," the full life cycle of the intern's relationships are explored. There is a serious attempt to explain and expound upon common medical jargon and slang, particularly valuable to lay readers. Very good explanations of dementia and congestive heart failure, to cite just two, are included.

The reader is provided with Dr Sacco's feelings regarding the threat of high technology medicine. Too often, he feels, money is the dominant force in medicine, and more often than not, doing too much is often worse than leaving the patient alone (ie, the risks seem to outweigh the benefits). As a primary care physician, I can certainly relate to these sentiments.

Two excellent chapters are "Smoking and Booze" and "Just Say No?" which describe the important medical topics of smoking, alcohol abuse, and drug abuse. The author expresses his opinion that most medical problems remain primarily social, not medical, in origin. He cites cigarettes, alcohol, drugs, obesity, violence, and various forms of pollution as America's number one killers. His inner city internship certainly provided him enough clinical experiences to support this. From a preventive medicine standpoint, I could not agree more.

Morphine, Ice Cream, Tears is written in a clear, direct style. Dr Sacco's views and opinions are straightforward and easy to follow. His primary motive for choosing medicine as a career has remained intact despite his cynicism. He still has an earnest desire to help his fellow man and to serve his patients.

This type of book has been done before. Two examples that immediately come to mind are *Intern* by Dr X and *The Making of a Surgeon* by William Nolen, MD. The city hospital approach provides a different emphasis, but is certainly not unique. I doubt this book will make the average doctor's bookshelf, but do think it has its niche in the hospital library.

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THE "FEELING GREAT!" WELLNESS PROGRAM FOR OLDER ADULTS

Jules C. Weis, MA, ATR. 218 pp, illustrated. Binghamton, NY, The Haworth Press, Inc, 1988. $24.95 (hardcover)

The purpose of this book is to instruct the reader on how to create and operate a community based comprehensive wellness program. Such a program consists of safe and proper exercises, relaxation techniques, health care lectures and discussions, and organized group socialization/recreational activities, all geared for the older adult. The book describes all aspects of the program, starting with a listing of the theoretical and observed benefits to the participants and sponsoring agency, and then progressing in great detail to the administration of the program. Areas of discussion include marketing of the program, required staffing patterns, fees to be charged, types and intensity of exercises and activities to be done, scheduling of activities, qualifications of the instructor, medical clearance required for the participants prior to enrollment, and necessary equipment, as well as printing of the actual forms used for the record keeping of the program. The book also presents several case histories of some of the participants and describes how each one benefited from enrollment, and prints the texts of actual lectures given in the program.

The author of the book is an art therapist who has been trained as an exercise instructor by the YMCA, and who was the Director of the "Feeling Great" program at the Lee Circle YMCA in New Orleans. His knowledge of proper and safe exercise techniques for the geriatric patient is good, and he describes the proper precautions that must be taken for the older adult who will increase his or her level of activity through enrollment in the program.

The book is geared towards those agencies and health professionals who are interested in starting and operating a wellness program for older adults. Although the book offers a good core of information, it is at times wordy and repetitive in presenting information. The content is accurate and the wellness program described is a safe and beneficial one for participants.

The physician reader on the whole would not find the book good reading as it is quite detailed regarding administrative and organizational matters. The physiologic and psychosocial sections would be of interest to physicians, especially those dealing with geriatric patients. However, these features are intermingled with other information that would not be of interest.

In summary, the book is a good reference for those who are interested in creating and operating a comprehensive wellness program for the older adult. In general, the nonphysician health professional would find the book most beneficial.

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TREATMENT PLANNING AND DOSE CALCULATION IN RADIATION ONCOLOGY

Fourth Edition. By Gunilla C. Bentel, Charles E. Nelson, PhD, and K. Thomas Noell, MD. 345 pp, illustrated. Elmsford, NY, Pergamon Press, 1989. $22.50 (softcover)

This is an excellent text on the practical aspects of radiation therapy planning. The authors are Ms Bentel, a dosimetrist; Dr Nelson, a radiation therapy physicist; and Dr Noell, a radiation oncologist. The book aims to give the reader an understanding of the several stages involved in the planning and execution of radiation treatment.

The book has eight chapters which are well arranged. There are chapters on basic radiation oncology, radiation physics, dose calculations for external beams, pretreatment procedures, brachytherapy, principles of external beam treatment planning, practical treatment planning, and normal tissue consequences.

The major part of the book (150 out of 330 pages) is devoted to the topic of "Practical Treatment Planning." This is the main strength of the book which gives emphasis to each of the commonly encountered tumor sites. Detailed descriptions of radiation therapy techniques to treat each of these sites are provided. Photographs and diagrams of treatment fields, field arrangements, simulation films, and port films are nicely presented. This section is comprehensive and extremely useful to the readers. The discussion on how common mistakes could be avoided is particularly valuable.

Another section of the book that is especially informative is the chapter on pretreatment procedures. Details are given of the construction of bolus, compensators, and immobilization devices as well as methods of radiographic localization. Here, too, the avoidance of common pitfalls and misconceptions is highlighted.

This book has long been a major reference for technologists, dosimetrists, and physicists for treatment planning, and this updated and expanded version will be welcomed by many in clinical physics and radiation oncology. The writing is clear, concise, and easy to understand and ample illustrations are included. Although the clinical section is excellent, the basic physics of radiation therapy section is not very detailed. However, this book is not meant to be a textbook of basic clinical physics. Several other texts fulfill this need.

In conclusion, we find this to be an excellent treatment planning textbook which is highly recommended for both students entering this field, physicians in training, and experienced practitioners in the field.

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EVOKED POTENTIALS IN CLINICAL MEDICINE

Second Edition. Edited by Keith H. Chiappa, MD. 667 pp, illustrated. New York, Raven Press, 1990. $58.00 (hardcover)

Evoked potentials represent the electrical activity associated with the nervous system's response/processing of external stimuli and are accepted test procedures in clinical medicine. Their utility has changed over the last decade because of restrictions by other modalities and expansion into different areas such as intensive care and intraoperative monitoring. During this time period, Dr Keith Chiappa's 1983 book, *Evoked Potentials in Clinical Medicine*, has been an extremely useful guide for methodology and use. Because of changes in the field since its initial publication, a second and expanded edition is now available.

Dr Chiappa is an acknowledged expert in this field and the new edition reflects this expertise. As in the first edition, the book is divided into chapters discussing methodology and interpretation of visual, auditory, and somatosensory evoked potentials. The methodology chapters provide a primer for the technical aspects of each test, normative data, and a clear description of factors that can affect test results. The sections on interpretation are excellent, providing information on anatomic underpinnings of evoked potentials and test findings in a myriad of disorders. Each chapter is well organized with lucid subsection headings and concise discussions. References are abundant and up to date.

The second edition also has new chapters. A section on pediatric somatosensory evoked potentials provides an up-to-date description in this field. A chapter on motor evoked potentials, which study corticospinal pathways, discusses previous studies and explores their potential utility. An extremely useful chapter on statistics provides guidance in establishment and limitations of normative lab values. The sections on endogenous event-related potentials and advanced techniques offer a brief review of these fields, although the former is limited in content and could use a better description of the psychological correlates of waveform components.

In the preface to the first edition, Dr Chiappa limited the discussion to "techniques that provide the maximum amount of information relative to the patient's clinical problem in a minimal amount of time." This second edition follows the same theme. Comments on topographic mapping are minimized because of its limited utility and intraoperative monitoring is not discussed because of space limitations. The discussion on electroretinography could be expanded into a separate chapter.

The second edition has been eagerly awaited and, as expected, provides no disappointment to those looking for a reference text in evoked potentials. This book is required reading for anyone who is training in evoked potentials or wishes to establish a clinical neurophysiology laboratory. For those with experience in the field, it is an extremely useful text that will provide technical and clinical information.

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ASK SIR JAMES: SIR JAMES REID, PERSONAL PHYSICIAN TO QUEEN VICTORIA AND PHYSICIAN-IN-ORDINARY TO THREE MONARCHS

By Michaela Reid. 315 pp, illustrated. New York, Penguin Books, 1989. $21.95 (hardcover), $12.95 (softcover)

Royal physicians occupy a unique niche in history because their profession permits a close and confidential relationship with those at the center of political power.

In 1881, at the age of 32, James Reid, a well trained and talented physician from Aberdeenshire, became personal physician to Queen Victoria who was then 30 years his senior. The choice of Reid was not only based on his professional qualifications, which were superior, but also on the Queen's idiosyncratic requirements which included that the candidate be a Scotsman, preferably from Aberdeenshire, and that he speak German. Reid filled the bill on all three counts, having been born in Aberdeenshire where the Queen's beloved Balmoral Castle was located, and having learned German during his year of medical study in Vienna. The German language requirement was an important one since the Queen had a large number of German-speaking relatives who regularly visited her.

Reid, who was from a modest middle class background, found himself suddenly propelled into the center of one of the most important royal courts of the era. Professional skills alone could not have carried the day for him. While these were critical to his success, so were his discretion, sense of humor, good judgment, and charm.

Unlike many of those in the Queen's inner circle, Reid never told her what she wanted to hear but rather what he believed she should know. That she tolerated this from him and not from others had its roots in her dependence on him as her male support. Although the Queen possessed a strong character and could at times be opinionated, she was highly dependent on a succession of male figures for her happiness. The first was her husband, Prince Albert, who died 20 years before Reid arrived at court; the second John Brown, a Scottish gillie; and the third Reid.

Michaela Reid, who is the wife of Sir James Reid's grandson, has produced not only a fascinating biography, but also an extremely unique and important historical document based on primary source materials, namely hitherto unquarried letters, documents, and diaries. She has synthesized all of this material against the backdrop of the era and the two decades during which Reid served the Queen. What emerges is a complicated but easy to comprehend portrait of the role this particular physician played in the personal life of his sovereign and in the political life of his country. He was responsible not only for the health of the Queen, but also for that of her family, visitors, and the members of the household. His medical skills had to be mustered to deal with everything from royal births to accidents. His wisdom and counsel were relied upon by the Queen and those around her to settle household disputes, family crises, and thorny political issues. In discharging those nonmedical duties, Reid never failed the Queen and consistently won the respect of her other counselors and the government. This was no small accomplishment given the Queen's tendency to be obstinate and opinionated at times and her penchant for meddling in the lives of all those around her.

Reid's remuneration was always modest but as he himself remarked, his expenses were few. The Queen eventually knighted him, 14 years after he began serving her, and gave him a baronetcy in 1897 on the day of her Diamond Jubilee celebration. That he was still a bachelor at 50 suited her and not surprisingly she reacted adversely to his plans to marry Susan Baring, one of her maids of honor. An interesting aside is that Susan Baring's sister was married to the sixth Earl Spencer, the great-grandfather of the present Princess of Wales. Reid was married in 1899 and for the next year juggled his commitments to his wife and to the Queen.

Queen Victoria's health was fairly robust during the time Reid served as her physician. However, she tended to overeat, was subject to fits of depression, and in her late years suffered from insomnia. That she was inclined to be hypochondriacal at times and had strong views on the management of certain illnesses did not make Reid's task easy. In 1900 the Queen's health began to decline as a result of what appear to have been a series of transient ischemic attacks which left her temporarily aphasic and drowsy. She was now 81 years old and in this weakened condition journeyed to Osborne House on the Isle of Wight where she died on January 22, 1901. Reid was constantly with her during the last few days of her life and as she had requested saw to certain details about her burial.

Reid had been out of the mainstream of the medical profession for two decades. At 52 he started a private practice in London after taking refresher courses in some of the leading hospitals. It was not an easy time for him and his growing family. Yet he rose to the occasion with the same success that characterized his previous career. He was frequently called upon by King Edward VII and King George V and was in regular contact with members of the Royal Family. Reid died on June 28, 1923, at his home in Aberdeenshire. His letters and diaries came close at times to being destroyed or discarded. Fortunately, they survived and have been given a new life in the form of this beautifully crafted and meticulously researched biography which is a pleasure to read.

PASCAL JAMES IMPERATO, MD
Editor

Diagnosing Alzheimer's disease with a biochemical assay

Alzheimer's disease (AD) is characterized by progressive loss of memory and deteriorating intellectual capacity. However, a diagnosis of AD requires a history of clinical dementia as well as the presence of neuritic plaques (NPs) and neurofibrillary tangles (NTs) in brain tissue. This presents several problems in diagnosing AD: for example, staining and analyzing the brain tissue is labor intensive; NPs and NTs also appear in elderly patients without AD; and NPs and NTs may not be distributed evenly throughout the brain, requiring sampling of brain tissue from multiple sites.

Ghanbari et al reported on a monoclonal antibody-based assay that detects a protein marker highly specific for AD that appears early in the course of the disease (*JAMA* 1990; 263:2907–2910). The sandwich enzyme immunoassay technique utilized by Ghanbari and colleagues overcomes the problems of cross reactivity previously seen with immunoassays and Western blots.

The authors used freshly frozen brain samples in this study. The tissue came from patients who had all had clinical dementia and evidence of AD on histopathologic evaluation of postmortem brain tissue. For comparison, the authors also examined brain tissue from normal controls and from cases of other neurologic diseases.

This biochemical immunoassay yielded no false positives in either control group. Among the 56 AD cases, the assay produced eight false negatives, for a sensitivity of 85.7%. The concentrations of AD-associated proteins (ADAP) in the AD group was significantly greater than in the non-AD group. The authors concluded that "clinical dementia, NPs, and old age, per se do not appear to be associated with the increased ADAP concentrations in the AD group," and therefore this biochemical assay for ADAP could aid in diagnosing Alzheimer's disease.

Risk of acute ibuprofen overdose remains low

"The risk of complications from acute ibuprofen overdose remains low despite increased availability of nonprescription dosage forms in the general population," report McElwee et al (*Ann Emerg Med* 1990; 19:657–662). Up until 1985, ibuprofen was available only by prescription. Subsequently, with easier availability, its use dramatically increased, and there were more frequent reports of acute ibuprofen overdose. However, the authors found that the safety record of ibuprofen has not worsened since its availability without a prescription, and the risk of life threatening symptoms associated with acute ibuprofen overdose is low.

Defining overdose as a dose greater than that recommended on the label, the authors evaluated all reported cases of ibuprofen overdose in the state of Utah during an 18-month period. Most (59%) overdoses were accidental ingestions in children and infants. Seventy-one percent of the patients were treated at home, with many asymptomatic cases receiving no treatment, and emesis induced with ipecac syrup or dilution used to treat some patients. Activated charcoal, cathartics, and gastric lavage were required for gastrointestinal decontamination in some patients treated at a health care facility.

Acute ibuprofen overdose produced no effects in 49.8% of the patients in this study. The authors recommended against routine ibuprofen serum concentration determination in patients who ingest excessive doses, because there is no specific antidote and treatment is primarily determined based on symptoms, and because the authors found a poor correlation between ibuprofen serum concentrations and clinical findings.

Lyme disease has become a "diagnosis of exclusion"

The Lyme Disease Center at the Robert Wood Johnson Medical Center is located in north-central New Jersey, a region endemic for Lyme disease. Leonard H. Sigal, MD, summarized the presenting symptoms, diagnoses, and treatments of the first 100 patients seen at this center between September 1988 and July 1989 (*Am J Med* 1990; 88:577–581). His findings suggest that Lyme disease has become a "diagnosis of exclusion" in endemic areas and that excessive antibiotic therapy is frequently the result of the incorrect assumption of persistent infection.

At the Lyme Disease Center, a diagnosis of Lyme disease was based on the presence or history of a rash characteristic of erythema chronicum migrans, seropositivity for *Borrelia burgdorferi* infection, and a clinical course associated with Lyme disease. The referral diagnosis was Lyme arthritis in more than 50% of patients, neurologic disorders, including memory loss, headache, and peripheral neuropathies, in nearly 25% of patients, and chronic fatigue in nearly 20% of patients.

Of the 100 patients referred to the center, 37 were believed to have had Lyme disease as the cause of their complaints. More and more frequently, a diagnosis of Lyme disease is made on the basis of a broad array of symptoms, because no other cause can be pinpointed. In endemic areas, persons may be seropositive for *B burgdorferi* but have no history of Lyme disease or exhibit clinical symptoms that are not due to Lyme disease.

Many courses of antibiotic therapy for refractory Lyme disease are probably unnecessary, concluded the author. "Over-diagnosis of Lyme disease," the author observed, ". . . probably occurs because of the absence of strict clinical criteria for the diagnosis of Lyme disease as well as because of the difficulties in interpreting currently available, non-standardized diagnostic tests and increasing concern among physicians and patients that late stages of Lyme disease may be overlooked."

Blood donors will now be tested for hepatitis C

The American Red Cross, the American Association of Blood Banks, and the Council of Community Blood Centers have announced plans to begin using the hepatitis C test, approved by the Food and Drug Administration (FDA) on May 2, to screen all blood donors and all previously stored blood (*Dentistry Today* 1990; 9(5):10). This new test is expected to prevent as many as 100,000 cases of

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NEWS BRIEFS are compiled and written by Vicki Glaser.

hepatitis C each year.

The hepatitis C virus, previously known as non-A, non-B hepatitis, causes the most common form of this liver disease. It took only two years since the 1988 discovery of the hepatitis C virus for an FDA-approved blood test to come to market. The test identifies antibodies to the hepatitis C virus in the blood, which is indicative of previous exposure. Although only about 10% of hepatitis C cases are related to blood transfusions, and only 0.5–1% of blood donors are expected to test positively, there had been a 1 in 25 chance of a blood recipient becoming infected with hepatitis C prior to the availability of this test.

Due to the window period between infection with a virus and development of antibodies—about three to six months for hepatitis C—the test detects only about half of the people in the early stages of hepatitis.

Cholesterol levels and ten-year mortality among men with and without preexisting cardiovascular disease

"Our results suggest that the potential impact of changes in lipid levels on the risk of death from cardiovascular disease may be considerably greater for men with preexisting cardiovascular disease than for healthy men." This conclusion is based on a study by Pekkanen et al (*N Engl J Med* 1990; 322:1700–1707) of cholesterol levels and the ten-year mortality from cardiovascular disease for 2,541 men aged 40–69 years at baseline who either did or did not have preexisting heart disease.

At the time of baseline measurements, the subjects with cardiovascular disease were older, had higher levels of total cholesterol, low-density lipoprotein (LDL), and triglycerides, and were more likely to have hypertension. For all of the men in the study, higher total and LDL cholesterol levels and lower high-density lipoprotein (HDL) levels were associated with an increased risk of death from coronary heart disease and cardiovascular disease.

"A high total cholesterol level was associated with an increase of 15.8% in the 10-year risk of death from cardiovascular disease among the men who had signs of cardiovascular disease at base line and an increase of 3.2% among the men without such signs." Similar findings were reported for high LDL and low HDL levels. Furthermore, men without cardiovascular disease (CD) at base line but who had high total blood cholesterol levels faced a greater risk of death from CD or coronary heart disease than did men with CD who had moderate blood cholesterol levels.

Edgar Hodgins Bates, MD, Douglaston. Died February 14, 1990; age 85. Dr Bates was a 1931 graduate of the New York University School of Medicine, New York. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the American Occupational Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Alfred Berl, MD, Bullville. Died May 11, 1990; age 65. Dr Berl was a 1949 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Charles Maxwell Biren, MD, Staten Island. Died December 1989; age 76. Dr Biren was a 1948 graduate of the Chicago Medical School, Chicago, Illinois. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Hyman Blume, MD, Scarsdale. Died December 12, 1989; age 84. Dr Blume was a 1931 graduate of the New York University School of Medicine, New York. His memberships included the New York Academy of Medicine, the New York State Surgical Society, the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Ben Albert Borkow, MD, New York. Died December 9, 1989; age 91. Dr Borkow was a 1924 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American College of Angiology. His memberships included the New York State Society of Internal Medicine, the American Geriatrics Society, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Adrian Brodey, MD, Lawrence. Died May 11, 1990; age 79. Dr Brodey was a 1937 graduate of Rush Medical College, Chicago, Illinois. He was a Diplomate of the American Board of Dermatology, Inc. His memberships included the New York Academy of Medicine, the New York State Dermatological Society, the American Academy of Dermatology, the Society for Investigative Dermatology, the Nassau County Medical Society, and the Medical Society of the State of New York.

DeWitt Clinton Brown, MD, Stony Brook. Died May 2, 1990; age 85. Dr Brown was a 1933 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the Suffolk County Medical Society, and the Medical Society of the State of New York.

William A. Busino, MD, Schenectady. Died February 3, 1990; age 72. Dr Busino was a 1950 graduate of the Albany Medical College of Union University, Albany. He was a member of the Medical Society of the County of Schenectady and the Medical Society of the State of New York.

Rita Mendelson Chalef, MD, Boca Raton, Florida. Died May 4, 1990; age 65. Dr Chalef was a 1950 graduate of the Medical College of Pennsylvania, Philadelphia, Pennsylvania. Her memberships included the New York Academy of Medicine, the American Psychiatric Association, the Nassau County Medical Society, and the Medical Society of the State of New York.

William D. Dugan, MD, Hamburg. Died April 15, 1990; age 77. Dr Dugan was a 1939 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a Diplomate of the American Board of Orthopedic Surgery. His memberships included the American Academy of Orthopaedic Surgeons, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

William Lawrence Einhorn, MD, Bolingbrook, Illinois. Died May 10, 1989; age 77. Dr Einhorn was a 1938 graduate of the New York Medical College, New York. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Benjamin Feldman, MD, Miami, Florida. Died March 7, 1990; age 98. Dr Feldman was a 1919 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Walter A. Fenstermacher, MD, Rochester. Died May 1990; age 87. Dr Fenstermacher was a 1927 graduate of the University of Michigan Medical School, Ann Arbor, Michigan. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Henry Fleck, MD, Bronx. Died April 6, 1990; age 79. Dr Fleck was a 1936 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Bologna, Bologna, Italy. He was a Diplomate of the American Board of Physical Medicine and Rehabilitation. His memberships included the New York Society for Physical Medicine and Rehabilitation, the Pan American Medical Association, the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

Harold Theodore Fuerst, MD, New York. Died April 13, 1990; age 81. Dr Fuerst was a 1933 graduate of the Jefferson Medical College of Thomas Jefferson University, Philadelphia, Pennsylvania. He was a Diplomate of the American Board of Preventive Medicine, Inc, and a Fellow of the American College of Preventive Medicine. His memberships included the New York Academy of Medicine, the American Public Health Association, the New York County Medical Society, and the Medical Society of the State of New York.

Peter Joseph Gatti, MD, Staten Island. Died April 29, 1990; age 77. Dr Gatti was a 1939 graduate of the Hahnemann Medical College of Philadelphia, Philadelphia, Pennsylvania. He was a member of the Richmond County Medical Society and the Medical Society of the State of New York.

Louis Jack Gelber, MD, Rockville Centre. Died August 1989; age 88. Dr Gelber was a 1928 graduate of the George Washington University School of Medicine, Washington, DC. His memberships included the Radiological Society of New York State, the Radiological Society of North America, Inc, the American Board of Legal Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Elias H. Gerchick, MD, Scarsdale. Died April 29, 1990; age 75. Dr Gerchick was a 1939 graduate of the New York University School of Medicine, New York. He was a Diplomate of the American Board of Psychiatry and Neurology and a Fellow of the American Psychiatric Association. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Lazar L. Gold, MD, Brooklyn. Died January 22, 1990; age 80. Dr Gold was a 1935 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. His memberships included the American Academy of Family Practice, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

George Theodore Golding, MD, Boca Raton, Florida. Died April 10, 1990; age 84. Dr Golding was a 1932 graduate of the Rush Medical College, Chicago, Illinois. He was a Diplomate of the American Board of Urology. His memberships included the American Urological Association, the Pan American Medical Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Morris E. Greenberg, MD, North Bay Village, Florida. Died April 25, 1990; age 83. Dr Greenberg was a 1934 graduate of the New York Medical College, New York. He was a Diplomate of the American Board of Internal Medicine. His memberships included the American Rheumatism Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Maxwell Harrison, MD, Woodmere. Died April 8, 1990; age 78. Dr Harrison was a 1940 recipient of the Registrable Qualification Granted by the Scottish Conjoint Board. He was a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Zubaida Hasan, MD, Staten Island. Died April 18, 1990; age 57. Dr Hasan was a 1956 graduate of the Dow Medical College, Karachi, Federal Capital Area, Pakistan. She was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. She was a member of the Richmond County Medical Society and the Medical Society of the State of New York.

Robert J. Healy, MD, Cutchogue. Died April 28, 1990; age 66. Dr Healy was a 1952 graduate of the New York Medical College, New York. His memberships included the New York State Society of Internal Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Herman Goodman Helpern, MD, New York. Died April 22, 1990; age 81. Dr Helpern was a 1935 graduate of the Rush Medical College, Chicago, Illinois. His memberships included the New York State Society of Internal Medicine; the Clinical Society, New York Diabetes Association; the American Society of Internal Medicine; the New York County Medical Society; and the Medical Society of the State of New York.

John Edward Heslin, MD, Albany. Died March 10, 1990. Dr Heslin was a 1915 graduate of the Albany Medical College of Union University, Albany. He was a Diplomate of the American Board of Urology and a Fellow of the American College of Surgeons. His memberships included the American Urological Association, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

Francis Robert Hunter, MD, Mamaroneck. Died April 4, 1990; age 84. Dr Hunter was a 1934 graduate of the Georgetown University School of Medicine, Washington, DC. His memberships included the New York Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Joseph Hyman, MD, Brooklyn. Died January 2, 1990; age 83. Dr Hyman was a 1933 graduate of the University of Maryland School of Medicine, Baltimore, Maryland. He was a Diplomate of the American Board of Ophthalmology and a Fellow of the American College of Surgeons. His memberships included the American Academy of Ophthalmology and Otolaryngology, the Pan American Association of Ophthalmology, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Stuart Kase, MD, Massapequa Park. Died May 6, 1990; age 56. Dr Kase was a 1959 graduate of the New York Medical College, New York. He was a Diplomate of the American Board of Urology and a Fellow of the American College of Surgeons. His memberships included the New York State Urological Society, the American Fertility Society, the Pan American Medical Association, the American Urological Association, the Nassau County Medical Society, and the Medical Society of the State of New York.

Ernest W. Kulka, MD, New York. Died April 21, 1990; age 93. Dr Kulka was a 1923 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a Diplomate of the American Board of Obstetrics and Gynecology, a Fellow of the American College of Obstetricians and Gynecologists, and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Abbott Lippman, MD, Brooklyn. Died December 12, 1989; age 82. Dr Lippman was a 1933 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a Fellow of the American College of Physicians and a Fellow of the American Psychiatric Association. His memberships included the American Psychoanalytic Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Armond V. Mascia, MD, Tarrytown.

Died April 28, 1990; age 69. Dr Mascia was a 1945 graduate of the New York University School of Medicine, New York. He was a Diplomate of the American Board of Pediatrics and a Diplomate of the American Board of Allergy and Immunology. He was a Fellow of the American Academy of Pediatrics, the American College of Chest Physicians, and the American College of Allergists. His memberships included the American Academy of Allergy, the American Thoracic Society, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Michael A. Muehlbauer, MD, Rego Park. Died January 28, 1990; age 84. Dr Muehlbauer was a 1932 graduate of the Medizinische Fakultaet der Martin Luther Universitaet, Halle, Germany. He was a Fellow of the American College of Gastroenterology and a Fellow of the American College of Physicians. His memberships included the New York State Society of Internal Medicine, the New York Gastroenterological Society, the American Gastro-Enterological Association, the American Society of Internal Medicine, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Thomas James Ravasz, MD, Brooklyn. Died May 4, 1990; age 45. Dr Ravasz was a 1971 graduate of the Medical College of Wisconsin, Milwaukee. He was a Diplomate of the American Board of Psychiatry and Neurology and a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Hyman Rappaport, MD, Flushing. Died April 19, 1990; age 91. Dr Rappaport was a 1923 graduate of the State University of New York Health Science Center at Brooklyn. He was a Fellow of the American Academy of Pediatrics. His memberships included the New York Academy of Medicine, the Endocrine Society, the Society for Pediatric Research, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Douglas H. Robertson, MD, Mount Kisco. Died April 20, 1990; age 75. Dr Robertson was a 1939 graduate of the University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Mitchell I. Rubin, MD, Charleston, South Carolina. Died April 11, 1990. Dr Rubin was a 1925 graduate of the Medical College of South Carolina, Charleston. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Robert Israel Rubinstein, MD, New York. Died May 1, 1990; age 92. Dr Rubinstein was a 1928 graduate of the Yale University School of Medicine, New Haven, Connecticut. His memberships included the American Academy of Family Practice, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Irving Arthur Sarot, MD, Westhampton Beach. Died April 23, 1990; age 80. Dr Sarot was a 1932 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Surgery and the American Board of Thoracic Surgery. He was a Fellow of the American College of Cardiology, the American College of Surgeons, and the American College of Chest Physicians. His memberships included the New York Academy of Medicine, the New York Society for Thoracic Surgery, the Society of Thoracic Surgery, the New York County Medical Society, and the Medical Society of the State of New York.

Earl Saxe, MD, Easton, Connecticut. Died January 14, 1990; age 82. Dr Saxe was a 1934 graduate of the Jefferson Medical College of Thomas Jefferson University, Philadelphia, Pennsylvania. He was a Diplomate of the American Board of Psychiatry and Neurology and a Fellow of the American College of Physicians. His memberships included the New York Neurological Society, the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Stephen J. Schmeiser, Jr, MD, Massapequa. Died April 2, 1990; age 78. Dr Schmeiser was a 1938 graduate of the Georgetown University School of Medicine, Washington, DC. He was a Diplomate of the American Board of Family Practice and a Fellow of the American Academy of Family Practice. His memberships included the New York Academy of Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Lewis F. Scribner, Jr, MD, Syracuse. Died March 24, 1990; age 62. Dr Scribner was a 1954 graduate of the Wayne State University School of Medicine, Detroit, Michigan. He was a Diplomate of the American Board of Internal Medicine and the American Board of Allergy and Immunology. His memberships included the New York State Society of Internal Medicine, the American Academy of Allergy, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Max Leon Som, MD, New York. Died April 24, 1990; age 86. Dr Som was a 1930 graduate of the New York University School of Medicine, New York. He was a Diplomate of the American Board of Otolaryngology and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the American Academy of Otolaryngology, the American Academy of Facial Plastic and Reconstructive Surgery, the American Laryngological Association, the American Society of Head and Neck Surgery, the New York County Medical Society, and the Medical Society of the State of New York.

James Michael Sonsire, MD, Elmira. Died April 29, 1990; age 51. Dr Sonsire was a 1965 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Plastic Surgery and a Diplomate of the American Board of Surgery. He was a Fellow of the American College of Surgeons. His memberships included the American Burn Association, the American Society of Plastic and Reconstructive Surgery, the American Trauma Society, the American Society for Surgery of the Hand, the Medical Society of the County of Chemung, and the Medical Society of the State of New York.

Max Charles Straitiff, MD, Orchard Park. Died March 29, 1990; age 80. Dr Straitiff was a 1940 graduate of the State University of New York Health Science Center at Brooklyn. His memberships included the New York Academy of Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Allen Donald Tanney, MD, New York. Died April 9, 1990; age 79. Dr Tanney was a 1939 graduate of the Loyola University Stritch School of Medicine, Maywood, Illinois. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Cardiology. His memberships included the New York Academy of Medicine, the New York Cardiological Society, the New York State Society of Internal Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Henry Vinicor, MD, Norwood. Died May 19, 1990; age 81. Dr Vinicor was a 1933 graduate of the New York University School of Medicine, New York. He was a member of the Medical Society of the County of St Lawrence and the Medical Society of the State of New York.

Rita G. Wallsh, MD, New York. Died March 28, 1990; age 57. Dr Wallsh was a 1964 graduate of the New York University School of Medicine, New York. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Robert G. Wilson, MD, Edisto Island, South Carolina. Died March 18, 1990; age 98. Dr Wilson was a 1919 graduate of the University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania. He was a member of the Medical Society of the County of Genesee and the Medical Society of the State of New York.

Nahum J. Winer, MD, New York. Died May 24, 1990; age 83. Dr Winer was a 1936 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Internal Medicine. He was a Fellow of the American College of Physicians and a Fellow of the American College of Cardiology. His memberships included the New York Academy of Medicine, the New York Cardiological Society, the American Society of Clinical Pathologists, the Pan American Medical Association, the International Society of Internal Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Morris J. Wolf, MD, New York. Died February 4, 1990; age 84. Dr Wolf was a 1931 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Internal Medicine. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Alfonso Yamin, MD, Yonkers. Died May 7, 1990; age 59. Dr Yamin was a 1955 graduate of the Facultad de Medicina de la Universidad de Santo Domingo, Ciudad Trujillo, Dominican Republic. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

George Eayres York, MD, Schenectady. Died April 23, 1990; age 70. Dr York was a 1947 graduate of the Albany Medical College of Union University, Albany. He was a member of the Medical Society of the County of Schenectady and the Medical Society of the State of New York.

Lynn R. Callin, MD
1908–1990

Lynn R. Callin, MD, of Rochester, NY, and Fort Myers, Florida, died on June 8, 1990. Dr Callin served with distinction as president of the Medical Society of the State of New York (MSSNY) from 1974 to 1975. He had long been a supporter and tireless worker for organized medicine in New York, and served as a MSSNY councilor from 1969 to 1973, as a delegate to the MSSNY from 1967 to 1973, and as a delegate to the American Medical Association for many years.

Dr Callin also served as Clinical Associate Professor of Orthopedic Surgery at the University of Rochester School of Medicine and Dentistry and was formerly chief of the Orthopedic Department of Rochester General Hospital. He had been an active member of the Rochester Regional Health and Hospital Council and Advisor to the Regional Medical Program.

Dr Callin is survived by his wife, Edith Callin, and three children, Rachel C. Spieler, Susan L. Callin, and Randall G. Callin. A memorial service for Dr Callin was held on June 12 at the Asbury First United Methodist Church in Rochester, New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1988; 88:456-462). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al*. Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925-927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337-338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

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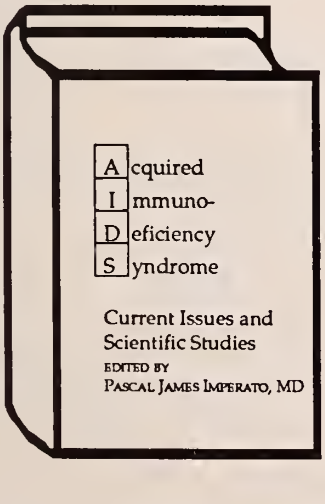
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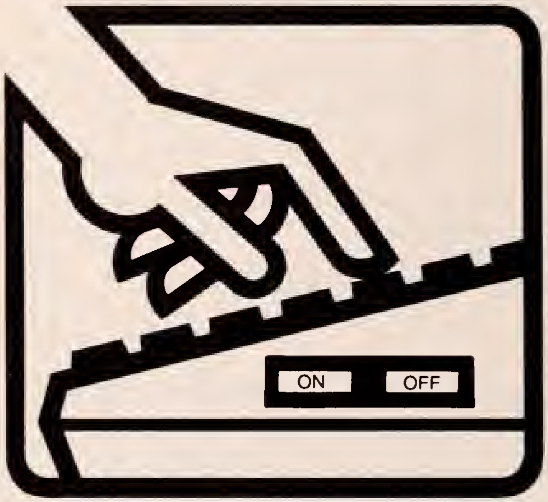

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Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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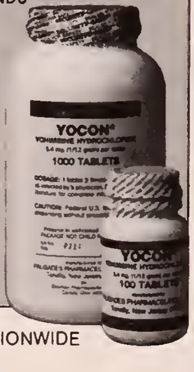

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(*continued from p 6A*)

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Sept 8–11. **The Hilton Head Addictions Conference.** Westin Resort, Hilton Head Island. Contact: Virginia Biegun, Coordinator, Community and Professional Services, The Oxford Institute Network of Care, 825 West Drahner Rd, PO Box 429, Oxford, MI 48051. Tel: (313) 628-0500.

AROUND THE WORLD

AUSTRIA

Sept 12–15. **Musculoskeletal Disorders.** 27 Cat I Credits. Salzburg Convention Center, Salzburg. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

BRAZIL

Sept 18–21. **IV International Conference on Pediatric Pathology.** Sheraton Rio Hotel & Towers, Rio de Janeiro. Contact: Congregare Marketing de Conferencias, Av. Rio Branco, 185 Suite 912, 20040 Rio de Janeiro, Brazil. Tel: (021) 240-9313.

CANADA

Sept 10–14. **Tenth Annual Postgraduate Course—Uroradiology '90.** 19 Cat I Credits. Westin Bayshore Hotel, Vancouver, British Columbia. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

GERMANY

Sept 2–9. **Hands-on Workshop of Phlebology and Sclerotherapy.** Germany and Switzerland. Contact: Anton Butie, MD, 101 South Rancho Santa Fe Rd, #302, Encinitas, CA 92024. Tel: (619) 942-8986.

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Sept 5–8. **3rd International Symposium on Anesthesia for Cardiac Patients.** Budapest Hilton. Contact: George Silvay, MD, PhD, Department of Anesthesiology, Box 1010, Mount Sinai School of Medicine, 1 Gustave L. Levy Place, New York, NY 10029. Tel: (212) 241-7467. □

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This seven day program is designed to provide a **comprehensive review** of the fund of knowledge necessary to **enable successful completion of the certification examinations** offered by the American Board of Emergency Medicine (ABEM). The official complete schedule of examination dates is available from the ABEM. Candidates for whom this course may be most helpful are those scheduled for: written recertification examination on November 4, 1990, written initial certifying examination on November 5, 1990, or the oral initial certifying examination on January 27-30, 1991. This course is recommended for both the established physician-in-practice and the physician who has recently completed residency training. It is anticipated that course participants will be near completion of a self-directed plan of study. Some written board candidates have chosen to begin their plan of study a year in advance by taking this course, others appreciate a **structured** and **systematic review** just **days prior to the written certifying exam.**

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The response to our course offering in the past has been most gratifying. **We** have **listened to** the **feedback** provided by prior registrants and course faculty, and this led to several new features. This year we will include a formal didactic session on preparation for the oral exam. Additionally **elective** opportunity for private **oral board preparatory tutorials** and **small group oral board simulation** will be available during the program. The underlying philosophy of our program is unchanged and our primary focus will remain the written board exam candidate. There are many resources from which to learn the information suggested by the core content curriculum. Among them are Rosen's Textbook of Emergency Medicine, the ATLS/ACLS manuals and numerous other texts, monographs and journals. This year we have chosen to present the core content information in a format that correlates organizationally with the text, Emergency Medicine: A Comprehensive Study Guide - American College of Emergency Physicians, edited by Tintinalli, Krome and Ruiz. Finally, we have added a three component special session which will provide practical suggestions and strategies on **how to achieve optimal performance on your examination day.**

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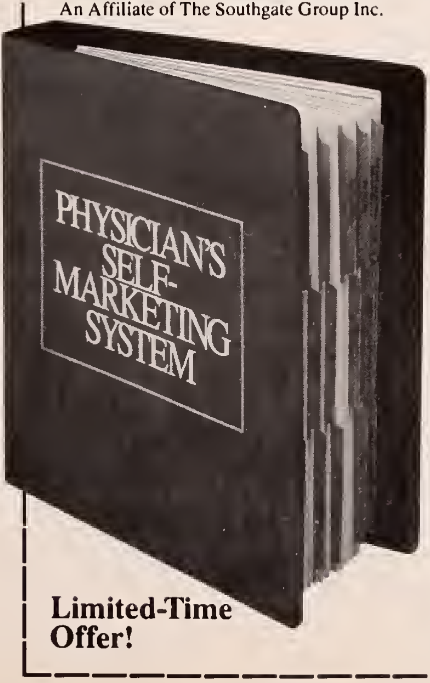

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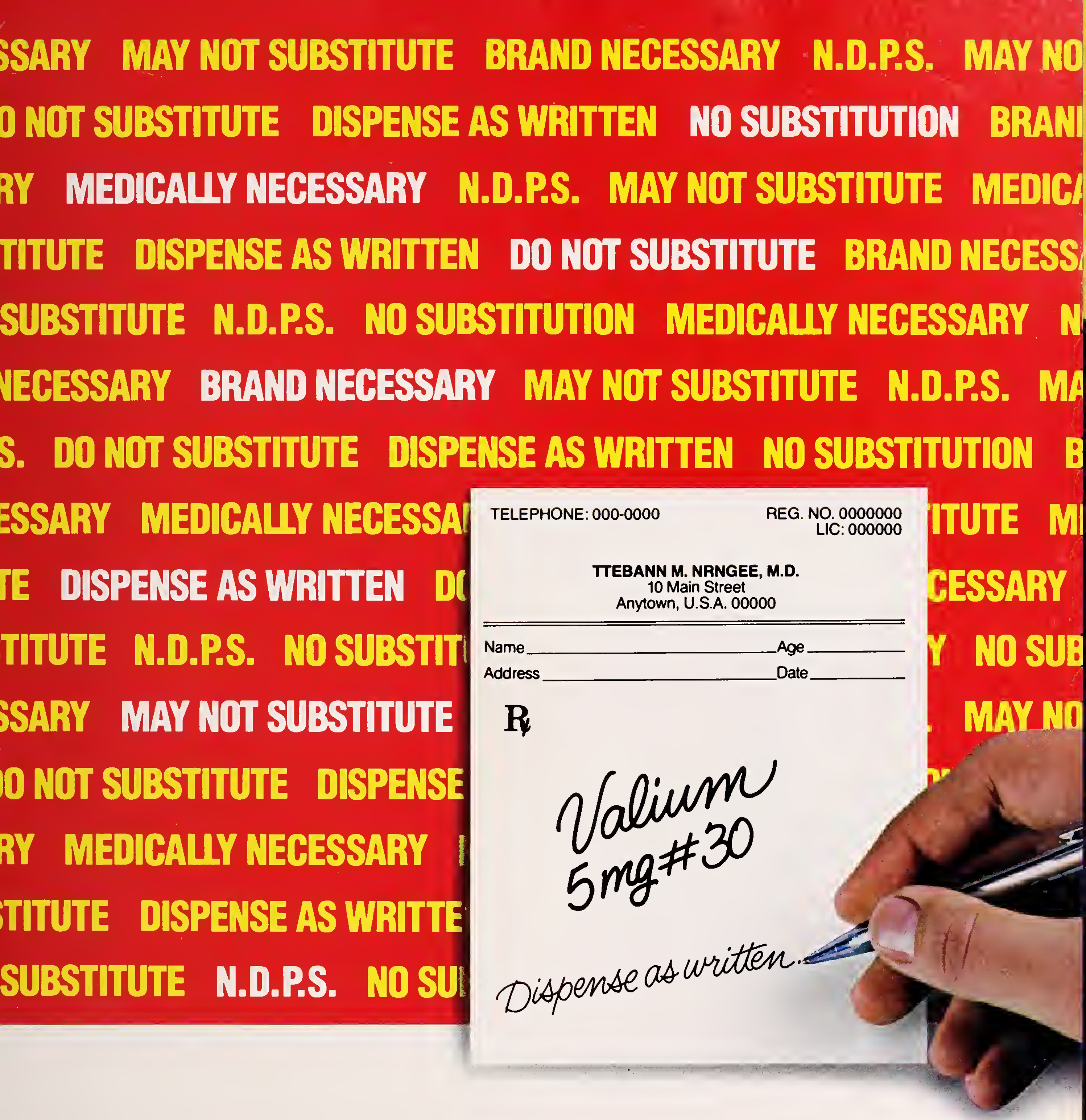
Index to Advertisers

AMA Advisers 20A
Classified Advertising 12A, 17A, 20A, 21A, 22A
Cutter Biological 8A
Eli Lilly and Company 10A
Marine Midland Bank .... 2nd Cover
Medical Directors Advancement Council 6A
MEGASYS, Ltd. 15A
Med Pro 14A
Palisades Pharmaceuticals 16A
Roche Laboratories .... 3rd & 4th Cover
Searle Pharmaceuticals 1A, 2A, 3A, 4A
Self-Marketing Management 19A
U.S. Air Force 12A, 21A
U.S. Army Reserves 7A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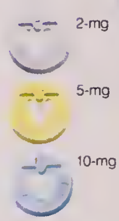

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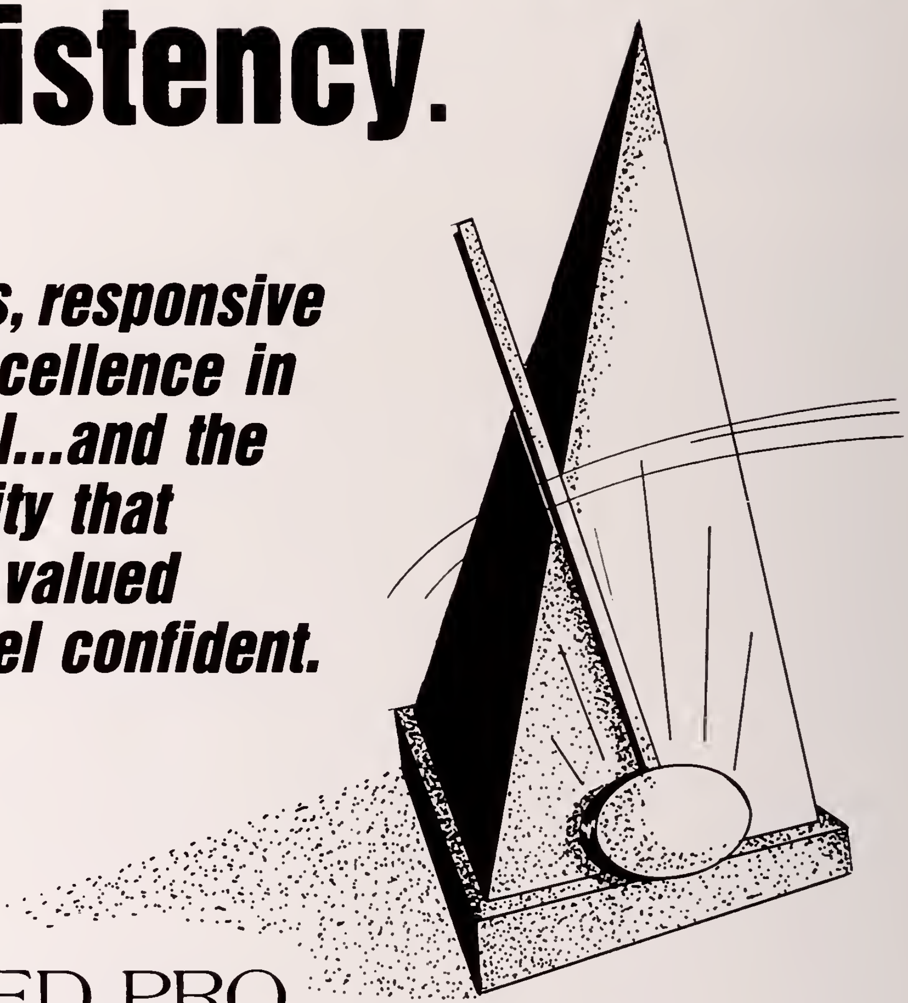

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Hydrocodone Signs and Symptoms: Serious overdose with hydrocodone is characterized by respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, (cyanosis), extreme somnolence progressing to stupor or coma, skeletal muscle flaccidity, cold and clammy skin, and sometimes bradycardia and hypotension. In severe overdosage, apnea, circulatory collapse, cardiac arrest and death may occur.

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Oct 31. **A Halloween Walk Down Cemetery Lane: Historic Review of Celebrity Deaths in New York.** Lehman College campus. Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 511 Hempstead Ave, Suite 2, West Hempstead, NY 11552. Tel: (516) 292-2300.

COOPERSTOWN

Oct 10. **Controversial Areas of Drug Use.** 3 Cat I Credits. Bassett Hall Conference Center. Contact: Charlotte L. Hoag, Medical Education Coordinator, Office of Medical Education, The Mary Imogene Bassett Hospital, One Atwell Rd, Cooperstown, NY 13326. Tel: (607) 547-3926.

EAST ELMHURST

Oct 5 or Oct 6. **Emergency Medicine Oral Board Review Course.** 7 Cat I Credits. The LaGuardia Marriott Hotel. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

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Oct 19. **The Michael M. Axinn Fifth Annual Memorial Conference: Prejudice: Historical, Cultural, & Psychological Perspectives.** CME credits pending. North Shore University Hospital-Cornell University Medical College. Contact: Linda Haberman, MA, Conference Coordinator, Department of Health Education/Community Affairs, North Shore University Hospital, 300 Community Dr, Manhasset, NY 11030. Tel: (516) 562-3045.

MANHATTAN

Oct 3–6. **MRI: Clinical State of the Art 1990.** 23½ Cat I Credits. NYU Medical Center. *Oct 5.* **Strengthening and Retaining Nurses: How to Implement a Professional Self Development Program for Nurses.** 5¾ Cat I Credits. NYU Medical Center. *Oct 8–12.* **Five Day Emergency Medicine Board Review Course.** 39 Cat I Credits. NYU Medical Center. *Oct 15–16.* **The Third Conference on DNA Topoisomerases in Therapy: Therapy of Infections.** 14 Cat I Credits. Alumni Hall, NYU Medical Center. *Oct 16–18.* **The Third Conference on DNA Topoisomerases in Therapy: Therapy of Cancer.** 18 Cat I Credits. Alumni Hall, NYU Medical Center. Contact NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Oct 4. **Lucie Rudd Memorial Lecture.** Department of Pediatrics, St Luke's/Roosevelt Hospital Center. Contact: Pediatric Primary/Ambulatory Care Office, St Luke's/Roosevelt Hospital Center, 428 West 59th St, New York, NY 10019. Tel: (212) 523-6300.

Oct 10–13. **Renal Biopsy in Medical Diseases of the Kidney.** 23 ½ Cat I Credits. College of Physicians & Surgeons of Columbia University. Contact: Center for Continuing Education, College of Physicians & Surgeons of Columbia University, 630 West 168th St, New York, NY 10032. Tel: (212) 305-3682.

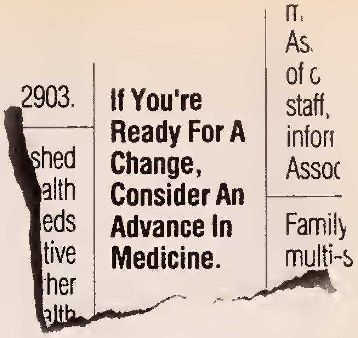
Oct 25–26. **13th Annual Current Concerns in Adolescent Medicine.** 12 Cat I Credits. The New York Hilton. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

AROUND THE NATION

ALABAMA

Oct 19–21. **Neuroradiology Update with Emphasis on Magnetic Resonance Imaging.** 18 Cat I Credits. Wynfrey Hotel, Birmingham. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

(*continued on p 16A*)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Contents

COMMENTARIES

439 **The utility of cross-cultural health research**
FERNANDO M. TREVINO, PHD

440 **The present and future importance of mortality statistics**
RICHARD J. HAVLIK, MD, MPH

RESEARCH PAPERS

442 **Mortality in the Hispanic population of Suffolk County, New York**
ANTHONY P. POLEDNAK, PHD

446 **Small bowel injuries following blunt abdominal trauma: Early recognition and management**
EDWARD J. COLEMAN, MD, PATRICK A. DIETZ, MD

HISTORY

450 **A history of the New York Society of Tropical Medicine**
PASCAL JAMES IMPERATO, MD

CASE REPORTS

463 **Lofgren's syndrome (acute sarcoidosis) sine erythema nodosum mimicking acute rheumatoid arthritis**
LEE D. KAUFMAN, MD

465 **Extraparotid Warthin's tumor: An unexpected finding in three female patients**
MIGUEL A. RODRIGUEZ BIGAS, MD; KUMAO SAKO, MD;
MOHAMED S. RAZACK, MD; NIEVA B. CASTILLO, MD

467 **Superior vena cava syndrome and right pleural effusion due to giant goiter**
PAUL COHEN, MD; SUBRAHMANYA BHAT, MD; KIRAN BHAT, MD;
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MD; STEPHAN L. KAMHOLZ, MD

(continued on page 8A)

Because safety cannot be taken for granted in H_2-antagonist therapy

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nizatidine

Minimal potential for drug interactions

Unlike cimetidine and ranitidine,[1] Axid does not inhibit the cytochrome P-450 metabolizing enzyme system.[2]

Swift and effective H_2-antagonist therapy

- *Most patients experience pain relief with the first dose[3]*
- *Heals duodenal ulcer rapidly and effectively[4,5]*
- *Dosage for adults with active duodenal ulcer is 300 mg once nightly (150 mg b.i.d. is also available)*

References

1. *USP DI Update, September/October 1988, p 120.*
2. *Br J Clin Pharmacol 1985;20:710-713.*
3. *Data on file, Lilly Research Laboratories.*
4. *Scand J Gastroenterol 1987;22(suppl 136):61-70.*
5. *Am J Gastroenterol 1989;84:769-774.*

AXID®
nizatidine capsules

Brief Summary. Consult the package literature for complete information.

Indications and Usage: 1. *Active duodenal ulcer*–for up to eight weeks of treatment. Most patients heal within four weeks.

2. *Maintenance therapy*–for healed duodenal ulcer patients at a reduced dosage of 150 mg h.s. The consequences of therapy with Axid for longer than one year are not known.

Contraindication: Known hypersensitivity to the drug. Use with caution in patients with hypersensitivity to other H_2-receptor antagonists.

Precautions: *General*–1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Dosage should be reduced in patients with moderate to severe renal insufficiency.

3. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

Laboratory Tests–False-positive tests for urobilinogen with Multistix® may occur during therapy.

Drug Interactions–No interactions have been observed with theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450 enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increased serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility–A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy–Teratogenic Effects–Pregnancy Category C–Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers–Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use–Safety and effectiveness in children have not been established.

Use in Elderly Patients–Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of varying durations included almost 5,000 patients. Among the more common adverse events in domestic placebo-controlled trials of over 1,900 nizatidine patients and over 1,300 on placebo, sweating (1% vs 0.2%), urticaria (0.5% vs <0.01%), and somnolence (2.4% vs 1.3%) were significantly more common with nizatidine. It was not possible to determine whether a variety of less common events was due to the drug.

Hepatic–Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to three times the upper limit of normal, however, did not significantly differ from that in placebo patients. Hepatitis and jaundice have been reported. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular–In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS–Rare cases of reversible mental confusion have been reported.

Endocrine–Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with equal frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic–Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental–Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity–As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Because cross-sensitivity among this class has been observed, H_2-receptor antagonists should not be administered to those with a history of hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other–Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance by approximately 84%.

PV 2098 AMP [091289]

Additional information available to the profession on request.

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LETTERS TO THE EDITOR

469 **Make way for tomorrow**
RANDALL D. BLOOMFIELD, MD

469 **The use of free phenytoin levels in averting phenytoin toxicity**
MARK FORREST GORDON, MD, DAN GERSTENBLITT, MD

470 **Capgras' syndrome**
JEROME M. SCHNECK, MD

ESSAY REVIEW

472 **Country doctor**
ROBERT L. PERKEL, MD

473 LEADS FROM EPIDEMIOLOGY NOTES

476 BOOK REVIEWS

480 NEWS BRIEFS

482 OBITUARIES

484 GUIDELINES FOR AUTHORS

5A MEDICAL MEETINGS AND LECTURES

Retrovir® (zidovudine) significantly delays the onset of AIDS in HIV-infected *asymptomatic* patients (CD4 ≤500 cells/mm^3) ...at 500 mg/day.

Now more than ever–
test, monitor, and treat
when appropriate.

See last pages for brief summary of full prescribing information.

Retrovir significantly delays progression to HIV disease. A recent clinical trial was stopped and patients receiving placebo were offered Retrovir at 500 mg/day.[1]

In a multicenter, placebo-controlled trial carried out in 32 major medical centers, involving 1338 asymptomatic HIV-infected patients (CD4 ≤500 cells/mm^3), RETROVIR was shown to slow HIV disease progression significantly.

The incidence and rates of progression to AIDS in patients receiving RETROVIR at 500 or 1500 mg/day in this trial were less than half those reported with placebo.[1] This significant difference resulted in early termination of the trial and the offering of RETROVIR to patients on placebo.

Patients in this study were monitored for an average of one year. Although treatment of asymptomatic patients delays progression of disease in those patients at risk of progression over the initial 1 to 2 years of treatment, it is not known whether early treatment prolongs survival.

Patients receiving Retrovir were statistically significantly less likely to progress to AIDS.[1]

Treatment Group	Progressions to AIDS	Rate of Progression*
Placebo (n=428)	33	6.6
RETROVIR 500 mg/day (n=453)	11	2.3

* Events per 100 person-years of observation.

Changes in immunologic and virologic parameters paralleled the observed clinical benefits.

The recommended daily dose of RETROVIR in asymptomatic patients with CD4 ≤500 cells/mm^3 is 500 mg. This lower dose offers patients efficacy, fewer and less severe adverse events than higher doses, and important cost savings.

Now more than ever, you can do something about HIV infection and disease. It is in your hands to test high-risk patients, monitor patients who test HIV-positive, and treat when appropriate with RETROVIR.

Now more than ever– test, monitor, and treat when appropriate.

Retrovir at 500 mg/day is generally well tolerated.*

All reported clinical adverse events were recorded, including those due to underlying infection.

Difference in Adverse Events From Baseline Placebo.

	RETROVIR 500 mg/day (n=453)	Placebo (n=428)	% Difference
Headache	62.5%	52.6%	**9.9%**
Malaise	53.2%	44.9%	**8.3%**
Anorexia	20.1%	10.5%	**9.6%**
Nausea	51.4%	29.9%	**21.5%**
Vomiting	17.2%	9.8%	**7.4%**

*Patients were monitored for tolerance to the study regimens for an average of one year. Difference in adverse events from baseline incidence with placebo (percent of patients). Differences were statistically significant for all events listed.

RETROVIR has been studied in controlled trials in significant numbers of asymptomatic and symptomatic HIV-infected patients, but only for limited periods of time. Therefore, the full safety and efficacy profile of RETROVIR has not been defined, particularly in regard to prolonged use and especially in HIV-infected individuals who have less advanced disease.

Hematologic adverse events with Retrovir in this study were not reported at a statistically significant greater incidence than placebo.[1]

The incidence of hematologic adverse events among asymptomatic patients receiving RETROVIR is lower than would be expected for patients with advanced symptomatic HIV disease.

Although hematologic abnormalities may cause dosing limitations in some patients, temporary dose reductions or interruptions in therapy can help maintain patients on long-term therapy.

The recommended daily dose of RETROVIR in asymptomatic HIV-infected patients (CD4 $\leq$500 cells/mm^3) is 500 mg since it offers patients efficacy, fewer and less severe adverse events than higher doses, and important cost savings.

Now more than ever, you can do something about HIV infection and disease. It is in your hands to test high-risk patients, monitor patients who test HIV-positive, and treat when appropriate with RETROVIR.

RETROVIR®

(zidovudine)

CAPSULES AND SYRUP

See last pages for brief summary of full prescribing information.

RETROVIR® Capsules
RETROVIR® Syrup
(ZIDOVUDINE)

Wellcome

Brief Summary

> **WARNING: THERAPY WITH RETROVIR (ZIDOVUDINE) MAY BE ASSOCIATED WITH HEMATOLOGIC TOXICITY INCLUDING GRANULOCYTOPENIA AND SEVERE ANEMIA REQUIRING TRANSFUSIONS (SEE WARNINGS).**
>
> IN ADDITION, PATIENTS TREATED WITH ZIDOVUDINE MAY CONTINUE TO DEVELOP OPPORTUNISTIC INFECTIONS (OI'S) AND OTHER COMPLICATIONS OF HIV INFECTION AND, THEREFORE SHOULD BE UNDER CLOSE CLINICAL OBSERVATION

Microbiology: ...The development of resistance to zidovudine has not been adequately studied and the frequency of zidovudine-resistant isolates existing in the general population is unknown. Reduced *in vitro* sensitivity to zidovudine has been observed, however, in viral isolates from some individuals who have received prolonged courses of zidovudine treatment. For the small number of patients from whom isolates where studied, no correlations were evident between the development of reduced sensitivity in the laboratory and clinical response Therefore, the quantitative relationship between *in vitro* susceptibility of HIV to zidovudine and clinical response to therapy has not been established Studies of the development of resistance to zidovudine are incomplete and the frequency and degree of changes in *in vitro* sensitivity of virus isolates from HIV infected patients with differing severity of immune compromise are unknown...

INDICATIONS AND USAGE: Retrovir is indicated for the management of patients with HIV infection who have evidence of impaired immunity (CD4 cell count of 500/mm³ or less) before therapy is begun.

This indication is based on the results of three randomized, double-blind, placebo-controlled trials of oral zidovudine conducted in the United States, in HIV-infected patients with CD4 counts of 500/mm³ or less. Separate studies evaluated zidovudine therapy in asymptomatic HIV-infected individuals, in patients with early symptomatic HIV disease, and in patients with advanced symptomatic HIV disease (AIDS or advanced ARC).

Asymptomatic HIV Infection: A randomized, double-blind, placebo-controlled trial of oral zidovudine was conducted in asymptomatic, HIV-infected individuals at 32 medical centers Entry was dependent on the absence of signs and symptoms consistent with HIV disease, such as significant weight loss, fever, diarrhea, secondary infections and evidence of neurological dysfunction. In the study, 1338 asymptomatic individuals with absolute CD4(T4) lymphocyte counts of less than 500 cells/mm³ received a regimen of either 100 mg zidovudine, 300 mg zidovudine, or placebo, each administered five times a day These study participants were monitored for the development of signs and symptoms of HIV disease and tolerance to the study regimens. The study was terminated early because of a statistically significant difference in progression to advanced symptomatic HIV disease (AIDS or advanced ARC) between the 500 mg/day zidovudine group and the placebo group Of the 1338 patients enrolled in the study, 38 of the 428 patients receiving placebo, 17 of the 453 individuals receiving 500 mg zidovudine, and 19 of the 457 recipients of 1500 mg zidovudine progressed to advanced symptomatic HIV disease when followed for a mean of 55 weeks (range 19-107 weeks) Of the progressions noted above, AIDS occurred in 33 placebo recipients, 11 individuals receiving 500 mg and 14 patients receiving 1500 mg zidovudine Changes in immunologic and virologic parameters (ie, CD4[T4] lymphocyte count and serum p24 antigen levels) paralleled the observed clinical benefits Consistent with various epidemiologic studies of individuals with HIV infection, patients in this study with lower CD4 cell counts or P24 antigenemia were more likely to progress to AIDS. Virtually all patients (for whom data are available) experienced a decline in CD4 cell counts to less than 200 per mm³ before developing an AIDS-defining opportunistic infection Although treatment of asymptomatic patients delays progression of disease in those patients at risk of progression over the initial 1-2 years of treatment, it is not known whether early treatment prolongs survival

Zidovudine was well tolerated in a majority of patients. The following adverse clinical events were reported at a significantly greater incidence in individuals receiving 500 mg/day of zidovudine versus placebo: headache, malaise, anorexia, nausea and vomiting (see ADVERSE REACTIONS).

The two most common laboratory abnormalities reported were anemia and granulocytopenia, each of which occurred more often in patients receiving higher doses These toxicities were easily managed in most cases by temporary dose interruption.

Early Symptomatic HIV Disease: This randomized, double-blind, placebo-controlled trial of oral zidovudine, conducted at 29 medical centers, studied 713 patients with early manifestations of HIV disease (ie, a baseline CD4[T4] cell count of 200 to 800/mm³ and symptoms such as oral thrush, oral hairy leukoplakia or intermittent diarrhea) Patients received either placebo or 200 mg zidovudine every 4 hours (1200 mg/day). The trial was terminated early because of a statistically significant difference in the rates of development of advanced symptomatic HIV disease between the zidovudine and placebo groups. Of the 352 patients receiving placebo, 36 progressed to advanced symptomatic HIV disease, of whom 21 progressed to AIDS; whereas of the 361 zidovudine recipients, 13 progressed to advanced symptomatic HIV disease, of whom 5 progressed to AIDS.

Minimal toxicity was observed Four percent of zidovudine recipients compared with none of the placebo recipients developed a hemoglobin concentration less than 8 g/dL and 4% of patients treated with zidovudine compared with 1% of placebo recipients developed a granulocyte count less than 750 cells/mm³. Asthenia, dyspepsia, nausea, and vomiting were the major adverse clinical events reported at significantly greater incidences in patients receiving zidovudine.

Advanced Symptomatic HIV Disease: This randomized, double-blind, placebo-controlled trial, conducted at 12 medical centers, was the original trial upon which the marketing of Retrovir was based In this trial, 281 patients with advanced symptomatic HIV infection including AIDS were studied for a mean of four and a half months.

The patient population of this controlled trial consisted of 160 patients with AIDS (85 Retrovir and 75 placebo) who had recovered from their first episode of PCP diagnosed within the previous four months and 121 patients with ARC (59 Retrovir and 62 placebo) with multiple signs and symptoms of HIV infection, including mucocutaneous candidiasis and/or unexplained weight loss (≥ 15 lbs or >10% of prior body weight). All patients had evidence of impaired cellular immunity with an absence of delayed cutaneous hypersensitivity and CD4 (T4) lymphocytes less than 500 cells/mm³. All patients began therapy at a dose of 250 mg every 4 hours around the clock. This dosage was reduced or temporarily or permanently discontinued after serious marrow toxicity occurred. The trial was stopped early because of a statistically significant difference in mortality There were 19 deaths in the placebo group and 1 in the Retrovir group. All deaths were apparently due to opportunistic infections (OI) or other complications of HIV infection. Treatment duration ranged from 12 weeks to 26 weeks, with a mean and median duration of 17 and 18 weeks, respectively

Retrovir also significantly reduced the risk of acquiring an AIDS-defining OI in patients after 6 weeks of treatment. In addition, patients who received Retrovir generally did better than the placebo group in terms of several other measures of efficacy including performance level, neuropsychiatric function, maintenance of body weight and the number and severity of symptoms associated with HIV infection. A small, transient increase in mean CD4 (T4) counts was seen in the zidovudine group but the significance of this finding is unclear.

The most significant adverse reaction noted in the study was a depression of formed elements in the peripheral blood, which necessitated dose reduction or drug discontinuation in 34% of patients receiving Retrovir Patients with lower CD4 counts were more likely to receive transfusions. 41% of Retrovir recipients and 16% of placebo recipients with <200 CD4 lymphocytes at entry were transfused (see ADVERSE REACTIONS). Only one of 30 Retrovir recipients and none of 30 placebo recipients with >200 CD4 lymphocytes required transfusion.

Although mean platelet counts in Retrovir recipients were statistically increased compared to mean baseline values, thrombocytopenia did occur in some patients. Twelve percent (12%) of Retrovir recipients compared to 5% of placebo recipients had >50% decreases from baseline platelet count.

At the conclusion of the placebo-controlled trial, approximately 80% of Retrovir and placebo recipients elected to enroll in an uncontrolled extension protocol in which all patients received Retrovir at a dose of 200 mg every four hours. This dose was chosen because of concern about hematologic toxicity and to allow for greater flexibility in dosing These patients have been followed for variable periods of time As the follow-up period lengthened, patient tracking became increasingly difficult The intended treatment period of the original study was six months; however, some follow-up data are available for over 90% of patients originally enrolled in the trial Data from patients in this and other studies show no new or unexpected clinical or laboratory adverse experiences for patients receiving Retrovir other than those listed (see ADVERSE REACTIONS), nor have previously reported adverse events increased significantly in frequency or severity with prolonged drug administration. At any time on study, approximately half of the patients received the recommended dose of zidovudine, while the remaining patients required reduction or interruption of their dosage regimen in response to myelosuppression and/or other clinical adverse events.

Benefits of therapy with Retrovir were observed during this extended period, although opportunistic infections continued to occur and additional patients died Survival for all patients originally randomized to receive Retrovir was 96.5% at six months, 84.7% at one year, 68.3% at 18 months and 41.2% at two years. One year and two year survival for patients who entered the trial with AIDS was 79% and 31% when calculated since initiation of therapy (87% and 43%, respectively, since the first diagnosis of PCP). These survival rates were determined by the "intention to treat" method, which assumes all patients assigned to a drug actually took the drug throughout the study period.

While a direct comparison with survival data from other cohorts is not possible, untreated patients with AIDS diagnosed in San Francisco in 1985 who had survived 60 days after PCP had a one year survival of 34.7% and a two year survival of 4.2% from diagnosis of PCP In a recent epidemiologic study of patients with AIDS diagnosed in San Francisco in 1986 and 1987, median survival was improved for patients receiving Retrovir compared to those not receiving therapy with Retrovir (21.6 vs 14.9 months). Actual survival of untreated patients is likely to be lower than reported because of the difficulty in complete ascertainment of mortality Caution is advised in making comparisons from such "natural history" experience since case definitions and follow-up practices vary

Other, uncontrolled studies have shown that Retrovir (zidovudine) may be of benefit in treating women, intravenous drug users, and racial minorities, in addition to the patient population (primarily white males) included in the controlled trials

Dose Comparison Study: Results from a randomized, unblinded, dose comparison study of zidovudine, in patients with AIDS who had experienced an episode of PCP, indicate that an induction dose of zidovudine 200 mg administered orally every four hours (1200 mg/day) for one month, followed by chronic administration of 100 mg every 4 hours (600 mg/day), was associated with survival rates and frequency of opportunistic infections comparable to those observed in patients administered higher dosages as tolerated The 600 mg per day regimen was also associated with a lower incidence of hematologic toxicity The effectiveness of this lower dose in improving the neurologic dysfunction associated with HIV disease, however, is unknown (see DOSAGE AND ADMINISTRATION)

CONTRAINDICATIONS: Retrovir Capsules and Syrup are contraindicated for patients who have potentially life-threatening allergic reactions to any of the components of the formulations

WARNINGS: Zidovudine has been studied in controlled trials in significant numbers of asymptomatic and symptomatic HIV infected patients, but only for limited periods of time Therefore, the full safety and efficacy profile of zidovudine has not been defined, particularly in regard to prolonged use and especially in HIV infected individuals who have less advanced disease (see following sections for more specific information: INDICATIONS AND USAGE, Microbiology, Carcinogenesis, Mutagenesis, Impairment of Fertility).

Zidovudine should be used with extreme caution in patients who have bone marrow compromise evidenced by granulocyte count <1000/mm³ or hemoglobin <9.5 g/dL. In all of the placebo-controlled studies, but most frequently in patients with advanced symptomatic HIV disease, anemia and granulocytopenia were the most significant adverse events observed (see ADVERSE REACTIONS). There have been reports of pancytopenia associated with the use of zidovudine, which was reversible in most instances after discontinuance of the drug.

Significant anemia most commonly occurred after 4 to 6 weeks of therapy and in many cases required dose adjustment, discontinuation of zidovudine, and/or blood transfusions Frequent blood counts are strongly recommended in patients with advanced HIV disease taking zidovudine. For asymptomatic HIV-infected individuals and patients with early HIV disease, most of whom have better marrow reserve, blood counts may be obtained less frequently, depending upon the patient's overall status If anemia or granulocytopenia develops, dosage adjustments may be necessary (see DOSAGE AND ADMINISTRATION).

Sensitization reactions, including anaphylaxis in one patient, have been reported in individuals receiving zidovudine therapy Patients experiencing a rash should undergo medical evaluation.

Coadministration of zidovudine with other drugs metabolized by glucuronidation should be avoided because the toxicity of either drug may be potentiated (see Drug Interactions under PRECAUTIONS). Zidovudine recipients who used acetaminophen during the controlled trial in advance HIV disease, had an increased incidence of granulocytopenia which appeared to be correlated with the duration of acetaminophen use.

PRECAUTIONS:

General: Zidovudine is eliminated from the body primarily by renal excretion following metabolism in the liver (glucuronidation) There are currently very little data available concerning the use of zidovudine in patients with impaired renal function (see Pharmacokinetics subsection of CLINICAL PHARMACOLOGY) and no data in patients with impaired hepatic function These patients may be at greater risk of toxicity from zidovudine

Information for Patients: See full prescribing information.

Drug Interactions: The interaction of other drugs with zidovudine has not been studied in a systematic manner. Coadministration of zidovudine with drugs that are nephrotoxic, cytotoxic, or which interfere with RBC/WBC number or function (eg, dapsone, pentamidine, amphotericin B, flucytosine, vincristine, vinblastine, adriamycin, or interferon) may increase the risk of toxicity Limited data suggest that probenecid may inhibit glucuronidation and/or reduce renal excretion of zidovudine In addition, other drugs (eg, acetaminophen, aspirin, or indomethacin) may competitively inhibit glucuronidation (see WARNINGS). Phenytoin levels have been reported to be low in some patients receiving zidovudine, while in one case a high level was documented These observations suggest that phenytoin levels should be carefully monitored in patients receiving zidovudine since many patients with advanced HIV infections have CNS conditions which may predispose to seizure activity

Some experimental nucleoside analogues which are being evaluated in HIV-infected patients may affect RBC/WBC number or function and may increase the potential for hematologic toxicity of zidovudine. Some experimental nucleoside analogues affecting DNA replication antagonize the *in vitro* antiviral activity of zidovudine against HIV and thus, concomitant use of such drugs should be avoided

Some drugs such as trimethoprim-sulfamethoxazole, pyrimethamine, and acyclovir may be necessary for the management or prevention of opportunistic infections. In the controlled trial, in patients with advanced HIV disease, increased toxicity was not detected with limited exposure to these drugs. However, there is one published report of neurotoxicity (profound lethargy) associated with concomitant use of zidovudine and acyclovir.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Zidovudine was administered orally at three dosage levels to separate groups of mice and rats (60 females and 60 males in each group). Initial single daily doses were 30, 60 and 120 mg/kg/day in mice and 80, 220 and 600 mg/kg/day in rats. The doses in mice were reduced to 20, 30 and 40 mg/kg/day after day 90 because of treatment-related anemia, whereas in rats only the high dose was reduced to 450 mg/kg/day on day 91 and then to 300 mg/kg/day on day 279

In mice, seven late-appearing (after 19 months) vaginal neoplasms (5 non-metastasizing squamous cell carcinomas, one squamous cell papilloma and one squamous polyp) occurred in animals given the highest dose One late-appearing squamous cell papilloma occurred in the vagina of a middle dose animal. No vaginal tumors were found at the lowest dose

In rats, two late-appearing (after 20 months), non-metastasizing vaginal squamous cell carcinomas occurred in animals given the highest dose. No vaginal tumors occurred at the low or middle dose in rats.

No other drug-related tumors were observed in either sex of either species

It is not known how predictive the results of rodent carcinogenicity studies may be for man. At doses that produced tumors in mice and rats, the estimated drug exposure (as measured by AUC) was approximately 3 times (mouse) and 24 times (rat) the estimated human exposure at the recommended therapeutic dose of 100 mg every 4 hours

No evidence of mutagenicity (with or without metabolic activation) was observed in the Ames *Salmonella* mutagenicity assay In a mutagenicity assay conducted in L517BY/TK$^{+/-}$ mouse lymphoma cells, zidovudine was weakly mutagenic in the absence of metabolic activation only at the highest concentrations tested (4000 and 5000 μg/mL). In the presence of metabolic activation, the drug was weakly mutagenic at concentrations of 1000 μg/mL and higher. In an *in vitro* mammalian cell transformation assay, zidovudine was positive at concentrations of 0.5 μg/mL and higher. In an *in vitro* cytogenetic study performed in cultured human lymphocytes, zidovudine induced dose-related structural chromosomal abnormalities at concentrations of 3 μg/mL and higher No such effects were noted at the two lowest concentrations tested, 0.3 and 1 μg/mL In an *in vivo* cytogenetic study in rats given a single intravenous injection of zidovudine at doses of 37.5 to 300 mg/kg, there were no treatment-related structural or numerical chromosomal alterations in spite of plasma levels that were as high as 453 μg/mL five minutes after dosing

In two *in vivo* micronucleus studies (designed to measure chromosome breakage or mitotic spindle apparatus damage) in male mice, oral doses of zidovudine of 100 to 1000 mg/kg/day administered once daily for approximately 4 weeks induced dose-related increases in micronucleated erythrocytes Similar results were also seen after 4 or 7 days of dosing at 500 mg/kg/day in rats and mice

No effect on male or female fertility (judged by conception rates) was seen in rats given zidovudine orally at doses up to 450 mg/kg/day

Pregnancy: Pregnancy Category C. Oral teratology studies in the rat and in the rabbit at doses up to 500 mg/kg/day revealed no evidence of teratogenicity with zidovudine. The incidence of fetal resorptions was increased in rats given 150 or 450 mg/kg/day and rabbits given 500 mg/kg/day The doses used in the teratology studies resulted in peak plasma concentrations in rats of 68 to 234 times the peak human plasma concentrations and in rabbits of 14 to 90 times the peak human plasma concentrations. It is not known whether zidovudine can cause fetal harm when administered to a pregnant woman or can affect reproductive capacity Zidovudine should be given to a pregnant woman only if clearly needed

Nursing Mothers: It is not known whether zidovudine is excreted in human milk Because many drugs are excreted in human milk and because of the potential for serious adverse reactions from zidovudine in nursing infants, mothers should be instructed to discontinue nursing if they are receiving zidovudine.

Pediatric Use: The effectiveness of zidovudine in patients 12 years and younger has not been confirmed. Experience from Phase 1 studies shows that zidovudine administered orally at doses of 120 to 180 mg/m² every six hours (480 to 720 mg/m² per day) appears to give a side effect profile similar to adults. Overall, the pharmacokinetics of zidovudine in children, 6 months through 12 years of age, are similar to those observed in adult patients.

ADVERSE REACTIONS: The frequency and severity of adverse events associated with the use of zidovudine are greater in patients with more advanced infection at the time of initiation of therapy. The following tables summarize the relative incidence of hematologic adverse events observed in the placebo-controlled clinical studies by severity of HIV disease present at the start of treatment:

Asymptomatic HIV Infection Study (n=1338)	Granulocytopenia (<750/mm³)			Anemia (Hgb<8 g/dL)		
	Zidovudine		Placebo	Zidovudine		Placebo
	1500 mg*	500 mg		1500 mg*	500 mg	
CD4 ≤ 500	6.4% (n=457)	1.8% (n=453)	1.6% (n=428)	6.4% (n=457)	1.1% (n=453)	0.2% (n=428)

Early Symptomatic HIV Disease Study (n=713)	Granulocytopenia (<750/mm³)		Anemia (Hgb<8 g/dL)	
	Zidovudine 1200 mg	Placebo	Zidovudine 1200 mg	Placebo
CD4>200	4%(n=361)	1%(n=352)	4%(n=361)	0%(n=352)

Advanced Symptomatic HIV Disease Study (n=281)	Granulocytopenia (<750/mm³)		Anemia (Hgb<7.5 g/dL)	
	Zidovudine 1500 mg*	Placebo	Zidovudine 1500 mg*	Placebo
CD4>200 CD4<200	10%(n=30) 47%(n=114)	3%(n=30) 10%(n=107)	3%(n=30) 29%(n=114)	0%(n=30) 5%(n=107)

*Three times the currently recommended dose in asymptomatic patients.

The anemia reported in patients receiving zidovudine appeared to be the result of impaired erythrocyte maturation as evidenced by increased macrocytosis (MCV) while on drug.

The HIV-infected individuals participating in these clinical trials often had baseline symptoms and signs of HIV disease and/or experienced adverse events at some time during study. It was often difficult to distinguish adverse events possibly associated with zidovudine administration from underlying signs of HIV disease or intercurrent illnesses. The following table summarizes clinical adverse events or symptoms which occurred in at least 5% of all patients with advanced HIV disease treated with zidovudine in the original placebo-controlled study. Of the items listed in the table, only severe headache, nausea, insomnia and myalgia were reported at a significantly greater rate in zidovudine recipients.

Percentage (%) of Patients with Clinical Events in the Advanced HIV Disease Study

Adverse Event	Zidovudine (n=144) %	Placebo (n=137) %	Adverse Event	Zidovudine (n=144) %	Placebo (n=137) %
BODY AS A WHOLE			MUSCULOSKELETAL		
Asthenia	19	18	Myalgia	8	2
Diaphoresis	5	4	NERVOUS		
Fever	16	12	Dizziness	6	4
Headache	42	37	Insomnia	5	1
Malaise	8	7	Paresthesia	6	3
GASTROINTESTINAL			Somnolence	8	9
Anorexia	11	8	RESPIRATORY		
Diarrhea	12	18	Dyspnea	5	3
Dyspepsia	5	4	SKIN		
GI Pain	20	19	Rash	17	15
Nausea	46	18	SPECIAL SENSES		
Vomiting	6	3	Taste Perversion	5	8

Clinical adverse events which occurred in less than 5% of all patients treated with zidovudine in the advanced HIV study are listed below. Since many of these adverse events were seen in placebo-treated patients as well as zidovudine recipients, their possible relationship to the drug is unknown.

Body as a whole: body odor, chills, edema of the lip, flu syndrome, hyperalgesia, back pain, chest pain, lymphadenopathy

Cardiovascular: vasodilation.

Gastrointestinal: constipation, dysphagia, edema of the tongue, eructation, flatulence, bleeding gums, rectal hemorrhage, mouth ulcer.

Musculoskeletal: arthralgia, muscle spasm, tremor, twitch.

Nervous: anxiety, confusion, depression, emotional lability, nervousness, syncope, loss of mental acuity, vertigo.

Respiratory: cough, epistaxis, pharyngitis, rhinitis, sinusitis, hoarseness.

Skin: acne, pruritus, urticaria.

Special senses: amblyopia, hearing loss, photophobia.

Urogenital: dysuria, polyuria, urinary frequency, urinary hesitancy

Subsequent to the initial trial, sensitization reactions, including anaphylaxis in one patient, have been reported in individuals receiving zidovudine therapy.

All unexpected events and expected events of a severe or life-threatening nature were monitored in the placebo-controlled studies in early HIV disease and asymptomatic HIV infection. Data concerning the occurrence of additional signs or symptoms were also collected. No distinction was made in reporting events between those possibly associated with the administration of the study medication and those due to the underlying disease. The following tables summarize all those events reported at a statistically significant greater incidence for zidovudine recipients in these studies:

Percentage (%) of Patients with Clinical Events in the Early Symptomatic HIV Disease Study

Adverse Event	Zidovudine (n=361) %	Placebo (n=352) %
BODY AS A WHOLE		
Asthenia	69	62
GASTROINTESTINAL		
Dyspepsia	6	1
Nausea	61	41
Vomiting	25	13

Percentage (%) of Patients with Clinical Events* in an Asymptomatic HIV Infection Study

Adverse Event	1500 mg Zidovudine*** (n=457) %	500 mg Zidovudine (n=453) %	Placebo (n=428) %
BODY AS A WHOLE			
Asthenia	10.1	8.6**	5.8
Headache	58.0**	62.5	52.6
Malaise	55.6	53.2	44.9
GASTROINTESTINAL			
Anorexia	19.3	20.1	10.5
Constipation	8.1	6.4**	3.5
Nausea	57.3	51.4	29.9
Vomiting	16.4	17.2	9.8
NERVOUS			
Dizziness	20.8	17.9**	15.2

*Reported in ≥5% of study population.
**Not statistically significant versus placebo.
***Three times the currently recommended dose in asymptomatic patients.

The following events have been reported in patients treated with zidovudine: seizures, myopathy, nail pigmentation, changes in liver function tests. They may also occur as part of the underlying disease process. As such, the relationship between these events and the use of zidovudine is uncertain.

U.S. Patent No. 4724232 (Use Patent)
Other Pats. Pending

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Research Triangle Park, NC 27709

Reference: **1.** Volberding PA, Lagakos SW, Koch MA, et al. Zidovudine in asymptomatic human immunodeficiency virus infection: a controlled trial in persons with fewer than 500 CD4-positive cells per cubic millimeter. *N Engl J Med.* 1990;322:941-949.

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New York State
Journal of Medicine

COMMENTARIES

The utility of cross-cultural health research

By the turn of the last century, the collection and analysis of vital statistics, largely an American adaptation of a British public health practice, was becoming a standard local public health activity.[1] Ninety or more years later, we still do not know "how many Hispanics die each year in this country, let alone their health status, use of services, or unmet health care needs."[2] At present the United States has the fifth largest Hispanic population in the world, exceeded only by Mexico, Spain, Colombia, and Argentina. Furthermore, the Hispanic population in the United States is undergoing tremendous growth due to its younger age distribution, higher fertility rate, and higher rate of immigration to this country. Despite the fact that the United States has the largest and most extensively funded health monitoring systems in the world, the state of our knowledge regarding the health needs of our second largest minority group would serve as a source of national embarrassment to even the least developed nations. Meeting the health needs of this very large and ever-growing population with our limited knowledge may well prove to be one of the most significant and difficult challenges to be faced by medicine and public health in the near future.

In the United States, the registration of vital statistics is a state function. The states and other registration areas routinely provide these data to the National Center for Health Statistics, an agency of the US Public Health Service, in order to facilitate the analysis of vital events on a national level. By law, each state is free to determine what data elements will be collected through its vital statistics registration systems (marriage, divorce, birth, and death records). The National Center for Health Statistics, as the national repository of vital statistics, can only make recommendations to the states and other registration areas regarding the types of information that should be collected through these records. For many years, states collected information by race on their birth and death certificates. However, it was not until 1978 that the National Center for Health Statistics recommended for the first time the addition of a Hispanic identifier on these certificates. The addition of a Hispanic identifier was needed because Hispanics are ethnic minorities and are racially diverse. Since the vast majority of Hispanics are classified as white, their records were being combined with white non-Hispanics, a group which is frequently culturally, genetically, and socioeconomically dissimilar.

In 1978, 19 registration areas, including New York State and New York City (an independent registration area), followed the recommendation of the National Center for Health Statistics and participated in the reporting of Hispanic origin deaths. These 18 states and New York City provided coverage for approximately 58% of the US Hispanic population. Unfortunately, not all persons responsible for completing the birth and death records in these areas completed the Hispanic origin question. Hispanic origin was unknown or not reported for approximately one fourth of the resident deaths of these reporting areas.[2] The large number of death certificates with an unanswered Hispanic origin question severely limited the analytic potential for investigating mortality among Hispanics.

Fortunately, more states are adding a Hispanic identifier on their birth and death certificates and reporting of this item has become more complete. States revise their birth and death certificates approximately every ten years. In an effort to assist the states in deciding which items to include in their certificates, the National Center for Health Statistics issues a Standard Certificate of Birth and a Standard Certificate of Death which includes the data elements of a minimally acceptable record. In the late 1980s, the National Committee on Vital and Health Statistics (a 15-member committee appointed by the US Secretary of Health and Human Services to advise the Department of Health and Human Services on health statistical policy issues) voted to add the Hispanic origin question on the Standard Certificates of Birth and Death. It is hoped, though deemed somewhat unlikely, that all states and registration areas will follow the National Center for Health Statistics' recommendations and add the Hispanic origin question to the vital statistics certificates they will use during the 1990s. Should this happen and should the medical personnel and funeral directors who complete the certificates understand the utility of the data and complete the Hispanic origin question accurately and completely, we will for the first time in this country be able to assess the mortality experience of Hispanic Americans. This would appear to be a timely issue given that Hispanics have resided in the United States for more than 400 years.

The article by Dr Polednak[3] in this issue of the *Journal* highlights some of the limitations as well as the potential utility of the vital statistics system for identifying the divergent health needs of our diverse populations. Over the years, the vital statistics system has demonstrated its utility as a relatively inexpensive yet effective means for monitoring the health status of our citizenry.

Cross-cultural health research, by investigating the similarities and differences in the prevalence of disease among ethnically and racially diverse populations, can help target those health needs in a more effective and efficient manner. Furthermore, such cross-cultural health research offers great potential for an understanding of the etiology of disease. At the present time, there are important differences between our ethnic/racial populations. These populations are different genetically, culturally, and socioeconomically. They oftentimes have different occupational histories, health habits, living conditions, and diets. As we find differences in disease prevalence rates, we can begin to ask which of our differences are responsible for our different experiences with disease.

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The present and future importance of mortality statistics

The report by Polednak[1] in this issue of the *Journal* concerning Hispanic mortality in a New York county has three very important messages. These are the unusual differentials in total mortality and cause of death in those of Puerto Rican origin; the potential usefulness of cause-of-death statistics from small geographic areas for etiologic research and public health programs; and, finally, the continued need to improve cause-of-death statistics through physician education in the state and country.

First, for 30 years there have been observations about decreased coronary mortality on the island of Puerto Rico compared with the general United States population.[2] These differences have not been explained by variation in certification practices between locations. More recently, comparisons, using comparable methods, have been made between an epidemiologic study in Puerto Rico and the Framingham Heart Study.[3] A persistent two-fold difference was found in cardiovascular mortality as well as incidence. However, the risk factors of elevated blood pressure and cholesterol level did predict coronary heart disease incidence in both populations. Also, Rosenwaike[4] has been particularly resourceful in using national mortality statistics in investigations of differences among Puerto Rican and other Hispanic subgroups.

In the report in this issue of the *Journal*, the author postulates that the Hispanic subgroup in Suffolk County, predominantly of Puerto Rican origin, could be at greater mortality risk than the general white population because of lower socioeconomic status, including lower levels of education and increased poverty. However, the study gave mixed results. In fact, total mortality in Hispanics was lower, principally from ischemic heart disease. This finding is consistent with previous reports.[3,4] Also, possibly with the exception of stomach cancer, total cancer mortality was lower, including specifically lung, breast, and colorectal cancer. On the downside, however, is the apparent increase in mortality from diabetes in Hispanic women. This was true both with the standardized and proportionate mortality ratios. Such an excess in Hispanics has been previously noted but is still unexplained.[5] Finally, even with relatively few deaths, the ominous finding of increased homicide mortality among Hispanic men cannot be overlooked. The author properly suggests that further specific hypothesis-driven investigations are necessary before making final conclusions.

Second, this report illustrates the potential usefulness of mortality data from a small geographic area to identify important epidemiologic and public health issues. Although a small number of deaths required the combination of data for five years and precluded any time trend analyses, study results such as these can be tremendously important for local jurisdictions in assessing their health needs. In the present case, the author was able to take advantage of special circumstances of a large concentration of persons born in Puerto Rico, who were of a slightly higher socioeconomic status than those in New York City or on the island. Also, local area death analyses might be used to alert practicing physicians to diseases causing large numbers of deaths in their community, such as cardiovascular disease and cirrhosis in Harlem.[6] Mortality data are among the most uniformly available health statistics and can be used for public health purposes or for addressing specific hypotheses. There are many applications in health planning.[7] For example, as state and county health organizations consider the implementation of health promotion and disease prevention objectives for the year 2000, the ability to track potential effects of interventions with mortality statistics needs to be considered.

Third, although mortality data, as collected in this and other studies, are useful and valid measures of disease burden, continued diligence by physicians in New York and elsewhere in the United States in striving to improve the quality of cause of death statistics is necessary.[8] The National Center for Health Statistics, part of the Centers for Disease Control, and the National Committee on Vital and Health Statistics, advisory to the Secretary of Health and Human Services, concluded in a recent workshop that enhanced efforts in physician education are an important aspect of this process. Such programs would need to occur with the active assistance of state vital statistics registrars, professional medical organizations, academic centers, and other groups. In these programs, for example, physicians should be reminded that mortality statistics are based on "underlying" cause, or the bottom line disease that initiated the chain of events leading to death. This cause is of most etiologic importance and potentially could lead to possible public health interventions. The "underlying" concept differs somewhat from the "principal" or primary cause as required on a hospital discharge face sheet to indicate the main reason for admission.

In addition, the physician needs to be encouraged to enter on the certificate the various conditions or disease manifestations that connect the underlying cause and the immediate cause of death. However, the immediate cause is more than a final common event, such as respiratory arrest or asystole, which adds no etiologic information. It should represent as specific an entity as possible. Also, the physician should take time to add other significant conditions contributing to death but not directly included in the final chain of events. This is particularly important because such additional information allows the preparation of analyses of secondary causes of death. For example, diabetes is often not listed as the underlying cause of death, but multiple cause tabulations highlight the overall importance of diabetes.[9] In the present report, multiple cause tabulations, if they had been available, might have added to or perhaps decreased the adverse differential in diabetes deaths in Hispanic women when compared to other whites.

Finally, physicians should be cognizant of the need to report the cause of death as accurately as possible. Ideally, this should be done without regard to the potential sensitivity of family, friends and other interested parties to the correct diagnosis, such as acquired immunodeficiency syndrome or suicide. Of course, in real life situations there may be blatant and/or subtle influences affecting such decisions. In order to minimize such pressures, many states have confidentiality laws that protect the death certificate from public disclosure of cause of death. However, other states have completely open records. The effect of these two extremes on the accuracy of death certificate data has not been fully documented. In the long run the public's health and that of future patients will be benefited most by more accurate and complete reporting of cause of death on death certificates. In order to assist physicians in this process, a *Physician's Handbook on Medical Certification of Death* or comparable document can be obtained from the State Vital Statistics Office or from the National Center for Health Statistics.

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Mortality in the Hispanic population of Suffolk County, New York

ANTHONY P. POLEDNAK, PHD

ABSTRACT. In the 1980 census, the "Spanish-origin" or "Hispanic" population of Suffolk County, New York (predominantly of Puerto Rican origin), had a lower median family income and a higher prevalence of poverty than the "all white" population of that county. Mortality among Hispanics, however, was not greater than expected on the basis of age-specific death rates for "all whites" in 1979–1983. The standardized mortality ratios for ischemic heart disease and all neoplasms were less than 1.00 for each sex, but were significantly elevated for diabetes mellitus in females and for both infectious diseases and homicides in males. Comparisons were made with mortality patterns in other American Hispanic populations predominantly of Puerto Rican origin, but with lower income levels (including those from New York City), and with data from Puerto Rico. The limitations of routinely reported mortality data are discussed, along with the value of such data in suggesting leads for more in-depth studies.

(NY State J Med 1990; 90:442–446)

The label "Hispanic" is commonly used despite lack of consensus on terminology.[1,2] Hispanic groups, even in the same geographic area, are heterogeneous with regard to socioeconomic status, sociocultural factors,[3] and genetic background. This heterogeneity may have implications for the prevalence of various diseases.

The 1980 US census reports contain responses to two separate items concerning "Spanish origin" and "race," so that "all whites" includes whites of "Spanish origin." Persons of Spanish origin living in Suffolk County (Long Island, NY) had lower median family incomes and higher proportions of the population with incomes near or below the poverty level in comparison with "all whites" in the 1980 census (Table I).[4,5] Median family income, however, was considerably higher for Hispanics in Suffolk County ($20,087) than for Hispanics in the entire United States ($14,711) or in the New York City standard metropolitan statistical area ($10,347). As in New York City, the predominant subgroup of Hispanics in Suffolk County is Puerto Rican, representing 61% of the total Spanish-origin population. The remainder includes persons of Mexican (3%), Cuban (4%), and "other Spanish" origin (32%).[4,5] Puerto Ricans, the second largest Hispanic group in the US, migrated from the Commonwealth of Puerto Rico primarily after 1945.

This report repesents data on mortality in Hispanics in comparison with "all whites" in Suffolk County for 1979–1983. Mortality data for the entire Hispanic population in the United States are limited to numbers of deaths in 15 states, with no estimated mortality rates.[6] Comparisons are also made with mortality data reported for other Hispanic populations of Puerto Rican origin, in the United States and for residents of Puerto Rico itself.

METHODS

Computerized individual records (without personal identifiers) of all deaths in Suffolk County during 1979–1983 were used to identify deaths among residents of the county. Underlying cause of death had been coded to the ninth revision of the International Classification of Diseases (ICD).[7] A specific question regarding "Spanish origin," with categories including "Puerto Rican," "Mexican," "Cuban," and "other," has been used on Suffolk County death certificates since 1979. Population data for all persons of Spanish origin and "all whites" in Suffolk County for 1980 were used for the period 1979–1983.

Average annual age, sex, and cause-specific death rates (per 100,000 people) were calculated for "all whites" in Suffolk County. Population estimates for the Spanish-origin population from the 1980 census were multiplied by the death rates in "all whites" to obtain expected numbers of deaths in Hispanics. Age groups used were <5, 5–19, 20–34, 35–44, 45–54, 55–64, 65–74 and 75+ years. The total number of deaths observed in Hispanics during 1979–1983 was divided by the expected number of deaths, to obtain a standardized mortality ratio (SMR).

Only 2.8% of Spanish-origin decedents in 1979–1983 were black, and 0.4% were listed as "other" race on death certificates, while in the 1980 census, 3.7% of Spanish-origin persons in Suffolk County were classified as black and 21% as "other" race. One possible explanation for this is that physicians who complete death certificates may use the "other" race category less frequently than persons who describe their own race in censuses, and/or Spanish origin may be under-reported on death certificates (especially among blacks and "other races"). In view of the latter possibility, SMRs were also calculated (but not shown) by using only the white Spanish-origin population to obtain expected numbers of deaths.

Proportional mortality ratios (PMRs) for the Puerto Rican born were compared with those for "non-Hispanic whites" (ie, "all white" decedents excluding those of Spanish origin) as reported on the death certificate. For comparison purposes, PMRs were calculated from mortality data for Puerto Rico in 1983.[8]

A chi-square test was used for analysis of PMRs for individual age strata. For analysis of SMRs, 95% confidence limits were based on the Poisson distribution.[9]

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TABLE I. Selected Characteristics of All "Spanish-Origin" Persons and "All Whites" in Suffolk County from 1980 Census

Group	Males	Females	Total	Family Income Mean ($)	Family Income Median ($)	Education ≥12 yrs*	Poverty Level[†] <75%	Poverty Level[†] <150%
All Spanish-origin	28,883 (49%)	29,856 (51%)	58,689 (100%)	22,185	20,087	59.4%	7.4%	22.3%
All whites[‡]	577,395 (49%)	607,714 (51%)	1,185,109 (100%)	27,254	24,484	74.5%	3.5%	11.3%

* Percent with 12 or more years of education among persons 25 years of age or older (US Bureau of the Census, 1982, 1983).

[†] Proportion of the population at <75% and <150% of the poverty level.

[‡] In the 1980 census, "Spanish-origin" persons may be of any "race," so that "all whites" includes Spanish-origin persons who classified themselves as whites.

RESULTS

SMRs for all causes were less than 1.00 for both male and female Hispanics (Table II), using the entire Hispanic population for the calculation of expected numbers of deaths. Using only the Hispanic white population to calculate expected numbers of deaths (not shown), SMRs were still not greater than 1.00.

SMRs for Hispanics were significantly lower than 1.00 for all neoplasms and for ischemic heart disease (Table II). Using only the Hispanic white population to calculate expected numbers of deaths (not shown), SMRs were still significantly lower for all neoplasms in females and for ischemic heart disease in males. Significantly elevated SMRs were evident, however, for diabetes mellitus in females and for both infectious diseases and homicides in males (Table II).

Numbers of deaths from specific cancers in Hispanics were small. SMRs for stomach cancer were not significantly elevated, although confidence limits were wide (Table III). SMRs significantly lower than 1.00 were evident for colorectal cancers in females, lung cancer in both sexes, and female breast cancer (Table III).

Some 92% of all decedents aged 45 years and older at the time of death and classified as "Puerto Rican" were born in Puerto Rico. The PMR for all neoplasms was significantly lower in Puerto-Rican born vs "non-Hispanic white" females aged 65 years or older in Suffolk County, and similar to the PMR for Puerto Rico (Table IV). PMRs for diabetes mellitus were higher for Puerto Rican born than for "non-Hispanic white" female decedents in Suffolk County, and were as high as (or higher than) those in females in Puerto Rico (Table IV). In contrast, PMRs for diabetes did not differ between Puerto Rican born and "non-Hispanic white" males in Suffolk County, and were lower than those for males in Puerto Rico. Puerto Rican born male decedents 45-64 years old had a significantly lower PMR for ischemic heart disease than "non-Hispanic white" males in Suffolk County. For females 45-64 years old, and for both sexes 65 years of age or older, PMRs for ischemic heart disease were similar by sex-ethnic group within Suffolk County, and consistently higher than those for Puerto Rico (Table IV).

DISCUSSION

Despite the lower median income level of Hispanics vs "all whites" in Suffolk County (Table I), SMRs for all

TABLE II. Standardized Mortality Ratios (SMRs) for All Causes and for Selected Causes of Death Among All Hispanics in Suffolk County, 1979-1983

Cause of Death (ICD Codes)	Sex	Observed No.	Expected No.*	SMR[†] (and 95% Confidence Interval)
All causes	Male	513	642.2	080 (0.73, 0.87)[‡]
	Female	392	523.9	0.75 (0.68, 0.83)[‡]
Infectious diseases (001-139)	Male	13	5.3	2.43 (1.26, 4.16)[‡]
	Female	5	4.5	1.12 (0.36, 2.61)
All neoplasms (140-239)	Male	105	152.5	0.69 (0.57, 0.84)[‡]
	Female	91	145.8	0.62 (0.50, 0.77)[‡]
Diabetes mellitus (250)	Male	8	10.2	0.79 (0.34, 1.56)
	Female	19	11.0	1.73 (1.04, 2.70)[‡]
Ischemic heart disease (410-414)	Male	130	197.2	0.66 (0.55, 0.79)[‡]
	Female	106	150.6	0.70 (0.40, 0.85)[‡]
Pulmonary circulation (415-429)	Male	26	28.7	0.90 (0.59, 1.32)
	Female	25	25.9	0.97 (0.63, 1.44)
Cerebrovascular diseases (430-438)	Male	27	31.3	0.86 (0.57, 1.26)
	Female	31	43.5	0.71 (0.48, 1.01)
Respiratory system (460-519)	Male	26	31.3	0.83 (0.54, 1.22)
	Female	15	22.1	0.68 (0.38, 1.12)
Chronic liver disease (571)	Male	12	14.1	0.85 (0.44, 1.48)
	Female	7	9.8	0.71 (0.29, 1.46)
Perinatal conditions (760-779)[§]	Male	12	12.6	0.95 (0.49, 1.66)
	Female	10	9.0	1.11 (0.53, 2.04)
Accidents (E800-929)	Male	54	61.7	0.88 (0.66, 1.16)
	Female	27	23.2	1.16 (0.76, 1.69)
Suicide (E950-959)	Male	11	15.6	0.71 (0.35, 1.27)
	Female	3	6.0	0.50 (0.10, 1.46)
Homicide (E960-969)	Male	15	6.0	2.48 (1.39, 4.09)[‡]
	Female	3	2.8	1.08 (0.22, 3.15)

* Expected number of deaths, on the basis of age- and sex-specific death rates for "all whites" in Suffolk County.

[†] Standardized mortality ratio, or ratio of observed to expected number of deaths.

[‡] $p < 0.05$ for SMR.

[§] Denominators for rates were population estimates for age <1 year.

TABLE III. Standardized Mortality Ratios for Selected Cancer Sites Among All Hispanics in Suffolk County, 1979–1983

Cancer Site (ICD codes)	Sex	Observed No.	Expected No.	SMR (and 95% Confidence Limits)
Oral cavity, pharynx (140–149)	Male	2	3.3	0.59 (0.07, 2.13)
	Female	0	2.3	0.00 (. . . , 3.81)
Stomach (151)	Male	7	5.9	1.18 (0.47, 2.43)
	Female	4	3.3	1.20 (0.33, 3.07)
Colorectal (153–154)	Male	14	19.8	0.71 (0.39, 1.19)
	Female	10	18.5	0.54 (0.26, 0.99)*
Pancreas (157)	Male	6	7.5	0.80 (0.29, 1.74)
	Female	4	7.1	0.56 (0.15, 1.43)
Lung (162)	Male	27	48.6	0.56 (0.37, 0.82)*
	Female	7	24.8	0.28 (0.11, 0.58)*
Breast (174)	Female	17	29.8	0.57 (0.33, 0.91)*
Cervix (180)	Female	2	2.9	0.70 (0.08, 2.53)
Prostate (185)	Male	10	11.0	0.91 (0.44, 1.67)

* $p < 0.05$.

causes were not elevated in Hispanics. This finding was due in part to lower than expected numbers of deaths from certain major chronic diseases, notably neoplasms and ischemic heart disease, among Hispanics (Table II).

As noted earlier, the majority of Hispanics in Suffolk County are of Puerto Rican origin, and almost all older decedents of Puerto Rican origin were born in Puerto Rico. For the entire United States, age-adjusted death rates for age 5 years or older were slightly lower for Puerto Rican born persons than for “all whites.”[10] For the entire United States, the ratios of age-adjusted cancer death rates for Puerto Rican born vs “all whites” were low for both males and females (0.78 for each sex).[10]

On the basis of small numbers, mortality from lung cancer, female breast cancer, and colorectal cancer in females was significantly lower in Hispanics vs “all whites” in Suffolk County (Table III). These findings are generally consistent with those from studies of Puerto Rican immigrants in New York City[11] and Illinois.[12] Although SMRs were not elevated for stomach and cervical cancer in the present study (Table III), in contrast to Puerto Ricans in New York City and Illinois, numbers of deaths were small. Further data are needed for these cancers in the Hispanic population of Suffolk County; the incidence of these cancers is inversely associated with socioeconomic status.

TABLE IV. Proportional Mortality Ratios (%) for Selected Chronic Diseases in Suffolk County and in Puerto Rico,[*] Ages 45–64 and ≥65 Years

	Suffolk County, New York					
	Non-Hispanic Whites		Hispanics, Born in Puerto Rico		Puerto Rico	
Age (yr)	Male	Female	Male	Female	Male	Female
			All neoplasms			
45–64	31.5	47.1	23.2	42.0	19.1[†]	27.9
≥65	26.6	20.6	20.4	13.3[‡]	18.6	13.4
			Diabetes mellitus			
45–64	1.9	1.7	2.4	6.0[‡]	4.9	7.4
≥65	2.0	2.4	2.9	10.4[‡]	4.0	7.1
			Ischemic heart disease			
45–64	36.3	18.1	23.2[‡]	20.0	17.2[§]	12.7
≥65	38.9	40.4	39.8	35.6	18.2	18.3
			Total deaths (no.)			
45–64	5,436	3,443	82	50	2,754	1,547
≥65	13,286	15,526	103	135	7,290	6,314

[*] For 1983 (from data in *1985 Demographic Yearbook*[8]).

[†] ICD codes 140–208 (malignant neoplasms) only for Puerto Rico, vs both ICD 140–208 and 209–239 (benign and unspecified neoplasms) for Suffolk County; few deaths are due to the latter subcategory.

[‡] $p < 0.05$ for chi-square test comparing the Hispanic subgroup with non-Hispanic whites in Suffolk County. Data for Puerto Rico are presented for comparison, but statistical tests were considered inappropriate (see text).

[§] ICD codes 410–414 for both Puerto Rico and Suffolk County.

The PMR for all neoplasms at age 65 years or older in Puerto Rican born females in Suffolk County was apparently similar to that for females in Puerto Rico (in 1983) (Table IV). Because higher death rates from other causes (eg, infections) in Puerto Rico could make PMRs from chronic diseases appear low, comparisons of PMRs between Suffolk County and Puerto Rico (Table IV) must be made with caution.

SMRs for ischemic heart disease among Hispanics in Suffolk County were also less than 1.00 (Table II). Among decedents 45–64 years old, PMRs were significantly lower in Puerto Rican born vs “non-Hispanic-white” males (but not females) in Suffolk County. For the entire United States, age-adjusted death rates for “diseases of the heart” were lower for Puerto Rican born than for “all white” males (a ratio of 0.86) but not for females (a ratio of 1.09).[10] In New York City in 1969–1971, age-specific death rates from heart disease were lower in Puerto Rican born vs “all others” for males aged 25 years or older but not for females younger than 75 years old.[13] One explanation for this sex difference is suggested by the higher PMRs for diabetes mellitus in Puerto Rican born females (Table IV), because diabetes is a risk factor for early onset coronary atherosclerosis[14] and death from ischemic heart disease.[15]

Data from Puerto Rico have indicated lower death rates from ischemic heart disease in comparison with the United States.[16,17] Possible increases in mortality in Puerto Rican born Hispanics in Suffolk County, in comparison with the present population of Puerto Rico, were suggested by the higher PMRs at older ages in comparison with PMRs in Puerto Rico (Table IV).

In contrast to the findings for neoplasms and ischemic heart disease, SMRs for diabetes mellitus were higher in Hispanic vs “all white” females in Suffolk County. The PMR for diabetes was also higher among Puerto Rican born vs non-Hispanic white females in Suffolk County (Table IV). For the entire United States, age-adjusted mortality rates for diabetes also were higher for Puerto Rican born vs "all white" women,[10] and the present finding of a similar pattern for females in a higher income Hispanic population is noteworthy.

Among the Puerto Rican born Hispanics in New York City in 1969–1971, only females had higher death rates from diabetes than the non-Puerto Rican born Hispanics[11]; this is in agreement with the present findings on SMRs in all Hispanics (Table II). In contrast, death rates for diabetes in the United States were also higher in Puerto Rican born vs "all white" males.[10] This suggests regional differences in mortality patterns for Puerto Rican born groups in the United States, although differences in death certification practices cannot be ruled out. Only the underlying cause of death was available for the present analysis of diabetes mortality. Accuracy of death certificate data for diabetes as the underlying cause of death may vary by racial/ethnic group, as has been suggested for mortality data for ischemic heart disease in American blacks (especially young adult females).[18] Community based studies, including information on clinical history, are needed to evaluate potential biases in reporting of diabetes mellitus as the underlying cause of death by ethnic group and sex.

Selection of Puerto Rican migrants for better health,[19] among males only, could explain the apparent mortality pattern for diabetes mellitus in Suffolk County vs. Puerto Rico (Table IV). Another possible explanation involves sex differences in degree of acculturation levels and/or acculturation effects (perhaps involving obesity). Among Mexican Americans in Texas, degree of acculturation was more important than socioeconomic status in predicting obesity and diabetes.[20] Length of residence in Suffolk County, and degree of acculturation, were unknown in the present study. The prevalence of obesity by socioeconomic status and degree of acculturation should be determined in samples of Puerto Rican born males and females in Suffolk County.

The significantly elevated SMRs for infectious diseases and homicides in Hispanic males in Suffolk County (Table II) may be related to the substantial ethnic differences in income and poverty level within the county (Table I)—this also requires further examination. For the entire US, age-adjusted rates for homicides were about 7.5 times higher in Puerto Rican born vs "all white" males in 1979–1981,[10] in comparison with the SMR of 3.3 for Hispanics in Suffolk County. Also, the SMR for chronic liver disease was not elevated for Hispanics in Suffolk County (Table II), while Puerto Rican born males in the United States had an age-adjusted death rate about 3.5 times higher than "all whites;" death rates for infectious diseases in the United States were not presented.[10] These apparent differences between Hispanics in Suffolk County, the majority of whom are of Puerto Rican origin (and Puerto Rican born, for older age groups), and the entire population of Puerto Rican born residents of the United States could be related to the higher income levels of the Suffolk County subgroup.

This study illustrates both the limitations of using death records for the analysis of ethnic differences in mortality, and the value of such analyses in generating hypotheses that can serve as the basis of further studies. Descriptive studies could be improved by the use of Spanish surnames for delineation of ethnic groups,[21] and by the availability of better population estimates for the Hispanic population. Underestimation of mortality rates for ischemic heart disease and neoplasms in Hispanics in Suffolk County may have occurred in this study. In contrast, significantly increased mortality from diabetes mellitus has been shown for Hispanic females in Suffolk County. While the death rates and PMRs for diabetes mellitus in Hispanics could be even higher than those reported here, studies are needed on the quality of data on diabetes reported on death certificates.

If these ethnic differences are confirmed, explanations should be explored through studies of life style factors that might protect against ischemic heart disease and certain cancers, but also increase the risk of diabetes mellitus in Hispanics in Suffolk County. Identification of such factors could lead to public health programs aimed at maintaining salubrious habits such as nonsmoking, and perhaps supporting aspects of the diet in certain ethnic groups, while reducing the prevalence of other factors such as obesity. Finally, results of such studies could be useful in encouraging the modification of certain life styles or specific habits among other ethnic groups.

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Small bowel injuries following blunt abdominal trauma

Early recognition and management

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ABSTRACT. **We reviewed the cases of 15 patients with intestinal perforation following blunt abdominal trauma, which occurred between 1971 and 1988. Twelve patients were treated at The Mary Imogene Bassett Hospital (Cooperstown, NY); three other patients were treated at surrounding area hospitals. These injuries included 11 motor vehicle accidents, three low-velocity impact injuries, and one blast injury. There were four duodenal, ten jejunal, and two ileal injuries. Five patients who were operated on within 12 hours had classic signs of peritonitis or gross blood on diagnostic peritoneal lavage. Four patients were operated on after 12 hours, and six after 24 hours; physical signs were subtle in this group. Pain was a universal finding but was usually moderate; nausea and vomiting were frequent early findings in the ten patients with late recognition. An altered sensorium due to intoxication or head injury was present in three of ten patients operated on after 12 hours.**

Laboratory determinations, including a white blood cell count and amylase analysis, as well as abdominal radiographs, were often not helpful. Diagnostic peritoneal lavage is an important test, but when it is performed within four hours post-injury, it may yield false negative findings in up to 50% of patients and may need to be repeated. Computer tomographic scanning should employ oral and intravenous contrast to increase accuracy; perforation of a hollow viscus may not be immediately recognized.

Four of the five patients operated on within 12 hours had an uncomplicated course. Complications occurred in all six patients operated on beyond 24 hours and included intra-abdominal abscesses in five, and death for one patient. The mean hospital stay was nine days for patients operated on within 12 hours and 38 days for those operated on after 24 hours. Appreciation of the often subtle signs and symptoms of small bowel injuries and the limitations of current diagnostic methods is essential for the prompt recognition of these injuries. Thorough abdominal exploration, including complete mobilization of the duodenum, is necessary. Early surgical repair markedly reduces complications and length of hospital stay.

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Intestinal perforations following blunt abdominal trauma remain a challenging problem today. Many may be recognized early because of multiple associated injuries. Nevertheless, a significant number of patients may have subtle physical findings and an insidious onset of abdominal sepsis due to delay in treatment. According to Harris,[1] this may be due to delayed peritoneal reaction to succus entericus; an altered sensorium from drugs, alcohol, or head injury; and false-negative results due to diagnostic peritoneal lavage (DPL), abdominal radiographs, or computer tomographic scans. Experimental studies by Meuller et al,[2] Levinson et al,[3] and Maull and Reath[4] have shown that DPL may not become positive for six hours in cases of intestinal perforation. Dauterive et al[5] reported that early recognition and treatment of these injuries has been shown to improve outcome markedly, decrease complications, particularly of abdominal sepsis, and shorten hospital stays.

Gastrointestinal fistulas are described in detail by Edmunds.[6] "Terminal" duodenal fistulas have been distinguished from lateral duodenal fistulas where gastrointestinal continuity is maintained because of the greater severity of the latter.[7,8] Surgical repair techniques have been developed to prevent fistulas, or to convert lateral to terminal duodenal fistulas to control drainage and improve prognosis. These techniques include primary repair with or without resection, pyloric exclusion, duodenal diverticulization, and pancreaticoduodenectomy.

This study is a retrospective review of our recent experience with intestinal perforation following blunt abdominal trauma. Numerous studies have documented the complications following these injuries, the efficacy of various surgical procedures, and the importance of early treatment. The purpose of this report is to characterize those patients with delayed diagnosis and understand the factors responsible for delay in diagnosis despite modern diagnostic capabilities.

PATIENTS AND METHODS

The hospital records of 12 patients from The Mary Imogene Bassett Hospital (Cooperstown, NY) with intestinal injuries following blunt abdominal trauma from 1971 to 1988 were reviewed. Three other cases were offered for review by area surgeons with an interest in this problem. There were eight male and seven female patients ranging in age from nine to 77 years (mean, 34 years) (Fig 1).

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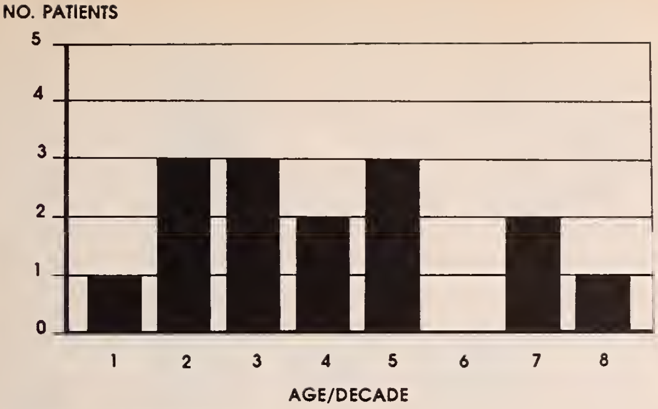

FIGURE 1. Age distribution.

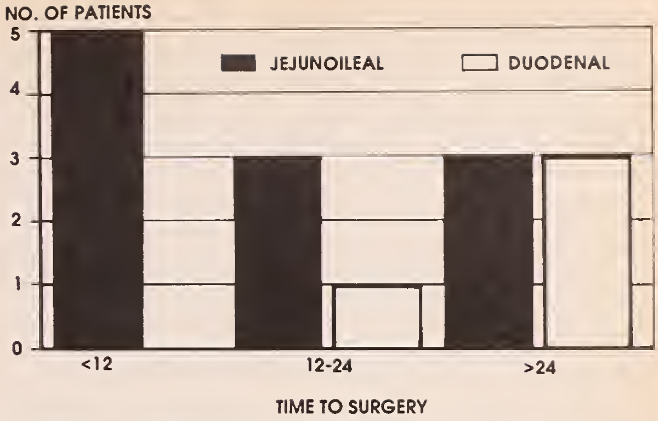

FIGURE 2. Distribution of injuries and time to surgery.

The mechanisms of injury included ten motor vehicle accidents (documented use of restraints in six, three of whom had a "seatbelt sign"); a blast from an exploding oil drum; a blow when one patient struck his abdomen on the steering wheel of his riding lawnmower when he hit a tree stump; two sports injuries, when one child fell on his bicycle handlebar and another youngster was struck in the abdomen playing football; and a tractor crush injury (Table I).

There were four duodenal injuries (three perforations, one hematoma), 11 jejunal injuries (ten perforations and one mesenteric avulsion), and three ileal injuries (two mesenteric avulsions and one hematoma) (Table II).

RESULTS

Clinical Course. Pain is a universal finding, occurring in all conscious patients; however, it is often only mild to moderate. Other symptoms referable to abdominal injury include nausea, vomiting, and fever. The classic triad of peritonitis, including tenderness, rigidity, and absent bowel sounds, was seen in only four of nine patients (45%) who were operated on within the first 24 hours, and was not present on admission in any of the patients in whom the diagnosis was delayed beyond 24 hours. Among the patients with delay in diagnosis beyond 24 hours, three of five did have the triad of pain, nausea, and vomiting (60%) at the time of admission. Fever is not a common presentation within the first 24 hours and was seen in only three of 15 patients (20%).

Five patients were operated on within 12 hours of injury. Delay in operation from 12 hours to 24 hours occurred in four patients, two of whom were intoxicated (50%). Delay in diagnosis beyond 24 hours occurred in six patients, one of whom was unresponsive, with a closed head injury. An altered sensorium therefore complicated the diagnosis in three of ten patients (33%) with a delayed operation, none of whom were operated on within 12 hours (Fig 2, Table III).

TABLE I. Trauma by Mechanism

Mechanism	Number	%
Motor vehicle accident	10	67
Low velocity impact	3	20
Blast	1	6.5
Crush	1	6.5
Total	15	100

TABLE II. Anatomic Distribution of Injuries

Site	Number and Type of Injuries
Duodenum	4 (3 perforation, 1 hematoma)
Jejunum	11 (10 perforations, 1 mesenteric avulsion)
Ileum	3 (2 mesenteric avulsion, 1 hematoma)
Total	18 injuries in 15 patients

Diagnostic Studies. Twelve abdominal roentgenograms were performed in 11 patients and were abnormal in six (50%). These included three patients with pneumoperitoneum. Interestingly, in one patient, the first film was normal and the second film four hours later demonstrated free air. Loss of the right psoas shadow was seen in two patients.

Diagnostic peritoneal lavage was performed in four patients and was positive in two (50%). One patient had return of gross blood, and lavage performed on the third hospital day in another patient was positive for blood and bacteria. There were two false-negative results in this group.

Six abdominal computed tomographic (CT) scans were performed in four patients and were abnormal in five (83%), two in retrospect. Two CT scans were performed on the second hospital day and were not helpful in establishing the diagnosis. Of the later CT scans, four out of four were abnormal, with three showing abscesses, and a large collection of peritoneal fluid in a fourth.

Upper gastrointestinal series were performed in three patients and were all abnormal, but none demonstrated leaks. There were duodenal hematoma in two, and jejunal mucosal irregularity in a third (Table IV).

Laboratory tests were not useful in the prediction of injury. The mean white blood cell count on admission was not significantly different in three groups based on time from injury to surgery, nor was there any relation between the white blood cell count on admission and subsequent complications. Amylase was elevated in only one of 15 patients on admission.

There was one death in the group (6%), following a tractor crush injury. This patient had a 4-cm duodenal tear and retroperitoneal

TABLE III. Relationship of Symptoms Present at Time of Admission to Time of Surgical Exploration

	Hours					
	<12		Δ12–24		>24	
Symptoms	No.	(%)	No.	(%)	No.	(%)
Pain	5/5	(100%)	4/4	(100%)	5/5	(100%)
Nausea, vomiting	1/5	(20%)	3/4	(75%)	3/5	(60%)
"Peritonitis"	3/5	(60%)	1/4	(25%)		
Fever			1/4	(25%)	2/5	(40%)

TABLE IV. Diagnostic Studies Performed

Studies	Number	%
AXR (abdominal plain film)	6/12 abnormal	(50%)
DPL (diagnostic peritoneal lavage)	2/4 positive	(50%)
CT (computed tomography)		
abdomen	4/4 abnormal	(100%)
UGI	3/3 abnormal	(100%)
abdominal tap, culdocentesis	1/3 positive	(33%)

abscess. Multiple associated injuries including rib fractures, and cerebral, myocardial, and pulmonary contusions were present. This patient underwent repair of his duodenal perforation, but expired on the third hospital day of sepsis and respiratory failure. Blood and peritoneal cultures subsequently grew *Clostridium perfringens*.

Delay in surgery had a marked effect on length of hospital stay and the incidence of complications. An uncomplicated postoperative course was experienced by seven of nine patients (77%) who were operated on within 24 hours of injury. The patient with a blast injury that resulted in ileal infarction from mesenteric avulsion developed four complications manifested by pneumonia, pulmonary embolus, arrhythmia, and wound dehiscence. A second patient, who suffered a low-velocity impact from a bicycle handlebar, developed a small bowel obstruction several months later that required enterolysis.

Peritoneal cultures were negative in three of five patients operated on within 12 hours. In two patients, positive cultures grew *Streptococcus*, *Klebsiella*, *Corynebacteria*, and *Candida* species. Only one of six patients operated on after 24 hours had a negative peritoneal culture. The remainder grew a mixture of gram-negative and gram-positive microbes, anaerobes, and yeast. Five of six patients (83%) operated on after 24 hours developed intra-abdominal abscesses. One death occurred in this group. Hospital stay was considerably prolonged in patients operated on after more than 24 hours. The five patients operated on within 12 hours had a mean stay of nine days. Those operated on between 12 and 24 hours had a mean stay of 12 days. Surviving patients operated on beyond 24 hours had a mean hospital stay of 38 days (Figs 3, 4).

Reparative techniques are listed in Table V. Three patients with duodenal perforation underwent pyloric exclusion and gastrojejunostomy; one duodenal fistula developed and closed with conservative management. Nine jejunoileal injuries resulted in 11 perforations and four segments of necrotic bowel due to mesenteric vascular injuries. All were treated with primary closure or simple resection and primary anastomosis. One patient with jejunal mucosal irregularity on an upper gastrointestinal series had an abdominal abscess that was presumed due to jejunal perforation; this was successfully managed by percutaneous drainage. There were no leaks following jejunoileal repairs, and no patients required a second operation because of missed injuries.

DISCUSSION

Intestinal injuries following blunt abdominal trauma continue to be a challenging problem for surgeons. Mortality is reported to be from 10% to 30% and is related to associated injuries.[3,6,9] Of major importance in improving outcome in these patients is early diagnosis and prompt surgical intervention to avoid serious infectious complications. Current diagnostic methods are a tremendous advantage to the trauma surgeon. Recognition of their limitations, a high index of suspicion, careful and repeated physical examination, and prompt surgical intervention are essential. Delayed diagnosis increases morbidity, mortality, and length of hospital stay. This study was undertaken to identify those characteristics that might expedite diagnosis and treatment in the future.

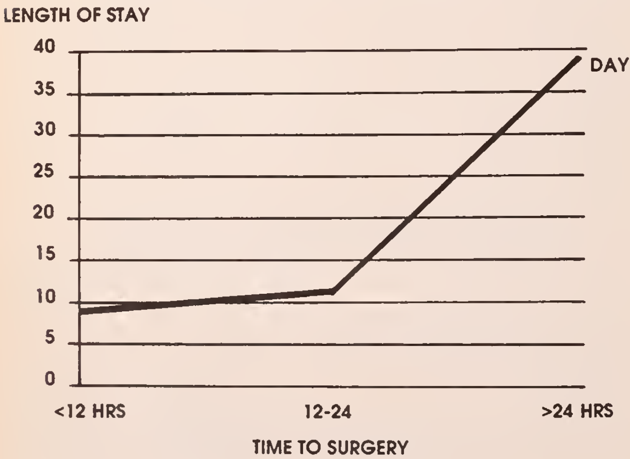

FIGURE 3. Hospital stay versus time to surgery.

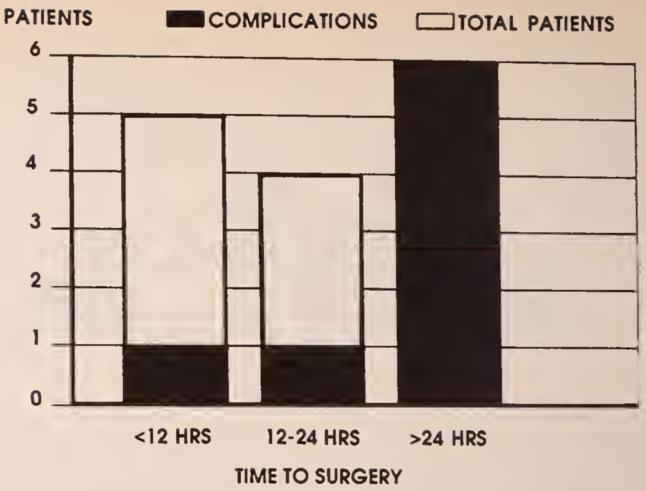

FIGURE 4. Complications.

Major intestinal injuries are reported to occur in 5% to 15% of cases of blunt abdominal trauma.[3] Motor vehicle accidents are responsible for most adult injuries. Cobb et al[10] found child abuse to be responsible for 8 of 12 cases of intestinal perforation in their pediatric population. The injuries included transmural perforation, mesenteric laceration, and bowel wall contusion. The mechanisms for these injuries include bursting of fluid-filled small bowel loops from sudden increase in intraluminal pressure from an extrinsic blow, compression of intestinal loops against the vertebral column, and tangential tears at fixed points or adhesions. Frey[11] has also described delayed diagnosis when a crushed segment that is initially intact becomes necrotic and perforates.

Intestinal perforation may be found at exploration for associated intra-abdominal injuries. Harris[1] pointed out that the neutral pH, low bacterial counts, and enzymatic activity of jejunal contents may be responsible for the insidious development of peritonitis following perforation. Absence of small bowel gas, short interval from time of injury to radiography, small size of perforations, and omentum overlying jejunum may mask development of free air within the peritoneum. Schenk et al[12] found abdominal pain to be the only universal complaint in 13 patients with jejunal perforation following blunt abdominal trauma. This is consistent with our findings. In addition, otherwise unexplained nausea and vomiting appeared to

TABLE V. Methods of Treatment

Treatment	Number	%
Debridement and primary repair	7	47
Pyloric exclusion, gastrojejunostomy and feeding jejunostomy	3	20
Resection and primary anastomosis	3	20
Drainage of abscess	1	6.5
Resolution of intramural hematoma (observation)	1	6.5
Total	15	100

be important findings in our group.

Flint et al[9] and Cobb et al[10] identified several of the difficulties in the diagnosis and management of these patients. Laboratory tests, including amylase level, white blood cell count, abdominal radiographs, and contrast duodenography, were often not helpful in identifying these injuries. They found the presence of abdominal tenderness and diagnostic peritoneal lavage useful in early recognition.

Flint et al[9] emphasized the need for wide mobilization of the duodenum in order to recognize all injuries. Root et al,[13] in 1965, described the technique of diagnostic peritoneal lavage, which has now become standard in trauma management. This technique is proven to be very sensitive in detecting free blood within the peritoneal cavity. False negatives, up to 50%,[5] following intestinal perforation have been reported. Experimental studies[2] have shown a low yield for diagnostic peritoneal lavage within four hours of injury, although 94% became positive by six hours. Peitzman et al[14] and Federle et al[15] have shown CT scanning to be a useful adjunct to DPL, particularly in the recognition of solid organ and retroperitoneal injuries. Kaufman et al[16] emphasized combining oral and intravenous contrast to maximize efficacy, but effectiveness in identifying loss of intestinal continuity or leakage of contrast from the gastrointestinal tract was limited in our study.

Celiotomy should be considered an extension of the surgeon's diagnostic options when the previous tests leave sufficient concern about the extent of unrecognized injuries. After control of hemorrhage, a thorough exploration must be performed. Attention must be directed to identifying any signs suggestive of duodenal injuries,[5] including periduodenal hematomas, bile staining, or air bubbles in the region of the bilary tree or duodenum. A careful exploration[12,17] must include wide mobilization of the duodenum employing the methods of Kocher and Cattell,[18] and taking down the ligament of Treitz.

Perl,[19] in 1935, distinguished "terminal duodenal fistula" from the more serious "lateral fistula," since the latter remain in continuity with gastrointestinal flow. Bartlett and Lowell,[20] in 1938, found a mortality of 14% with terminal fistulas, while lateral duodenal fistulas had a 40% mortality at that time. The complexity of these injuries and their complications have been recognized for years. Several surgical techniques have now evolved to deal with these problems.

Techniques of duodenal diverticulization and pyloric exclusion are aimed at converting lateral to terminal fistulas. Berne et al[21] described the technique of duodenal diverticulization, which included antrectomy, duodenostomy, vagotomy, gastrojejunostomy, feeding jejunostomy, and drainage. Selective biliary drainage was employed when necessary. Vaughan et al[8] popularized the role of pyloric exclusion in 1977 as a means of avoiding antrectomy; vagotomy was not employed. Of 75 patients treated with pyloric exclusion, Vaughan et al dealt with the development of three fistulas and three marginal ulcers.[8] Pancreaticoduodenectomy is infrequently necessary, and most series report mortalities of 30% often related to associated injuries. Oreskovich et al[22] recently reported a series of ten patients with no deaths following pancreaticoduodenectomy for trauma.

Intestinal perforation following blunt abdominal trauma remains a challenge for the surgeon. Numerous authors[1,5,23] have reported morbidity and mortality following these injuries, and sound methods have been developed for management. The development of diagnostic peritoneal lavage, CT scanning, newer antibiotics, and surgical techniques have all played an important role in improving outcome for these patients. Nevertheless, delay in recognition, and treatment of these injuries after 24 hours, have been associated with marked increase in both morbidity and mortality.[3,4,24] It is important to realize that the signs and symptoms of these injuries are often subtle. Pain is a constant finding, often associated with nausea and vomiting; fever may not be present. Diagnostic peritoneal lavage may be misleading within the first six hours of hollow viscus injury, and repeat lavage may be necesary. CT scanning should employ both oral and intravenous contrast to improve yield, and CT scanning may not specifically recognize bowel injuries.

It is essential to maintain a high index of suspicion for these injuries and recognize the limitations of current diagnostic techniques. It is only after careful, complete, serial examination, and, on occasion, "diagnostic celiotomy," that prompt therapy is instituted in an effort to reduce morbidity and expeditiously treat the injury to the small bowel.

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A history of the New York Society of Tropical Medicine

PASCAL JAMES IMPERATO, MD

The New York Society of Tropical Medicine was organized in the spring of 1933 by a group of 17 men who shared a common interest in the specialty. They came from a diversity of professional backgrounds which included medical teaching, research, overseas mission work, industrial medicine, dermatology, and military medicine.[1] The heterogeneous nature of this original group has come to characterize the society's membership over the years. It has drawn to it clinicians, basic science researchers, public health specialists, and others whose pooled experiences, training, and skills have consistently given it strength. In addition, the society has functioned as a forum for the discussion of clinical, laboratory, and field problems in tropical medicine, thus enriching the knowledge of its individual members.

The necessary environment for such a society to function has been provided by the unique character of the greater New York City metropolitan region. This includes a cosmopolitan population; the steady flow of immigrants and travelers from tropical areas; several large research institutions and medical centers; philanthropic, industrial and religious organizations involved in work in the tropics; pharmaceutical firms with a focus on antiparasitic drugs and parasitic diagnostics; and the presence of professionals with a past and/or current involvement with tropical diseases. The society has historically drawn most of its membership from New York, New Jersey, Connecticut, and Pennsylvania, and presently includes on its rolls a few individuals from places as distant as California and Washington, DC. Diversity of professional background has long characterized the society's membership and been a source of strength and intellectual enrichment. However, the society has not attracted to it great numbers of molecular biologists, the most recent professional group to show an interest in parasites.

Prior to the founding of the society, national professional societies of tropical medicine had existed for several decades. The Royal Society of Tropical Medicine and Hygiene was founded in 1907.[2] In the United States, the American Society of Tropical Medicine was founded in 1903 and the National Malaria Society in 1916. In 1951, these two societies merged as the American Society of Tropical Medicine and Hygiene.[3] Local societies of tropical medicine have also existed in various parts of the country, the one in Washington, DC, being the oldest.

CONSTITUTION AND BY-LAWS

The society's constitution and by-laws were adopted at a meeting on November 23, 1934, a year and a half after its founding. The purpose of the society then, as now, "... shall be to further interest in tropical medicine and the related sciences, and to offer a forum for informal discussion of clinical, field, and laboratory problems." Three officers, a president, vice-president, and secretary-treasurer, were elected for one-year terms, a pattern that has persisted to the present. Membership was restricted to those "actively engaged in or interested in tropical medicine and hygiene, and in related sciences." The number of active members was first restricted to 25, but this was changed to 50 by the time the constitution and by-laws were promulgated in 1934. In 1947, the limit was set at 75 in response to large numbers of applicants who had returned from World War II service. In 1949, limits on the number of members were removed. Members in good standing had to attend at least one meeting per year and had to pay their annual dues, which in 1934 were $1.00. Dues were raised to $5.00 in 1968, and by 1990 were $15.00. Failure to meet either of the above requirements resulted in a member's being dropped from the society's rolls. The attendance requirement, fulfilled by some in recent years by attending the annual dinner meeting, was rigorously enforced until the 1970s.

Since the promulgation of the constitution and by-laws in 1934, the society has generally held four regular meetings per year, in November, January, March, and May.[4] On occasion, specially called meetings have been held to honor distinguished visitors. The cycle of holding the first meeting in the fall has been consistent, except for 1961 when the first meeting was held in April.

The incoming officers of the society take office at the first meeting of the year, which is often in November. A nominating committee of three members is appointed by the president at the third regular meeting of each year, and the nominations are voted on at the fourth meeting in May. As in many societies of this type, the secretary-treasurer carries a heavy burden of keeping minutes, sending out notices of meetings, collecting dues, making arrangements for meeting places, and organizing the annual dinner in May. In the early years of the society, the secretary-treasurer often secured the speakers for the four scientific meetings. However, in later years, some presidents assumed this responsibility, often in consultation with the other officers and members. The selection of the speaker for the Theobald Smith Memorial Lecture has long been the prerogative of the president of the society.

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EARLY DEVELOPMENTS

The group of 17 men who met at the New York Hospital on April 25, 1933, to found the society, were led by Ralph Welty Nauss, MD, who at the time was an Instructor in Public Health and Preventive Medicine at Cornell University Medical College (Fig 1). The group voted to have Nauss, who chaired the meeting, serve as chairman of the group, and Thomas Turlay Mackie, MD, to serve as secretary-treasurer for the first year (Fig 2). Mackie, who would serve in this post for nine years, was a Research Associate in Public Health and Preventive Medicine at Cornell. Two years previously, he had obtained a Diploma in Tropical Medicine and Hygiene from the London School of Hygiene and Tropical Medicine, and had been the recipient of the school's prestigious Duncan Medal.[5] It was decided at this meeting to postpone the election of regular officers until the following year after a constitution had been approved.[1(p26)]

The original 17 or charter members intended the society to be an informal grouping which would meet four times a year to discuss clinical, field, and laboratory issues in tropical medicine. From a certain perspective, it was initially an elitist group given its small size and the fact that admission to membership was by invitation only. In time, this character of the society changed, particularly after World War II, when the ranks of those interested in tropical medicine in the greater New York City area were swelled by returning physician veterans with extensive field and clinical experience. Many of these retained an interest in tropical medicine and entered private practice. Thus, by 1953, 25 years after the society's founding, of the then 96 members, 18 were in private practice compared to only one among the original founders.[1(p6)] In commenting on this increase in private practice members, the society's History Committee said:

> . . . there has been a threefold increase in the proportion of those who are in private practice. This trend may point the way to further expansion and wider usefulness in the future; but of one thing we can be sure, the Society will continue to prosper just as long as it adapts itself to changing conditions and responds to new needs."[1(p6)]

FIGURE 1. Ralph Welty Nauss, MD, who served as the chairman of the society during the first year of its existence (*Rev Med Trop Parasit* 1940; 6:104).

FIGURE 2. Thomas Turlay Mackie, MD, the society's first secretary-treasurer and 18th president (courtesy of the New York Academy of Medicine).

The charter members held four scientific meetings the first year, the first of which took place on November 3, 1933, seven months after the initial organizational meeting. The speaker, Wilbur A. Sawyer, MD, spoke on "Recent Developments Concerning Yellow Fever." Sawyer, who at the time was Associate Director of the International Health Division of the Rockefeller Foundation, had been a member of the Foundation's West African Yellow Fever Commission in 1926–1927. Previous Foundation commissions had set up a yellow fever laboratory in Lagos and had initiated research projects in West Africa.[6] Initiating a pattern that was to become the rule in later years, the second meeting on January 5, 1934, featured two speakers. One, Arthur W. Grace, spoke on "Filariasis in British Guiana and Jamaica," and the other, Cecil K. Drinker, on "Experimental Elephantiasis in Dogs." This balancing of the scientific program for a given year with clinical, epidemiological, and basic science research presentations was necessary in order to maintain the interest of the society's heterogeneous membership. In the late 1940s when two or more speakers per regular meeting became the rule, attempts were made to achieve this same balance within a given evening's program.

With only occasional changes in location, meetings were held at Cornell University Medical College-New York Hospital on a regular basis from 1933 to 1941. Because of labor problems at the hospital at the outset of World War II, the society was told it would have to meet elsewhere.[1(p5)] During the following year, meetings were then held at Roosevelt Hospital, New York University, and at the New York Academy of Medicine before the society once again returned to meet at Cornell in November 1942. The Cornell site remained consistent for the next six years until January 1949. Thereafter, meetings were held at either Columbia University School of Public Health or at New York University School of Medicine. Cornell once again hosted the society on January 9, 1963, after a hiatus of 14 years. The three institutions were the site of most meetings for the next two years. There were exceptions, as when occasional meetings were held at the Esso Standard Oil Auditorium in New York City. On May 27, 1965, the society held its annual dinner meeting and the 27th Theobald Smith Memorial Lecture at the Rockefeller Institute, which had moved to the east side of Manhattan from Princeton, New Jersey. Although the annual dinner and the Theobald Smith Memorial Lecture had been held at the Rockefeller Institute for the first time in March 1958, 1965 marked the beginning of a tradition which has been followed for most years ever since, with only occasional departures.

During the 1970s, meetings were held at Cornell, New York University, Rockefeller, and occasionally at Columbia. Beginning in the 1980s, increasing numbers of meetings were held at Rockefeller University. By the latter part of that decade, virtually all meetings were held there through the good offices of Dr William Trager, who served as the society's president in 1982–1983, and the university's generous hospitality.

The society's History Committee left a poignant description of the mood of the early meetings at Cornell in the 1930s:

> . . . the . . . members . . . will always cherish memories of those early winter twilights when they would gather in the great bow-window high up in the new hospital overlooking the East River and watch the yellow, twinkling lights of the city, while they waited to greet the speaker of the evening, whether he were Sir Aldo Castellani, fresh from the Abyssinian Campaign, or their own Max Theiler, from his laboratory just across the street.[1(p6)]

THE THEOBALD SMITH MEMORIAL LECTURE

At the society's sixth meeting on May 4, 1934, Dr Theobald Smith, then president of the board of the Rockefeller Institute in Princeton, New Jersey, addressed the 16 members and three guests present (Fig 3). Smith was one of the most eminent pathologists and microbiologists of his time, who received wide international recognition for his research on the transmission of various diseases. Among these was his demonstration in conjunction with F. L. Kilborne that ticks transmit Texas cattle fever (Piroplasmosis), a subject about which he spoke that evening.[7] Although he was never a member of the society, the society membership held him in very high regard.

Born on July 31, 1859, in Albany, New York, Smith received a PhB degree in 1881 from Cornell University and an MD degree from Albany Medical College in 1883. In 1884, he was appointed Director of the Pathological Laboratory and Assistant Chief of the Division of Pathology of the Bureau of Animal Industry in the Department of Agriculture. During the ten years he worked for the bureau, his research on a wide range of parasitic and infectious diseases led to important discoveries for which he received international recognition. He and Elmer Salmon, the chief of the bureau, elucidated swine plague and hog cholera, and later showed that cultures of killed bacteria could be used to produce immunity. This discovery made possible the development of vaccines against diseases such as typhoid fever and cholera.

FIGURE 3. Theobald Smith, MD (National Cyclopedia of American Biography).

While in Washington, DC, Smith organized the first department of bacteriology in a US medical school at Columbian University (later George Washington University). In 1895, he moved to Harvard as Professor of Applied Zoology and in 1896 became the first George Fabyan Professor of Comparative Pathology. While at Harvard, he also served as Director of the Pathological Laboratory of the Massachusetts Department of Health where he conducted research on antitoxins for tetanus and diphtheria. In 1901, he became a member of the Board of the Rockefeller Institute for Medical Research, and in 1914 joined the institute as Director of the Department of Animal Pathology. In 1933, he became president of the Board of the Rockefeller Institute.[8]

Smith was at the end of a long and illustrious career at the time he addressed the society on May 4, 1934. In fact, his talk was to be his last public appearance, as he died seven months later in New York City on December 10th. The History Committee left a vivid description of his visit and talk.

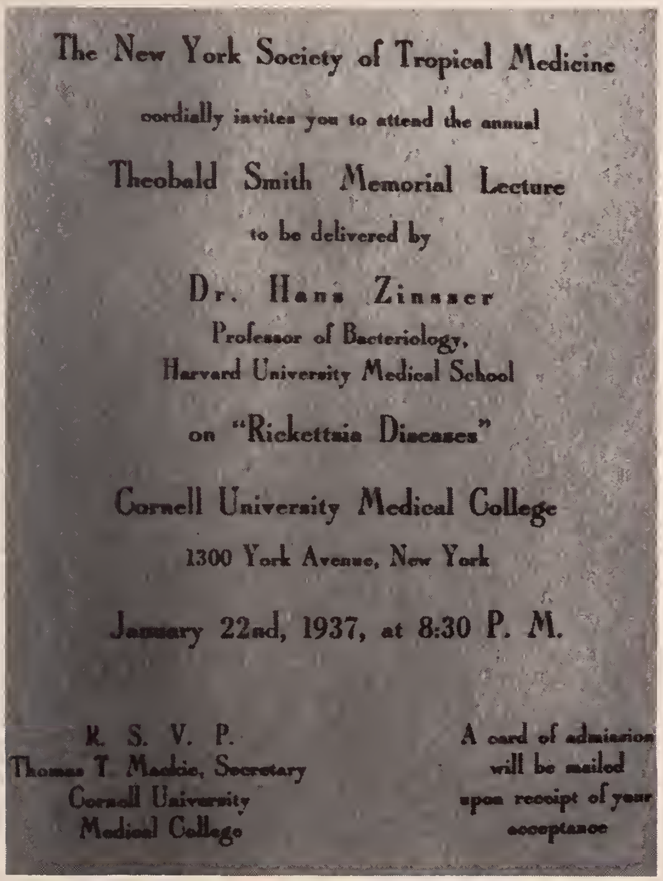
> During the meal, he conversed happily about his twenty years on the Harvard faculty. Later on, he spoke without notes for over an hour, showing lantern slides and telling the story of his earlier work for the Bureau of Animal Industry. He described the eco-

> nomic importance of Texas fever, and how he was able to prove that it was caused by the bite of an infected cattle tick . . . he expressed considerable regret that he could not return to the study of this most interesting disease . . .[1(p4)]

In 1936, the society voted to establish an annual Theobald Smith Memorial Lecture in his honor. The first lecture was delivered on January 22, 1937, by Hans Zinsser of Harvard University (Fig 4). Since 1937, the annual lecture has been delivered every year, except for 1960 and 1961 (Fig 5, Table I). Two lectures were held in 1949, the second to coincide with the US visit of the speaker, Sir Philip Manson-Bahr. As a result, no lecture was delivered in 1950. The lecture has been held in conjunction with the annual dinner, generally during the month of May, every year since 1959, except for 1989 when no dinner preceded the lecture.

SCIENTIFIC ACTIVITIES

By the late 1940s, the society's meetings usually included more than one lecturer. In addition, demonstrations consisting of 35 mm projection slides and microscopic slides were also presented at some meetings, especially in the 1960s and 1970s. The society also occasionally co-sponsored meetings, as for example when it joined with the Section on Medicine of the New York Academy of Medicine on February 17, 1942, to present three speakers. Dr Thomas T. Mackie spoke on "Florence Nightingale and Tropical and Military Medicine," Dr Henry E. Meleny spoke on "Tropical Medicine in United States Military History," and Dr John C. Snyder spoke on "Typhus Fever." The society has regularly presented groups of speakers who have covered diverse aspects of a given topic. These topics have either been addressed in an evening session or in a larger symposium format, sometimes co-sponsored by another organization such as the New York Academy of Medicine. In 1953, the society devoted one meeting to schistosomiasis (January 15) and another to medical mission work (April 24). The toxicity of drugs then used in treating schistosomiasis was covered on January 27, 1955, and clinical difficulties in relation to parasitism on November 15, 1963. The tropical disease activities of the New York City Department of Health were presented by five speakers on March 20, 1964, and on March 11, 1966, Dr Harry Most and four of his colleagues at New York University spoke on different aspects of "Recent Studies in Rodent Malaria."

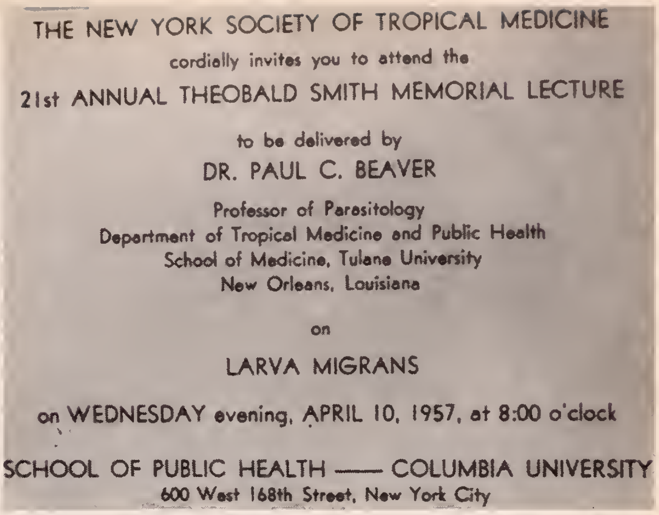


The New York Society of Tropical Medicine
cordially invites you to attend the annual
Theobald Smith Memorial Lecture
to be delivered by
Dr. Hans Zinsser
Professor of Bacteriology,
Harvard University Medical School
on "Rickettsia Diseases"
Cornell University Medical College
1300 York Avenue, New York
January 22nd, 1937, at 8:30 P. M.

R. S. V. P.
Thomas T. Mackie, Secretary
Cornell University
Medical College

A card of admission
will be mailed
upon receipt of your
acceptance

FIGURE 4. Announcement of the First Annual Theobald Smith Memorial Lecture, 1937 (Society Archives).



THE NEW YORK SOCIETY OF TROPICAL MEDICINE
cordially invites you to attend the
21st ANNUAL THEOBALD SMITH MEMORIAL LECTURE
to be delivered by
DR. PAUL C. BEAVER
Professor of Parasitology
Department of Tropical Medicine and Public Health
School of Medicine, Tulane University
New Orleans, Louisiana
on
LARVA MIGRANS
on WEDNESDAY evening, APRIL 10, 1957, at 8:00 o'clock
SCHOOL OF PUBLIC HEALTH —— COLUMBIA UNIVERSITY
600 West 168th Street, New York City

FIGURE 5. Announcement of the 21st Annual Theobald Smith Memorial Lecture, 1957 (Society Archives).

In addition to covering topics in depth, the society has also sponsored symposia. These have included "Leprosy—New Concepts," co-sponsored with the New York Academy of Medicine, the American Leprosy Missions, and the Leonard Wood Memorial, held on April 1, 1969. On March 14, 1974, the society sponsored a symposium, "Anisakiasis—A New Disease from Raw Fish," which featured six speakers, and on October 29, 1980, the society co-sponsored a "Symposium on Amebiasis—A New Look at an Old Disease," with the New York Academy of Medicine. This symposium focused on sexually transmitted amebiasis, which was then a major problem in New York City and elsewhere.[9]

In 1970, Lassa Fever was a new and significant disease just uncovered in Africa. On January 20, 1970, the society heard presentations from Dr John D. Frame of Columbia University and Dr Jordi Casals of Yale University, leading authorities on the disease. Dr Casals became ill with infection himself while working with blood samples from a patient who had contracted the disease in Africa. He was later successfully treated with this patient's convalescent serum.[10,11] Continuing its tradition of presenting up-to-date information to its members, the society recently heard Dr Mahfouz Zaki, the Director of Public Health of Suffolk County, New York, speak on "Current Trends in

TABLE I. Theobald Smith Memorial Lectures of the New York Society of Tropical Medicine, 1937–1990

Number	Year	Speaker and Topic
1st	1937	Hans Zinsser (Harvard University Medical School): *Rickettsia Diseases*
2nd	1938	George W. McCoy (National Institute of Health): *Plague in the United States*
3rd	1939	Richard P. Strong (Harvard University Medical School): *Filarial Disease in the Western Hemisphere*
4th	1940	Edward B. Vedder (Col. MC, USA, Retired): *Beri Beri and Vitamin* B_1 *Deficiency*
5th	1941	Carl Ten Broeck (Rockefeller Institute): *Theobald Smith as I Knew Him*
6th	1942	Edward E. Tyzzer (Harvard University Medical School): *Blood Infections in Small Native Mammals*
7th	1943	M. Ruis Castaneda (Director, Typhus Fever Research, Mexico): *The Role of Strain Variation in Typhus Vaccination*
8th	1944	Marshall C. Balfour (Rockefeller Foundation): *Current Problems of India and China in Relation to Tropical Medicine*
9th	1945	Paul F. Russell (Rockefeller Foundation): *Malaria in India*
10th	1946	Willard H. Wright (National Institute of Health): *Experiences with Schistosomiasis Japonica in the Philippine Islands and Japan*
11th	1947	Abner Wolf (Columbia University College of Physicians and Surgeons): *Congenital Toxoplasmosis*
12th	1948	Cornelius B. Philip (Rocky Mountain Laboratory, National Institute of Health): *Tsutsugamushi Disease (Scrub Typhus) in World War II*
13th	1949	Fred L. Soper (Director of the Pan American Sanitary Bureau): *The 1949 Yellow Fever Situation in the Americas*
14th	1949	Sir Philip Manson-Bahr (Director, Division of Clinical Tropical Medicine, London School of Hygiene and Tropical Medicine): *Diarrheal Diseases with Special Reference to the Late Effects of Amebiasis*
15th	1951	Kenneth C. Smithburn (International Health Division of the Rockefeller Foundation): *Studies of Neurotropic Viruses Isolated in Africa from Human Beings and from Insects*
16th	1952	Karl F. Meyer (Director, G. W. Hooper Foundation, University of California Medical Center): *Plague in the Light of Newer Knowledge*
17th	1953	Herald R. Cox (Director of Virus and Rickettsial Disease Section, Lederle Laboratories): *Living Viruses as Immunizing Agents*
18th	1954	Louis L. Williams, Jr (Medical Director (retired) USPHS): *Tropical Medicine and International Relations*
19th	1955	William H. Taliaferro (Professor and Chairman of the Department of Microbiology, University of Chicago): *The Effects of X-Rays on Antibody Formation*
20th	1956	John B. Poole (Sterling-Winthrop Research Institute): *Echinococcus Disease in North America and Its Prevalence among Natives in Canada's Northwest Territories*
21st	1957	Paul C. Beaver (Professor of Parasitology, Department of Tropical Medicine and Public Health, Tulane University): *Larva Migrans*
22nd	1958	Donald B. McMullen (Chief, Department of Medical Zoology, Walter Reed Army Institute of Research): *Biological Aspects of Schistosomiasis Control*
23rd	1959	Benjamin Schwartz (Principal Parasitologist, US Department of Agriculture, Agricultural Research Service, Animal Disease and Parasite Research Division): *Evolution of Knowledge Concerning Ascaris lumbricoides*
24th	1962	Norman Stoll (Associate Professor, The Rockefeller Institute): *On Endemic Hookworm, Where Do We Stand Today?*
25th	1963	R. Elsdon-Dew, FRSSAf (Director of the Amoebiasis Research Unit, Institute for Parasitology, Durban, SA): *Amoebiasis*
26th	1964	Arnold Gabaldon (Former Minister of Health and Social Welfare of Venezuela): *Bilharziasis, Chagas' Disease and Malaria in Venezuela*
27th	1965	Nevin S. Scrimshaw (Head, Department of Nutrition, Massachusetts Institute of Technology): *Nutrition and Infection*
28th	1966	Col. William D. Tiggert, MC (Director, Walter Reed Army Institute of Research): *Important Aspects of Tropical Medicine in Viet Nam*
29th	1967	Harry Hoogstraal (US Naval Medical Research Unit No. 3, Cairo, UAR): *Tickborne Hemorrhagic Fevers, Encephalitis and Typhus in USSR and Southern Asia*
30th	1968	Michael Heidelberger (New York University School of Medicine): *Chemical Constitution and Immunological Specificity*
31st	1969	Jordi Casals (Yale University School of Medicine): *Certain Aspects of the Immune Response of Vertebrates to Arbovirus Infections*
32nd	1970	P. C. C. Garnham (London School of Hygiene and Tropical Medicine): *Malaria Research: Old Problems Solved and the Challenge of the New*
33rd	1971	George S. Nelson (London School of Hygiene and Tropical Medicine): *Zooprophylaxis: The Prevention or Amelioration of Disease in Man as a Result of Previous Exposure to Heterologous Infections of Animal Origin*
34th	1972	Edward F. Knipling (Science Advisor, Agricultural Research Service, US Department of Agriculture): *The Principles of Insect Control by Genetic Means*
35th	1973	Robert M. Lewert (Department of Microbiology, University of Chicago): *Schistosoma japonicum: Field and Laboratory Immunology of the Philippine Disease*
36th	1974	Wilbur G. Downs (Clinical Professor of Epidemiology, Yale University School of Medicine): *Malaria—The Great Umbrella*

TABLE I continued

Number	Year	Speaker and Topic
37th	1975	Thomas H. Weller (Professor and Chairman, Department of Tropical Public Health, Harvard University School of Public Health): *Manson's Schistosomiasis—Retrospective and Prospective Reflections*
38th	1976	William Trager (Professor of Parasitology, Rockefeller University): *Human Malaria Parasites in Continuous Culture*
39th	1977	George B. Mackaness (President, Squibb Institute for Medical Research): *Prospects for an Anti-Malarial Vaccine*
40th	1978	Kenneth S. Warren (Director for Health Sciences, Rockefeller Foundation): *Worms and People, A Unique Relationship*
41st	1979	Carl Alving (Chief, Department of Membrane Biochemistry, Walter Reed Army Institute of Research): *Liposomes as Drug Carriers in Parasitic Diseases*
42nd	1980	Frans von Lichtenberg (Professor of Pathology, Peter Bent Brigham Hospital, Harvard Medical School): *In vivo veritas*
43rd	1981	Ernest Bueding (Johns Hopkins University): *Selective Approaches to the Chemotherapy of Schistosomiasis*
44th	1982	Elmer Pfefferkorn (Dartmouth Medical School): *Genetic Approaches to Understanding the Biochemistry of the Host-Parasite Relationship in Cultured Cells Infected with Toxoplasma gondii*
45th	1983	B. H. Kean (Clinical Professor (Emeritus) of Tropical Medicine and Public Health, The New York Hospital-Cornell Medical Center): *Patients, Parasites and Parasitologists*
46th	1984	Saul Krugman (Professor of Pediatrics, New York University School of Medicine): *Viral Hepatitis: New Light on an Old Disease*
47th	1985	William C. Campbell (Senior Director, Basic Parasitology, Merck Institute for Therapeutic Research): *Tropical Flukes and Frozen Hands—An Excursion in Historical Parasitology*
48th	1986	Ruth S. Nussenzweig (New York University School of Medicine): *Experimental Basis for the Development of a Malaria Vaccine*
49th	1987	Barry R. Bloom (Albert Einstein College of Medicine): *Learning from Leprosy*
50th	1988	Myron Levine (Director, Center for Vaccine Development, University of Maryland School of Medicine): *Clinical Trials of Candidate Malaria Vaccines*
51st	1989	Jonathan I. Ravdin (University of Virginia Health Sciences Center): *Update on Amebiasis*
52nd	1990	John P. Craig (Professor of Microbiology and Immunology, State University of New York, Health Science Center at Brooklyn): *The Continuing Search for the Cause of Cholera*

the Epidemiology of Lyme Disease." In addition to the titles of regular lectures, those of the annual Theobald Smith Lecture also chronicle the changing challenges in tropical medicine and topics of concern to the society's members (Table I).

THE SOCIETY'S SEAL

At the time of its founding, the society did not adopt a seal or logo. However, in 1974, Dr Meir Yoeli, then president of the society and Professor of Preventive Medicine at New York University Medical Center, proposed an official seal.[12] It was his hope that the seal would be used on letterheads, invitations, and possibly as a model for an engraved medallion to be given to invited distinguished lecturers. He had a preliminary drawing made by an artist friend of his, Sam Kweskin (Fig 6).

Dr Yoeli sent out copies of the design with a circular letter inquiring if the society should have a seal and asking for opinions of the one Kweskin had designed. A committee was appointed to study the seal and make a recommendation for or against adoption. As it was deliberating, a number of members sent in their opinions about the seal.

A small minority strongly opposed the need for a seal citing unnecessary costs and the fact that it was incompatible with the informal character of the society. Others had numerous suggestions to make about the design itself. The mosquito hovering over the New York City skyline came in for especially sharp criticism on the grounds that malaria and yellow fever were no longer locally transmitted. Some suggested substituting an airplane for the mosquito, and an ocean liner for the ferry/tug boat. Others found it disturbing that the head of the Statue of Liberty merged into the skyline, and one favored an historical skyline of the time when malaria and yellow fever were endemic. The pair of worms on the right side disturbed some who favored use of a larvated worm egg. And one member found the whole design lacking in dignity. Artistic criticism was extended to the lettering (too crowded at the top), the absence of the society's founding date, and to what some saw as a lack of symmetry.

Despite the criticism, a resolution for the acceptance of

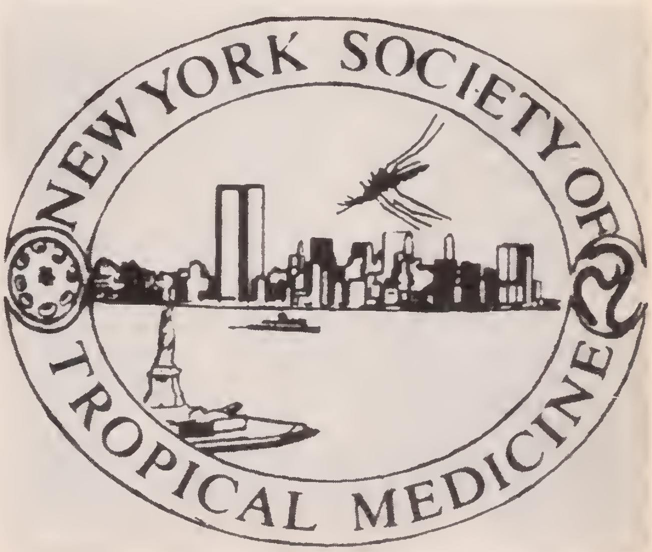


FIGURE 6. Society seal adopted in 1975 (Society Archives).

the seal was presented at the society's March 4, 1975 meeting, and a request made for a written ballot. The members present overwhelmingly approved adoption of the original seal. On May 4, 1975, Dr Yoeli wrote to Dr Herman Zaiman, the society's secretary-treasurer, informing him that the seal could be used on all documents and stationery.[13]

Although the seal was approved, it was never embraced with enthusiasm by subsequent officers. In part this was due to the fact that each new president and secretary-treasurer generally makes up his or her own stationery, which is usually rudimentary since it is used for only a short time. However, resistance to using a seal which did not have the weight of historical precedent behind it must have also played a role.

PROFILES OF EARLY OFFICERS

A brief look at the professional profile of some of the society's early officers provides insights into the broad scope of their interests.

Ralph Welty Nauss, MD (1905–1959). A graduate of Northwestern University Medical School, Nauss served as the chairman of the society during its first year of existence and the following year as its vice-president. He was a faculty member of Cornell University Medical College who also spent three years as a medical missionary in the Belgian Congo (now Zaire) and later authored *Medical Parasitology and Zoology*.[14]

Wilbur A. Sawyer, MD (1879–1952). Dr Sawyer, a graduate of Harvard Medical School, succeeded Nauss as president of the society in 1934 when he was in charge of the Rockefeller Foundation's Yellow Fever Laboratory (Fig 7). Eventually he became Director of the Foundation's International Health Division. In 1943–1944, he served as president of the American Society of Tropical Medicine.[15]

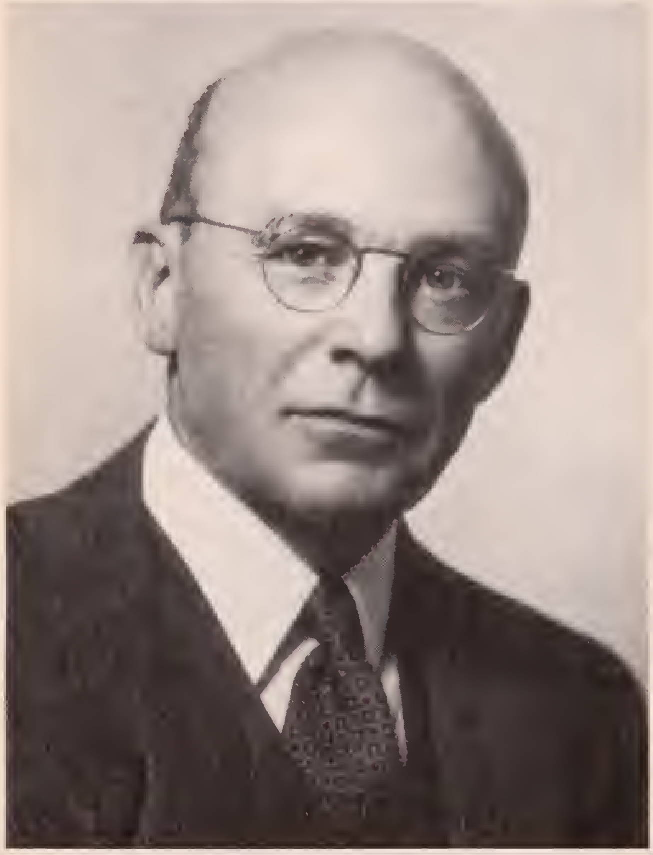


FIGURE 7. Wilbur A. Sawyer, MD (1951), the society's second president (courtesy of the Rockefeller Archive Center).

Charles St. John Butler (1875–1944). Dr Butler, a graduate of the University of Virginia Department of Medicine, was the society's third president at a time when he was commander of the US Naval Hospital in Brooklyn, New York. He later became commanding officer in charge of the US Naval Medical Center in Washington, DC. He authored *Syphilis Sive Morbus Humanus* in 1936.[16,17]

Alvin W. Schoenleber, MD (1887–1972). A graduate of the University of Illinois, Dr Schoenleber became the society's president in 1936. He was the Medical Director of the Standard Oil Company of New Jersey.[18]

Eugene Lindsay Opie (1875–1971). A graduate of Johns Hopkins University School of Medicine, he had previously served as vice-president and became president when he was Professor of Pathology at Cornell University Medical College. Opie was among the most eminent pathologists of his time and was associated with both the Rockefeller Institute and the International Health Division of the Rockefeller Foundation.[19]

Wade Wright Oliver, MD (1890–1965). Dr Oliver was a 1915 graduate of the University of Cincinnati and became president at the time that he was Professor and Chairman of the Department of Bacteriology at the Long Island College of Medicine, where in 1935–1937 he also served as Acting Dean. In 1916–1917, he participated in the Rockefeller Foundation's Hookworm Survey in Brazil and then joined the faculty at the Long Island College of Medicine. Dr Oliver was a noted bacteriologist and also a highly respected poet. He was the author of two volumes of poetry, *Sky Rider* (1928) and *Fantasia* (1938), and the recipient of several poetry prizes in the US and Great Britain. In 1930, his book, *Stalkers of Pestilence*, a history of bacteriology and medicine, won critical acclaim. His biography of William Hallock Park, *The Man Who Lived for Tomorrow*, was published in 1941. In 1948–1950, he served as the president of the Harvey Society.[20,21]

Louise Pearce, MD (1885–1959). Dr Pierce was the first woman officer of the society, serving as vice-president from 1928 to 1929 (Fig 8). A graduate of Johns Hopkins University School of Medicine, she was associated with the Rockefeller Institute for many years. In 1920–1921, she led the Institute's African Sleeping Sick-

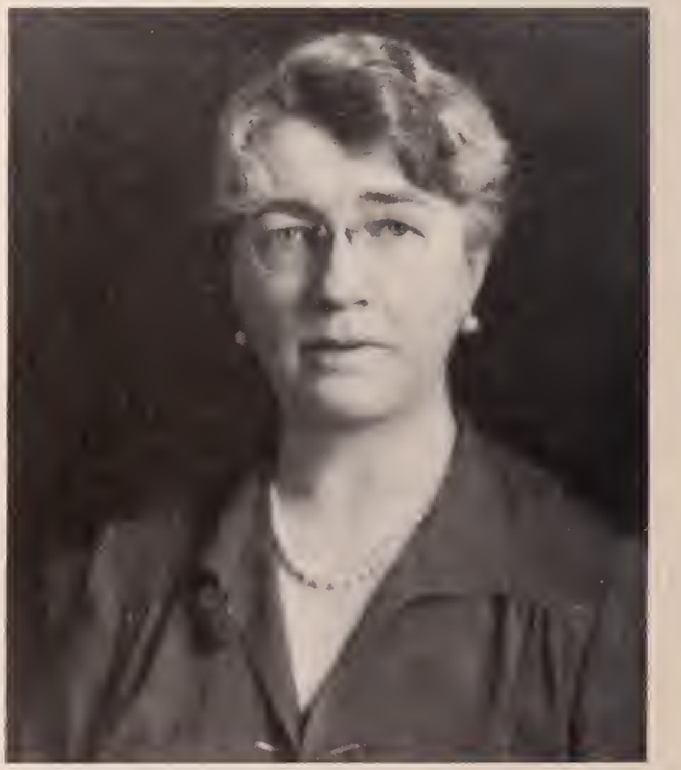


FIGURE 8. Louise Pearce, MD (1934), vice-president, 1938–1939 (courtesy of the Rockefeller Archive Center).

ness Mission to the Belgian Congo (now Zaire). From 1946 to 1951, she served as the president of the Women's Medical College of Pennsylvania.[22]

Howard Fox, MD (1873–1954). Dr Fox succeeded Dr Oliver as president in 1939. A graduate of Columbia University College of Physicians and Surgeons, he was a distinguished dermatologist and Professor of Dermatology and Syphilology at New York University College of Medicine. He served as president of the American Dermatological Association in 1925–1926 and was for many years president of the American Academy of Dermatology and Syphilology. He was a consulting editor of the *New York State Journal of Medicine* and editor-in-chief of the *Archives of Dermatology and Syphilology*.[23]

Horace W. Stunkard, PhD (1889–1989). Dr Stunkard, a founding member of the society, died last year at the age of 100. In 1916, he received his doctorate in biology from the University of Illinois and then joined the Department of Biology at New York University, of which he eventually served as chairman for 29 years. He was also a Research Associate at the American Museum of National History from 1921 until his death. He served as president of the New York Academy of Sciences in 1927, 1942, and 1943, and president of the American Microscopical Society in 1936.[24] Dr Stunkard was a loyal supporter of the society. During his later years, he regularly rose to his feet during the short business meeting following the annual dinner and moved that the nominations for officers be closed. This ritual was repeated for so many years that it became an integral part of the dinner meetings.

McIver Woody, MD (1886–1970). A graduate of Harvard Medical School, Dr Woody served as a physician with the Esso Standard Oil Company of New Jersey beginning in 1922. He then served as Medical Director of the company from 1944 to 1951. He was the first president of the society to serve two consecutive terms, 1941–1942 and 1942–1943.

Wilson George Smillie, MD (1886–1971). Dr Smillie, who also served two terms as president, was a graduate of Harvard Medical School, from which he also obtained a DrPH degree in 1916 (Fig 9). After working as a fellow at the Rockefeller Institute for a year, 1916–1917, he was Assistant Professor at the State Medical School in Sao Paulo, Brazil, from 1918 to 1920. From 1923 to 1935, he was associated with the International Health Division of the Rockefeller Foundation, and from 1937 to 1955 was Professor and Chairman of the Department of Public Health and Preventive Medicine at Cornell University Medical College. He was the author of numerous books including *Public Health Administration in the United States* (1937, 1940, 1947), *Preventive Medicine and Public Health* (1946), and *The History of Public Health in America* (1953).[25]

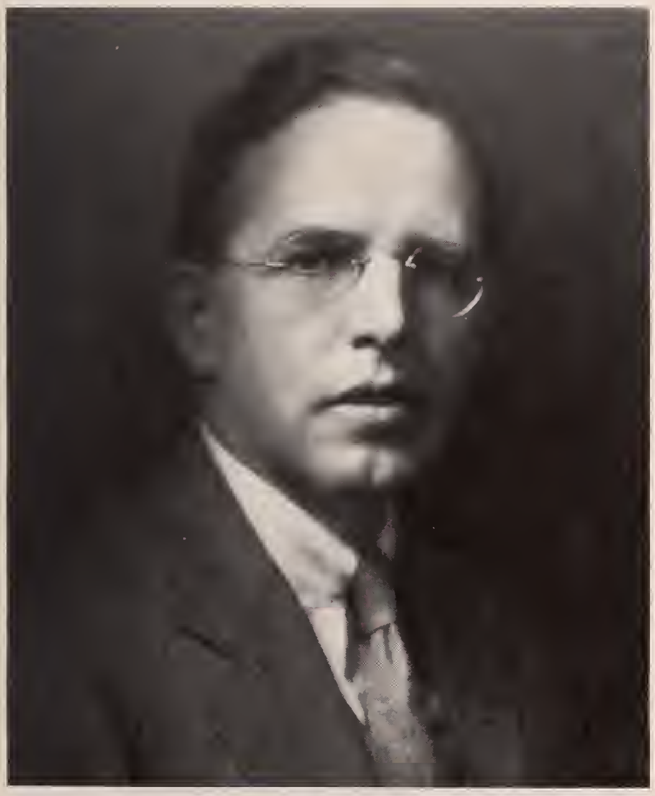


FIGURE 9. Wilson George Smillie, MD, who served as vice-president in 1942–1943 and as president in 1943–1945 (courtesy of the Rockefeller Archive Center).

Henry Edmund Meleney, MD (1887–1971). Dr Meleney, who graduated from Columbia University College of Physicians and Surgeons, served for two terms as president at a time when he was Professor of Preventive Medicine at New York University School of Medicine. A noted pathologist who made many original research contributions, he spent two years (1920–1922) as a pathologist at Peking Union Medical College. He served as president of the American Society of Tropical Medicine in 1935, and as president of the American Society of Parasitology in 1942–1943. His research interests centered on malaria, leishmaniasis, amebiasis, schistosomiasis, and relapsing fever.[26]

Norman Rudolf Stoll, ScD (1892–1976). Dr Stoll, who became president in 1947, is considered one of the most eminent parasitologists of this century (Fig 10). After receiving his doctorate from Johns Hopkins University School of Hygiene and Public Health, he taught high school biology in Massilon, Ohio. He later served on the Commission to Investigate Hookworm Disease in Puerto Rico (1922), China (1923–1924), and Panama (1926). While in China, he served as an associate in parasitology at Peking Union Medical College. In 1925, he joined the staff of the Rockefeller Institute as an associate member and remained with the institution until his retirement in 1962. He was a member of the World Health Organization's Expert Advisory Panel on Parasitic Diseases between 1952

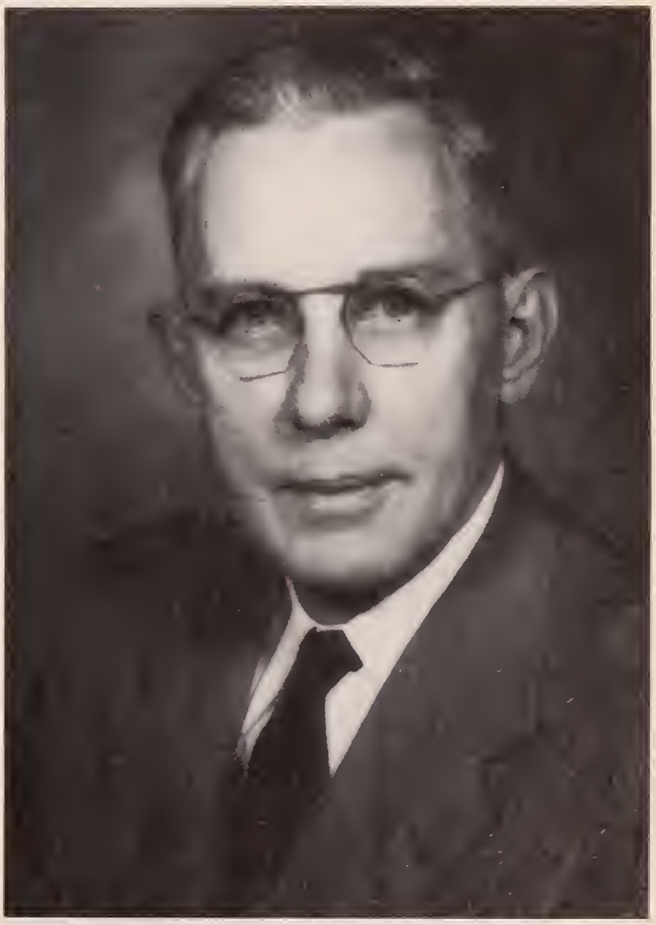


FIGURE 10. Norman Rudolf Stoll, ScD (1953), vice-president in 1939–1940 and president in 1947–1948 (courtesy of the Rockefeller Archive Center).

and 1973. A charter member of the American Society of Parasitology, he served as its president in 1946. Widely known for his research in helminthology, he authored a classic paper, "This Wormy World."[27,28]

Edward Hicks Hume, MD (1876–1957). Dr Hume served four terms as the society's vice-president during the period 1943 to 1948 (Fig 11). Born in India, he received his MD from John Hopkins University School of Medicine in 1901. He then worked in India for two years (1903–1905) as an acting associate surgeon with the United States Public Health Service. From 1906 to 1927, he worked in China with Yale University, serving for several years as dean of the Hunan-Yale Medical College. He was the author of several highly acclaimed books including *The Chinese Way of Medicine* (1940), *Doctors East, Doctors West* (1946), and *Doctors Courageous* (1950).[29-32]

Morton Charles Kahn, PhD (1895–1959). Dr Kahn, who received his PhD from Cornell University in 1927, served as the society's second secretary-treasurer for five years, 1942–1947. In 1922, he participated in a Collecting Expedition to Central America and to British Guiana in 1925. A bacteriologist, he taught for many years at Cornell University Medical College where his research interests focused on anaerobic spore-bearing bacteria and the bacteriology of gas gangrene.[33]

Victor George Heiser, MD (1873–1972). Dr Heiser, the society's thirteenth president, ranks among the leading American physicians of the 20th century (Fig 12). In 1889, when he was 13 years old, he survived the Johnstown flood but lost both of his parents in it. Working as a plumber and a carpenter, he saved up enough money to attend Jefferson Medical College, from which he graduated in 1897. He joined the United States Health Marine Service (the predecessor of the United States Public Health Service), and in 1903 became Chief Quarantine Officer in the Philippines. He remained in the Philippines until 1915, eventually becoming Director of Health. It was while he was in the Philippines that he first developed what would become a life-long interest in leprosy. In 1915, he became Director for the East on the International Health Board of the Rockefeller Foundation, a position he held until 1927. For the next six years, he served as Associate Director of the Rockefeller Foundation's International Health Division. From 1931 to 1938, he served as the first president of the International Leprosy Association, and for many years was a member of the medical board of the Leonard Wood Memorial for the Eradication of Leprosy. Dr Heiser circled the globe 17 times while on medical missions, some of which are described in his bestselling and now classic autobiography, *An American Doctor's Odyssey*.

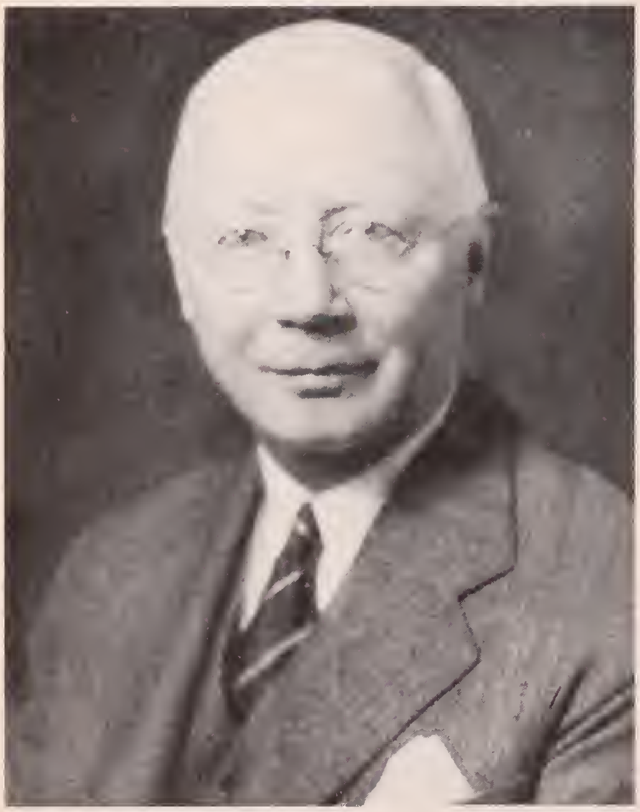


FIGURE 11. Edward Hicks Hume, MD, who served for four terms as vice-president (reproduced with permission from *The Chinese Way of Medicine*, Baltimore, Md, The Johns Hopkins University Press, 1940).

Dr Heiser, who had served as president of the American Society of Tropical Medicine in 1921–1922, became president of the New York Society of Tropical Medicine in 1948, when he was 75 years old. He remained physically active throughout the later years of his life and regularly attended the society's meetings. When he died in 1972, he left a multi-million dollar bequest to the New York Community Trust to be used for basic biomedical research on leprosy. The Heiser Program currently consists of research grants, post-doctoral research fellowships, and visiting research awards.[34-37]

Harold William Brown, ScD, MD (1902–1988). Dr Brown, who was the society's 14th president (1949–1950), received his doctorate in parasitology from Johns Hopkins University in 1928, and his MD degree from Vanderbilt University in 1933. He also received a DrPH degree from Harvard in 1936. From 1937 to 1943, he was Professor of Public Health at the University of North Carolina School of Public Health, and later served as dean of the school from 1941 to 1943. In 1943, he became Professor of Parasitology at Columbia University School of Public Health, and from 1947 to 1951 was acting director of the school, becoming director in 1951 for a period of four years. Following this, he remained at Columbia as Professor of Parasitology. Between 1957 and 1964, he was a Visiting Professor at the University of Taiwan on several occasions, and in 1962 and 1964 was Visiting Professor at Airlangga University in Indonesia. He served as president of the American Society of Parasitology in 1960, and vice-president of the American Society of Tropical Medicine in 1944. Dr Brown's research focused on anti-helminthics, malaria, and the chemotherapy of amebiasis. Recognized in his lifetime as an eminent parasitologist and teacher, he was the recipient of numerous honors and awards including the Medal of the New York Academy of Medicine in 1969. Dr Brown was co-author of a standard text in parasitology, *Basic Clinical Parasitology*, which is widely used by medical students.[38-40]

Herald Rea Cox, ScD (1907–). Dr Cox, who succeeded Dr Brown as president, received his doctorate in filterable viruses at Johns Hopkins University in 1931. During the 1930s, he was associated with the Rockefeller Institute and the United States Public Health

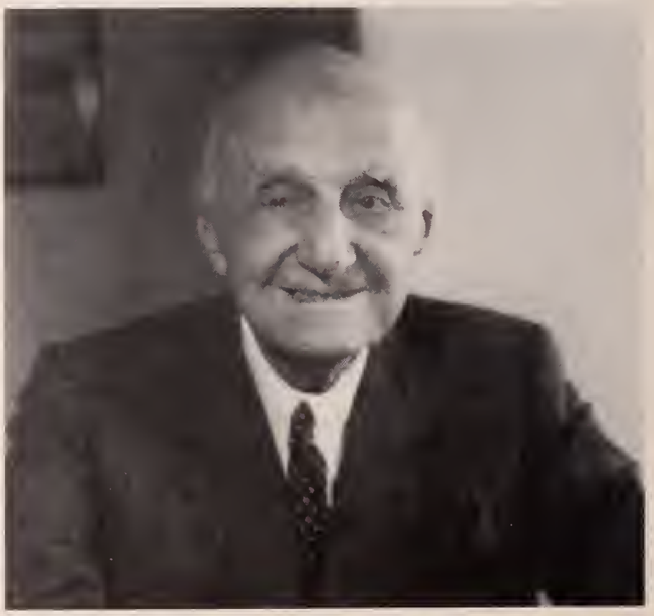


FIGURE 12. Victor George Heiser, MD, who served as president in 1948–1949 (courtesy of the Rockefeller Archive Center).

Service. In 1940, he joined Lederle Laboratories, and in 1944 became Director of the Viral and Rickettsial Disease Section, a position he held for many years. In 1960–1961, he served as president of the American Society of Microbiology. From 1968 to 1972, he was Director of Cancer Research, Viral Oncology Section, Roswell Park Memorial Institute. His research interests focused on neurotropic viral diseases, rickettsial diseases, and live virus vaccines.[41]

Zacharias Taylor Bercovitz, PhD, MD (1895–1984). Dr Bercovitz served as vice-president of the society for three years and became president in 1951. He received a PhD degree in physiology in 1924 and an MD degree the same year from Rush Medical College. From 1924 to 1929, he was a medical missionary in Korea, serving as the Physician in Charge of the Presbyterian Mission Hospital in Andong, and from 1929 to 1930 was a student at the London School of Hygiene and Tropical Medicine. He took a year's fellowship at the New York Post-Graduate Medical School and also joined the faculty of the Department of Medicine of Columbia University College of Physicians and Surgeons and the staff of the Presbyterian Hospital. From 1934 to 1943, he was the Physician in Charge of the Parasitology Service of the Bureau of Laboratories of the New York City Department of Health. He was associated with New York University Medical Center from 1949 to 1964. In 1964, he entered private practice, and in 1975 moved to LaJolla, California. His areas of interest were clinical tropical medicine, medical parasitology, and ulcerative colitis.[42]

Kenneth Rushton Fourcher, MD (1904–1977). Dr Fourcher, who served as the society's 17th president in 1952–1953, received his MD degree from the Medical College of Georgia in 1928. After a year of internship, he became associate physician at the Samaritan Hospital in Panama for a period of two years. In 1930, he became associate physician at the Talara Hospital in Talara, Peru, a position he held for ten years. In 1940, he joined the Standard Oil Company of New Jersey as Associate Medical Director, remaining in this position until 1953. For the next nine years, he was Medical Director of the Standard-Vacuum Oil Company. In 1962, he returned to the Standard Oil Company for two years as Associate Medical Director.[43]

Thomas Turlay Mackie, MD (1895–1955). Dr Mackie was among the most eminent tropical medicine specialists of his time and the society's 18th president, serving in 1953–1954. A graduate of Columbia University, College of Physicians and Surgeons (1924), he did post-graduate work at the London School of Hygiene and Tropical Medicine. From 1926 until 1935, he was associated with his alma mater in a variety of capacities. However, in 1933 he joined the faculty of Cornell University Medical College as a Research Associate in Public Health and Preventive Medicine. During World War II, he served as a Colonel in the Medical Corps and as Executive Officer of Tropical and Military Medicine and Chief of the Division of Parasitology at the Army Medical School. Following the war, he became Professor and Chairman of the Department of Preventive Medicine and Director of the Institute of Tropical Medicine at Bowman-Gray School of Medicine of Wake Forest College, positions he held until 1951. He became a consultant and branch section chief, tropical medicine, for the Veterans Administration and served as a consultant in tropical medicine to companies working in the tropics. Dr Mackie had served as the society's first secretary-treasurer for a period of nine years, a term longer than anyone else who has since served in that position. His term as the society's president ended a year before his untimely death from a pulmonary embolism.[5] He was the co-author of what has become a classic volume, *A Manual of Tropical Medicine* (1945).[44]

Donald Vincent Moore, PhD (1915–). Dr Moore, the society's 19th president, received his doctorate in parasitology from Rice Institute in 1942. After working as a senior parasitologist with the Bureau of Laboratories of the Texas State Department of Health, he joined the faculty of Cornell University Medical College in 1946 as an Assistant Professor of Preventive Medicine. He remained at Cornell until 1974, serving from 1955 onwards as Assistant Professor of Microbiology. In 1974, he became Associate Professor of Pathology, Microbiology, and Medical Laboratory Science at the University of Texas Health Science Center in Dallas.[45]

Howard B. Shookhoff, MD (1910–1977). Dr Shookhoff, an internationally respected authority on tropical diseases, became the society's 20th president in 1955 (Fig 13). He received his MD degree from Columbia University, College of Physicians and Surgeons in 1933, and a diploma in tropical medicine from the London School of Hygiene and Tropical Medicine in 1938. From 1938 to 1942, he practiced internal medicine in New York City. During World War II, he served as the chief of a field party in Colombia for the Health and Sanitation Division of the Office of Inter-American Affairs. In 1944, he became an epidemiologist in tropical diseases with the New York City Department of Health, and in 1946 Physician in Charge of the department's Tropical Disease Diagnostic Service. In 1954, this service was made a division in the department's Bureau of Preventable Diseases. As Chief of the Tropical Disease Division, Dr Shookhoff supervised four public clinics which provided free diagnostic and therapeutic services. The influx of large numbers of new immigrants from the Caribbean, especially Puerto Rico, and intensification of air travel resulted in a rapid growth in the numbers of patients seen by the division's clinics. By the early 1970s, these clinics were managing 8,000 patients and 25,000 patient visits per year.[46] Schistosomiasis was a major problem among many of the Caribbean immigrants. Beginning in 1940, Dr Shookhoff became associated with Columbia University College of Physicians and Surgeons, where at the time of his death he was Adjunct Professor of Tropical Medicine. He was also Visiting Associate Professor of Preventive and Environmental Medicine at the Albert Einstein College of Medicine. Dr Shookhoff was associated with the Presbyterian Hospital, Montefiore Hospital, and Bronx Municipal Hospital. He is affectionately remembered by his colleagues as a warm and personable man, and as a caring and sensitive physician. Highly respected for his vast knowledge of tropical diseases and his clinical acumen, he was also admired for his modesty.[47,48] In 1980, the society co-sponsored a symposium with the New York Academy of Medicine entitled, "Amebiasis—A New Look at an Old Disease," which was dedicated to Dr Shookhoff's memory.[9]

Meir Yoeli, MD (1912–1975). Dr Yoeli, who became the socie-

FIGURE 13. Howard B. Shookhoff, MD, who was vice-president in 1954–1955 and president in 1955–1956 (courtesy of Mrs Muriel Shookhoff).

ty's 38th president in 1974, was among the leading malaria researchers of his time. Born in Kaunas, Lithuania, he received his MD degree from Basel in 1939 and studied tropical medicine at the University of Padova in Italy. From 1934 to 1938, he was a scientific assistant at the malaria research station, Hebrew University, Israel. During World War II, he was a medical officer with the British Army Medical Corps in North Africa, and participated in the siege of Tobruk. In 1948, he served as the head of the Department of Preventive Medicine of the Israeli Defense Forces. From 1949 to 1956, he was a lecturer in parasitology and tropical medicine at the Hebrew University in Israel. In 1956, he joined the faculty of New York University Medical Center as an Associate Professor of Preventive Medicine where he worked with Dr Harry Most, the department's chairman. Dr Yoeli and his associates received worldwide recognition for their research advances in malaria. They demonstrated how rodent plasmodia could be used in place of human and simian ones for experimental research.[49,50]

The above profiles provide some indication of the scientific and medical excellence embodied in the society's early leadership (see Table II). This excellence has continued down through the years and space does not permit a fuller documentation of it. However, two of the society's subsequent presidents deserve special mention. They are Dr B. H. Kean of Cornell University Medical Center, whose recent autobiography has received widespread critical acclaim,[51] and Dr Harry Most of New York University Medical Center, whose pioneering malaria research has received international recognition.

LATER DEVELOPMENTS

When the society first began, it was an all-male group. Dr Louise Pearce was the first woman elected to membership at the society's meeting of January 18, 1935. She later served as vice-president in 1938–1939. It was not until January 16, 1948, that two additional women, Dr Florence Stone, Assistant Professor in the Department of Bacteriology at the Long Island College of Medicine, and Dr Kathleen L. Hussey, Research Associate in Parasitology at the Columbia University School of Public Health, were elected to membership. Others soon followed, including Dr Eva J. Weddigen of the Christian Medical Council for Overseas Work, and Dr Suzanne Howe, who were elected to membership later in 1948. The small number of women members during the early years paralleled their representation in the disciplines and institutions from which the society drew its support.

It was not until the 1960s that women began to regularly occupy officer positions. Dr Kathleen L. Hussey served as vice-president in 1964–1965, and Dr Eileen H. Pike in 1970–1971. Dr Pike, who was then Associate Professor of Parasitology at New York Medical College, later became the society's 35th president in 1971–1972. Since that time, two other women have served as president, Dr Ruth S. Nussenzweig, New York University Medical School, in 1981–1982, and Dr Jan S. Keithly, then of Cornell University Medical College, in 1984–1985.

Local medical societies which draw members from those in clinical practice tend to have low levels of membership turnover. This results from the infrequency with which those in private practice move, once they are established. However, those working in research and full-time academic settings tend to move on to other institutions with a fair degree of frequency. Thus the society, drawing as it does on these types of institutions for a significant proportion of its members, witnessed constant turnover, even in its early days. Such turnover became more frequent after World War II as inter-institutional moves were facilitated by newer opportunities at academic settings outside of the New York City metropolitan area. Despite this, a number of individuals have remained faithful members for several decades, and the society has consistently attracted newer members.

As with all societies whose aim is to serve as a forum for the exchange of scientific information, the society has had to compete with a wide range of local and national meetings. At any one medical center or research institute, there are now almost enough meetings scheduled to occupy most hours of the working day. Despite this competition, the society has continued to draw from 20 to 30 individuals to its meetings, a level that has been consistent for the past few decades.

As it enters the 1990s, the New York Society of Tropical Medicine counts among its members both internationally renowned senior researchers and practitioners and younger men and women in the early stages of their careers. They continue to share the society's aim, which is to provide a forum for the collegial exchange of scientific and clinical information about tropical medicine and related sciences.

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TABLE II. Officers of The New York Society of Tropical Medicine, 1933–1991

Year	President	Vice-President	Secretary-Treasurer
1933–34	Ralph W. Nauss	—	Thomas T. Mackie
1934–35	Wilbur A. Sawyer	Ralph W. Nauss	Thomas T. Mackie
1935–36	Charles S. Butler	Howard Fox	Thomas T. Mackie
1936–37	Alvin W. Schoenleber	Eugene L. Opie	Thomas T. Mackie
1937–38	Eugene L. Opie	Claude H. Lavinder	Thomas T. Mackie
1938–39	Wade W. Oliver	Louise Pearce	Thomas T. Mackie
1939–40	Howard Fox	Norman R. Stoll	Thomas T. Mackie
1940–41	Horace W. Stunkard	Edward M. Dodd	Thomas T. Mackie
1941–42	McIver Woody	Morton C. Kahn	Thomas T. Mackie
1942–43	McIver Woody	Wilson G. Smillie	Morton C. Kahn
1943–44	Wilson G. Smillie	Edward H. Hume	Morton C. Kahn
1944–45	Wilson G. Smillie	Henry E. Meleney	Morton C. Kahn
1945–46	Henry E. Meleney	Edward H. Hume	Morton C. Kahn
1946–47	Henry E. Meleney	Edward H. Hume	Morton C. Kahn
1947–48	Norman R. Stoll	Edward H. Hume	Irving Rappaport
1948–49	Victor G. Heiser	Zacharias T. Bercovitz	Irving Rappaport
1949–50	Harold W. Brown	Zacharias T. Bercovitz	Donald V. Moore
1950–51	Herald R. Cox	Zacharias T. Bercovitz	Donald V. Moore
1951–52	Zacharias T. Bercovitz	Kenneth R. Fourcher	Donald V. Moore
1952–53	Kenneth R. Fourcher	C. Zent Garber	Kathleen L. Hussey
1953–54	Thomas T. Mackie	C. Zent Garber	Kathleen L. Hussey
1954–55	Donald V. Moore	Howard B. Shookhoff	Kathleen L. Hussey
1955–56	Howard B. Shookhoff	Leslie A. Stauber	James R. McMillan
1956–57	Leslie A. Stauber	Kathleen L. Hussey	James R. McMillan
1957–58	C. Zent Garber	J. Horton Daniels	James R. McMillan
1958–59	Irving Rappaport	B. H. Kean	John M. Daley
1959–60	Edward I. Salisbury	Harry Most	John M. Daley
1960–61	Ashton C. Cuckler	Daniel M. Jobbins	John M. Daley
1961–62	Ashton C. Cuckler	Daniel M. Jobbins	John M. Daley
1962–63	B. H. Kean	Max M. Sterman	John M. Daley
1963–64	Harry Most	Frans C. Goble	John M. Daley
1964–65	Frans C. Goble	John M. Daley	William C. Campbell
1965–66	John M. Daly	Kathleen L. Hussey	William C. Campbell
1966–67	George L. Graham	Clifford L. Spingarn	William C. Campbell
1967–68	Clifford L. Spingarn	William C. Campbell	George J. Jackson
1968–69	John P. Craig	Anne C. Kimball	Eileen H. Pike
1969–70	Roger W. Williams	Morton H. Edelman	Eileen H. Pike
1970–71	William C. Campbell	Eileen H. Pike	J. Allen Waitz
1971–72	Eileen H. Pike	David N. Reifsynder	J. Allen Waitz
1972–73	George J. Jackson	Meir Yoeli	Howard L. Howes, Jr.
1973–74	John D. Frame	Charles W. Kim	Howard L. Howes, Jr.
1974–75	Meir Yoeli	William B. Foster	Herman Zaiman
1975–76	Howard L. Howes	Herman Zaiman	Thomas C. Cheng
1976–77	Michael Katz	Pascal James Imperato	Robert Herman
1977–78	Araxie Kilejian	Kermit G. Dwork	Robert Herman
1978–79	Kermit G. Dwork	Robert Herman	Vern L. K. Eveland
1979–80	Dickson Despommier	Thomas C. Jones	Vern L. K. Eveland
1980–81	Thomas C. Jones	Ruth S. Nussenzweig	Jerome Vanderberg
1981–82	Ruth S. Nussenzweig	Donald Hoskins	Jerome Vanderberg
1982–83	William Trager	Jerome Vanderberg	Jan S. Keithly
1983–84	Jerome Vanderberg	Jan S. Keithly	Michael Rosenbluth
1984–85	Jan S. Keithly	Peter Nicholas	Suzanne H. Giannini
1985–86	Charles Kim	Suzanne H. Giannini	Allen B. Clarkson
1986–87	Philip A. D'Alessandro	Murray Wittner	Allen B. Clarkson
1987–88	David R. Nalin		Jorge L. Benach
1988–89	Gary Wormser	Hannah Lustig Shear	Jorge L. Benach
1989–90	Pascal James Imperato	Jorge L. Benach	Haseeb A. Siddiqi
1990–91	Kevin M. Cahill	Cyrus J. Bacchi	Haseeb A. Siddiqi

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FROM THE LIBRARY

REPORT OF AN ITCHING DERMATITIS APPARENTLY DUE TO SCHISTOSOMA CERCARIAE

Beginning about the latter part of July, bathers at Interlaken Lake in the Town of Eastchester, Westchester County, New York, began to experience an itching eruption that at first was considered to be the result of insect bites. The lesions developed, shortly after bathing, as minute red macules or papules which increased in diameter, in some cases remaining more or less flattened and in others becoming slightly raised. In the more extensively involved cases the distribution was irregular but more or less general, except for the face and arms. Some of the lesions became quite red, appearing almost purpuric. The fastigium lasted about three days with more or less discomfort due to the itching, after which the eruption tended to subside during the next few days. The itching did not respond to the usual local applications. One patient, an adult woman with a very extensive involvement, was hospitalized for relief from the intense irritation. Among the children there was evidence of mechanical injury and mild secondary infection with crusting due to scratching. Two or three of the patients mentioned some edema about the eyes.

The lake in question is part of a multiple apartment development, housing about 1,500 people. Bathing is restricted to the tenants. Formerly a reservoir for the City of New Rochelle, Interlaken Lake, which is shaped like a V, is a little over a mile long and a little less than ¼ of a mile at the widest part. There is an artificial sandy beach about 100 feet long on the outer shore near the center of the V, with a dock extending out into deep water for the swimmers and a fenced-in pen for young children. A short distance from the dock on the side opposite the pen are some boats and the resting place of a flock of waterfowl. The waterfowl, which consist of some domestic white ducks and a few wild ducks, together with occasional wild geese, have been more numerous this year than formerly. As they are fed by the children, they tend to congregate near the same part of the shore in close proximity to the bathing area.

In view of the supposed absence of water itch in this part of the country it was at first thought that the itching eruption was probably due to insect bites. It was generally agreed, however, that it did not resemble mosquito bites nor did there appear, on casual inspection, to be any other insects present in sufficient numbers to account for the trouble.

In view of the inconclusive nature of the investigation, and having in mind the possibility of a causal connection with the water fowl, some snails obtained in the area of the bathing beach were submitted for examination to the Department of Preventive Medicine, New York University, College of Medicine. Dr. Donald V. Moore, who conducted the investigation in the laboratory, discovered cercariae in one of the snails which, when placed on his wrist, produced a typical water itch eruption. Two of his associates also developed a dermatitis. The snails, which were sent by Dr. Moore to the Museum of Comparative Zoology at Harvard University, were identified by Dr. William J. Clench, curator of mollusks, as Physa heterostropha Say. Dr. Moore was unable to find cercariae in a pail of the lake water obtained at the bathing beach. However, one of the white ducks that was sent to him for examination was found to harbor schistosomes which proved to be Trichobilharzia physellae.

It appears probable from the foregoing that the bathers at Interlaken Lake were affected by schistosome dermatitis. This in turn evidently resulted from the presence of some infected waterfowl that have been visiting the lake as well as other collections of water in lower Westchester County in increasing numbers in recent years. It is evident that this condition, formerly associated primarily with the Great Lakes region, is now present in this part of the country. With the increasing influx of waterfowl it is probable that water itch will be encountered with greater frequency hereabouts in the future.

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EDWARD A. LANG, MD
(*NY State J Med* 1947; 47:2458)

Lofgren's syndrome (acute sarcoidosis) sine erythema nodosum mimicking acute rheumatoid arthritis

LEE D. KAUFMAN, MD

The triad of bilateral hilar lymphadenopathy, erythema nodosum (EN), and polyarthralgias-arthritis has been linked to Lofgren since he initially described it in 1952.[1] Nearly half of sufferers from this syndrome have acute sarcoidosis as the cause. However, among patients with acute sarcoid arthritis, the prevalence of EN varies widely.[1,2] In Scandinavia, about two thirds of patients with acute or subacute sarcoid arthritis have associated EN, while in France EN is reported as being unusual.[2]

Rheumatoid arthritis is a common disorder of unknown cause, which is diagnosed on the basis of a constellation of clinical signs and symptoms. These are descriptive criteria and may be found in many different arthropathies considered in the differential diagnosis of rheumatoid arthritis.[3]

This report describes a patient with acute sarcoidosis who presented with a clinical syndrome identical to early seropositive, polyarticular rheumatoid arthritis. This patient had acute sarcoid arthritis in the absence of EN. The articular manifestations and often self-limited course of that process are features that distinguish it from acute rheumatoid arthritis.

CASE REPORT

A 25-year-old woman presented to the rheumatology clinic of the University Hospital at Stony Brook, NY, in February 1989, with a four-week history of polyarthralgias involving the knees, ankles, elbows, wrists, and proximal interphalangeal joints of the hands bilaterally. Morning stiffness occurred for one hour daily. Articular swelling was noted, predominantly in the ankles. There was no history of fever, prodromal illness prior to the onset of articular complaints, dyspnea, cough, skin rash, Raynaud's phenomenon, oral mucosal lesions, colitis, or symptoms suggesting inflammatory eye disease. She had no previous history of joint swelling or pain. There were no other medical problems.

On physical examination, there was no adenopathy, and the heart, lungs, abdomen, and extremities were normal. Lacrimal gland enlargement was detected. Moderate synovitis was noted in both ankles and was associated with subtalar tenderness. Patellofemoral tenderness was also present and was symmetric, with a positive "bulge" sign.

Laboratory studies revealed a white blood cell count of 8,300/mm^3 with a normal differential count; hemoglobin, 12.4 g/dL; erythrocyte sedimentation rate (ESR), 83 mm/h; rheumatoid factor, 1/160; negative antinuclear antibody; nonreactive rapid plasma reagin (RPR) test for syphilis; normal CH-50; and negative serologic tests for Lyme borreliosis.

The prominent ankle swelling, even in the absence of EN, was suggestive of acute sarcoidosis. A chest film subsequently revealed bilateral hilar adenopathy (Fig 1). The angiotensin converting enzyme level was elevated to 86.3 U/L (normal range, 10–50). A gallium scan demonstrated prominent uptake in the hilar nodes, salivary glands, and ankles bilaterally (Figs 2, 3).

The patient was treated with ibuprofen, and over the following month her condition significantly improved. On follow-up visits in March and May 1989, there was progressive improvement and she was able to discontinue the anti-inflammatory medication. A repeat ESR was 23 mm/hr, and the physi-

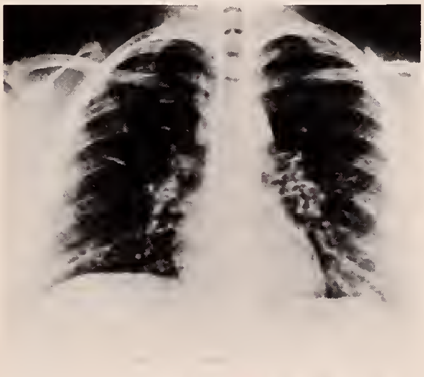

FIGURE 1. Prominent bilateral hilar adenopathy.

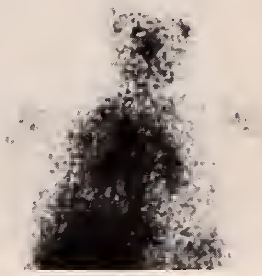

FIGURE 2. Gallium (^{67}Ga) scintigraphic study demonstrating uptake in the parotid glands and hilar lymph nodes symmetrically.

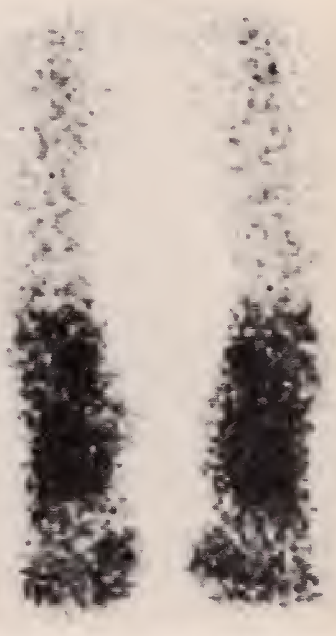

FIGURE 3. Gallium (^{67}Ga) scintigraphic study showing prominent uptake in the ankles bilaterally.

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cal examination demonstrated full range of motion, no articular tenderness, and resolution of the ankle synovitis.

DISCUSSION

In the years between 1942 and 1945, Lofgren studied 212 patients with the bilateral hilar adenopathy syndrome.[1] The majority of these patients were female, and the prevalence of EN among women was 66%, as opposed to 12% among men. Almost half of these individuals were thought to have sarcoidosis. This presentation of the disease has subsequently been noted to have an excellent prognosis for spontaneous recovery in most series.[4] In this stage of disease, however, one must also consider that infection with tuberculosis, coccidioidomycosis, histoplasmosis, and North American blastomycosis may produce the triad of EN, hilar adenopathy, and peripheral arthritis.[5]

Arthritis occurs with a prevalence of 11–39% in patients with sarcoidosis and can present as three variants. The most common is illustrated in the patient described in this report, and includes migratory or symmetric polyarthritis, with or without EN, and hilar adenopathy. Some of these patients may also have prominent tenosynovitis with periarticular swelling or tenderness. Polyarticular arthritis is usually appendicular, with involvement of the small and large joints of the upper and lower extremities. In decreasing order, the ankle, knee, wrist, and proximal interphalangeal joints are most frequently affected. Axial disease is unusual but can occur. Most acute attacks resolve over a six-week period. A second variant includes patients with recurrent polyarthritis or, rarely, monoarticular disease. Finally, in the presence of advanced parenchymal lung disease associated with cutaneous lesions and visceral involvement, chronic deforming arthritis may develop. In all stages, sarcoid joint disease may resemble rheumatoid arthritis. In children, articular disease in sarcoidosis is more often associated with skin and ocular involvement than in patients without arthritis.[6] Such an association has not been statistically confirmed in adults.

Synovial effusions are more common with chronic arthritis and are usually type II (inflammatory) in nature. Granulomas can be identified in synovial biopsies; however, these are usually found with chronic arthropathy and have only rarely been reported with the acute syndrome. Radiographic changes are generally not seen in the acute form. Subchondral cysts, punched out lesions, and a "lace-like" trabecular pattern characterize the radiographic appearance of chronic deforming arthritis.[7-9]

Spilberg et al[7] described 28 patients with sarcoid arthropathy. EN was seen in seven (25%) and was always associated with hilar adenopathy. Fifteen patients experienced a single attack, five had two attacks, and eight sustained multiple recurrences. Of the latter, persistent chronic arthritis developed in four patients. The authors found no difference in the pattern of arthritis among patients with or without EN. As in Lofgren's patients, EN occurred more often in women than in men. Caplan et al[10] described a group of 19 patients with bilateral hilar adenopathy and polyarthritis without EN. Based on clinical findings, these patients were otherwise similar to those with skin lesions, and 73% had biopsy evidence for sarcoidosis.

A recent study by Pennec et al[2] in France compared the manifestations of acute sarcoid arthritis in 11 patients with and five patients without EN. No difference was noted with regard to the joints involved, ESR, or disease duration. Although not statistically different, there was a trend toward fewer joints affected per patient in individuals with EN.

A positive rheumatoid factor is seen in approximately 6–10% of patients with sarcoid arthritis and was present in this patient.[5] Random muscle biopsies demonstrate noncaseating granulomas in 50–60% of patients with sarcoid. This has been reported to be as high as 89% in patients with EN and arthralgias.[11]

In summary, acute sarcoid arthritis is almost always accompanied by hilar adenopathy and is most frequently polyarticular and symmetrical. The presence of rheumatoid factor in some patients may be misleading; however, prominent ankle involvement and tenosynovitis (periarthritis) should suggest sarcoidosis. Furthermore, a diagnosis of rheumatoid arthritis should not be made until synovitis has been present for at least six weeks. Using that duration of disease activity, the 1987 revised criteria will correctly classify most patients with rheumatoid arthritis (specificity of 89.3%) and exclude individuals with other disorders.[3] An elevated angiotensin converting enzyme level and a positive gallium scan are also useful in confirming the diagnosis of sarcoidosis.[12] The patient described in this report illustrates the importance of considering acute sarcoid arthritis in the absence of erythema nodosum.

Acknowledgment. The author thanks Ms Maureen Veprek for her secretarial expertise.

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Extraparotid Warthin's tumor: An unexpected finding in three female patients

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In 1895, Hildebrand[1] described a tumor with epithelial and lymphoid elements, which he considered to be one variant of the congenital cysts that arise in the lateral aspect of the neck. It was not until 1929 that this tumor was first reported in the American medical literature by Alfred Scott Warthin.[2] Since that time, this peculiar tumor has been described by many authors. The most commonly used names for it have been adenolymphoma, oncocytoma, papillary cystadenoma lymphomatosum, and Warthin's tumor.[3]

Warthin's tumor is a benign tumor that primarily involves the parotid gland. Its incidence has been reported to vary from a low of 2%[3] to a high of 24%[4] of all parotid tumors. However, the accepted incidence rate is 6–10%.[5] These tumors most commonly arise in men in their sixth and seventh decades of life. The tumors have been reported to be bilateral in up to 13.4% of cases[6] and multicentric in up to 12%.[7]

Extraparotid Warthin's tumors have been reported in the submandibular gland,[8-10] in cervical lymph nodes,[7,11-15] and in the minor salivary glands of the oral cavity[16-18] and the oropharynx.[19] The incidence of extraparotid Warthin's tumors has been reported to be 8% of all Warthin's tumors.[7,12] We report three cases of extraparotid Warthin's tumor and review the histogenesis and treatment of these neoplasms.

CASE REPORTS

Case 1. An 83-year-old woman with a history of an endometrial adenocarcinoma treated in 1960 and an infiltrating duct cell carcinoma of the breast treated in 1986 presented with a progressively enlarging mass over a six-month period in the left upper neck. The patient underwent direct laryngoscopy, nasopharyngoscopy, and excisional biopsy of a 2-cm mass in an upper jugular lymph node. The pathologic diagnosis was Warthin's tumor in a cervical lymph node. Salivary duct inclusions were present in the lymph node. The patient has had an 18-month follow-up with no evidence of recurrence.

Case 2. A 74-year-old woman with a history of arterial hypertension noted a 3-cm painless mass in her upper neck that developed over a period of six weeks. Direct laryngoscopy, nasopharyngoscopy, and excisional biopsy of the mass were performed. The pathologic diagnosis was Warthin's tumor in an upper jugular lymph node which also contained salivary duct inclusions. The patient was lost to follow-up four months after excision of the tumor, at which time she had no evidence of recurrence.

Case 3. A 68-year-old woman with a 40 pack/year history of cigarette smoking was admitted with a clinical T1N2cM0,[20] well differentiated squamous cell carcinoma of the anterior floor of the mouth. She underwent resection of the primary tumor and bilateral supraomohyoid neck dissections. Pathologic examination of the specimen revealed chronic sialadenitis of the left submandibular gland and Warthin's tumor in two lymph nodes adjacent to the right submandibular gland. As in the two previous cases, salivary duct inclusions were also present.

DISCUSSION

Warthin's tumors are usually soft, well-encapsulated neoplasms with a smooth or lobulated external surface. On sectioning, multiple irregular cystic spaces with or without papillary projections may be seen. These cysts contain fluid which is usually mucoid and brown colored.

The histologic diagnosis of a papillary cystadenoma lymphomatosum requires the presence of both a lymphoid component and an epithelial component (oncocytes). The epithelial component consists of papillary cystic structures or tubular structures lined by two layers of granular eosinophilic cells. The inner or luminal layer consists of columnar cells, the nuclei of which are aligned toward the luminal surface. Squamous metaplasia of the epithelium is occasionally seen. The lumina contain eosinophilic material which, at times, resembles colloid. Corpora amylacea-like bodies may be seen (Fig 1). The lymphoid tissue contains sinuses, follicles, and a connective tissue capsule (Fig 2). The lymphoid tissue is composed predominantly of B-cell lymphocytes, as shown by Cossman et al,[21] who postulated that these B lymphocytes were residual lymph node elements that were reacting against the epithelium. However, Diamond and Braylan[22] showed that T-cell lymphocytes predominated over B-cell lymphocytes in Warthin's tumor. This supports the concept that Warthin's tumor origi-

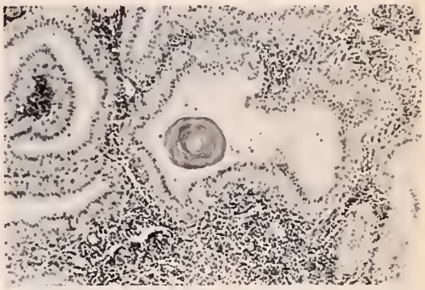
FIGURE 1. Corpora amylacea-like bodies within tubular structure of Warthin's tumor (original magnification × 240).

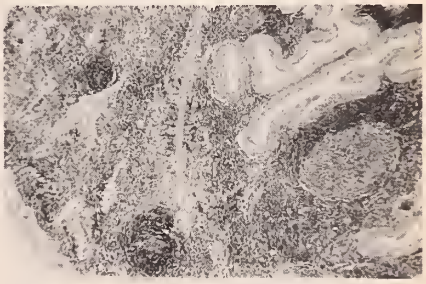
FIGURE 2. Lymph node showing marginal sinuses, cortex with reactive germinal centers, and Warthin's tumor (original magnification × 80).

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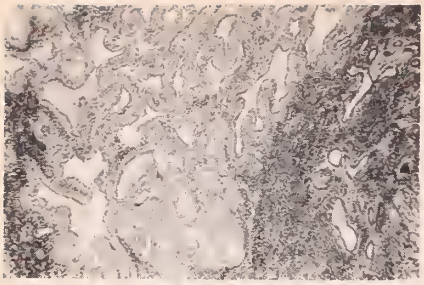

FIGURE 3. Lymph node with salivary duct inclusion and Warthin's tumor (original magnification × 80).

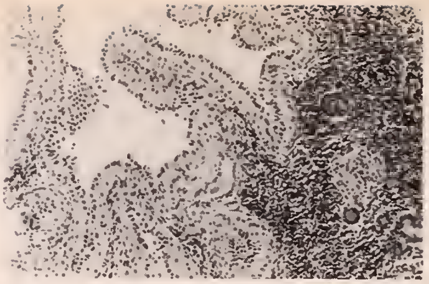

FIGURE 4. High power view of epithelial and lymphoid elements of Warthin's tumor with adjacent salivary gland ductal elements (original magnification × 240).

nates from preexisting lymph nodes.

The most widely accepted theory concerning the histogenesis of Warthin's tumor is the heterotopic theory, which states that these neoplasms originate from neoplastic proliferation of salivary gland ducts present within intra or para-parotid lymph nodes.[5,23] Another theory, proposed by Allegra[24] in 1971, suggested that Warthin's tumor was not a neoplasm, but a delayed hypersensitivity reaction. As compared to the submandibular and sublingual salivary glands, the late encapsulation of the parotid gland during embryogenesis accounts for the fact that lymph nodes containing salivary tissue may be found both within and outside of the parotid gland.[15] This explains the presence of ectopic salivary gland tissue in extraparotid lymph nodes. In the three cases described here, salivary duct elements were found adjacent to the Warthin's tumor (Figs 3, 4).

When a papillary cystadenoma lymphomatosum is diagnosed in the preoperative period, the treatment of the patient can be individualized. However, if no definite preoperative diagnosis is entertained, the treatment must proceed as with any other parotid gland neoplasm—ie, lateral or total parotidectomy depending on the location of the tumor.

The male to female ratio of Warthin's tumors has been reported as ranging from a low of 1.5:1[25] to a high of 10:1.[6] At Roswell Park Cancer Institute, over a 24-year period, the male to female ratio in 33 patients with parotid Warthin's tumors was 15.5:1. It is interesting to note that the three patients reported here with extraparotid Warthin's tumors were female.

Usually, extraparotid Warthin's tumors are diagnosed incidentally after pathologic examination of the resected specimen. In their series of 14 patients with extraparotid Warthin's tumors, Snyderman et al[12] reported that six patients (46%) had their neoplasms diagnosed on pathologic examination of the specimen after resections for other primary tumors. They also reported a 23% (three patients) incidence of multifocal tumors.[12] One of the patients presented here had multifocal extraparotid Warthin's tumor.

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Superior vena cava syndrome and right pleural effusion due to giant goiter

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Superior vena cava syndrome is a well-known complication of various malignancies including lymphomas, lung carcinomas, thyroid malignancies, and metastatic breast carcinoma. There are also numerous nonmalignant causes, including aneurysms of the thoracic aorta, teratomas, thymomas, mediastinitis secondary to granulomatous disease (histoplasmosis, tuberculosis), and pericarditis. Rarely, benign intrathoracic goiters cause superior vena cava obstruction. We describe a patient with superior vena cava syndrome secondary to giant intrathoracic colloid goiter, in whom chest roentgenography revealed an opacified right hemithorax due to the presence of pleural effusion and the thyroid lesion.

CASE REPORT

A 53-year-old woman was referred for evaluation of increasing shortness of breath and worsening jugular venous distension. The patient had a history of hyperthyroid goiter, which was initially treated with methimazole. She had noted a two-week history of nonproductive cough, but denied dysphagia, fever, night sweats, wheezing, hoarseness, and sore throat.

The patient was afebrile on admission. The physical examination was remarkable for a diffusely enlarged thyroid. The left lobe was larger than the right, was smooth in texture, and had no palpable nodules. No bruits were noted, and stridor was absent. The jugular veins were bilaterally distended to the mandibular angle, and there was no apparent facial edema. Decreased breath sounds were present throughout the entire right lung field, with percussion dullness over the lower three quarters and egophony throughout the entire right lung field. Venous distension was noted over the anterior chest wall. The cardiac examination was unremarkable, and pulsus paradoxus was absent.

The white blood cell count was 5,900/mm^3 with normal differential. Arterial blood gases (room air) were as follows: pH, 7.44; PACO_2, 52 torr; PAO_2, 64 torr. Thyroid function tests were within normal limits.

The chest roentgenogram (Fig 1) revealed diffuse opacification of the right lung field, tracheal narrowing at the level of the clavicles, and leftward mediastinal shift. Right lateral decubitus films revealed a free-flowing pleural effusion.

Right thoracentesis yielded sanguinous, exudative fluid with total protein of 3.2 g/dL; pH, 7.48; white blood cell count, 5,000 with 90% small lymphocytes; red blood cell count, 15,000; and pleural fluid lactate dehydrogenase (LDH) level, 138 (serum LDH, 203; pleural fluid to serum LDH ratio, 0.68). Bacterial cultures were negative, and no malignant cells were found on cytologic examination.

A contrast enhanced computerized tomographic (CT) scan (Fig 2) of the chest revealed a large, well-encapsulated mass involving almost the entire right hemithorax. The mass extended laterally and was in close proximity to the pleura. It was not possible to discern whether the mass originated from the mediastinum or the lung. There was a small area of normal-appearing lung anterior and inferior to the mass.

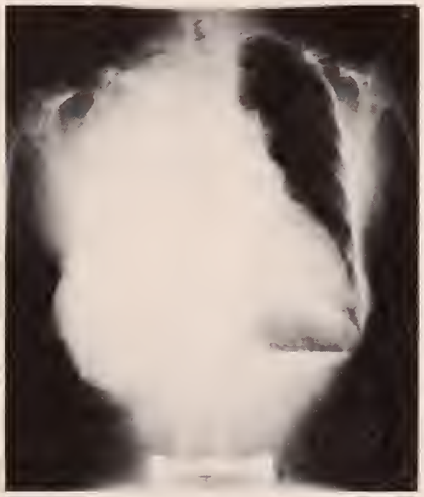

FIGURE 1. Chest film showing opacification of right hemithorax with mediastinal shift to the left.

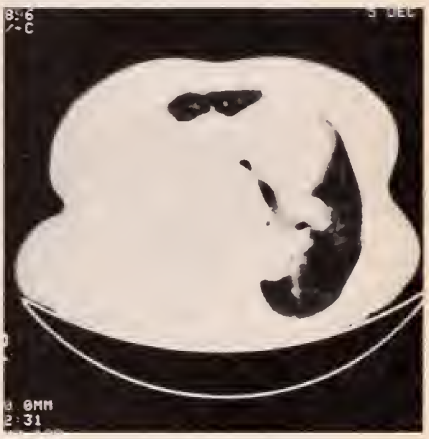

FIGURE 2. CT of the chest demonstrating a mass in the right hemithorax with a small area of aerated lung. Note also a right pleural effusion.

Fluoroscopically guided transthoracic fine needle aspiration was performed via the lateral and anterior approaches. Both aspirates yielded sanguinous fluid. Needle biopsy (Abrams) of the pleura was performed at the right eighth intercostal space, and adequate tissue was obtained. Pathologic examination revealed normal thyroid tissue, indicative of the diagosis of intrathoracic colloid goiter. Subsequent re-examination of both needle aspirate samples revealed normal thyroid tissue.

The patient underwent right thoracotomy, and a 1,500-g intrathoracic colloid goiter was removed. At the time of surgery, the goiter was noted to extend posterior to the great vessels and was also adherent to the pleura of the right lung. This accounted for the diagnosis having been made from the pleural biopsy. The patient's right lung was encapsulated in fibrotic tissue and re-expanded to near normal after decortication.

DISCUSSION

Transudative pleural effusions result from an imbalance or disruption in the hydrostatic-oncotic forces that control movement of fluid across pleural membranes. The superior vena cava syn-

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drome is a well known though rare cause of transudative right pleural effusions, which develop because the superior vena cava obstruction leads to increases in systemic venous pressure and consequent increase in filtration from the parietal pleural capillaries.[1] Obstruction of the superior vena cava is only rarely associated with benign disease processes. In a review from the Mayo Clinic,[2] only 22% of the cases were due to benign causes. Similarly, the reported experience from the Cleveland Clinic[3] totalled only 16 patients with superior vena cava syndrome due to benign causes, 75% of which resulted from mediastinal granuloma, and only two of which resulted from retrosternal thyroid.

In many series, malignant lesions are reported to be the underlying cause in 85–90% of the cases of superior vena cava syndrome.[4,5] In the current report, we document superior vena cava syndrome with an exudative right pleural effusion caused by a giant colloid goiter. The exudative nature of the pleural effusion was most likely due to the inflammatory/fibrotic pleural reaction to ingrowth of the goiter. Benign intrathoracic goiter in association with the superior vena cava syndrome has only occasionally been described. Transient acute superior vena cava obstruction occurred in one patient with substernal goiter treated for hyperthyroidism with propylthiouracil.[6] The patient presented here had previously received antithyroid therapy, but symptoms of superior vena cava obstruction were not described at that time. Other reports of superior vena cava syndrome due to intrathoracic goiter have been infrequent,[7–9] and the presence of exudative pleural effusion appears to be a new finding. The mechanism of arterial hypercapnia in this patient may be related to the combined effects of extrinsic upper airway compression (note tracheal narrowing in Fig 1) and the reduction in gas exchanging surface area from mass effect and effusion in the right hemithorax. This combined etiology is most likely since hypercapnia is not seen in previously normal subjects until the upper airway diameter constricts to below 5 mm,[10] which is more severe than the narrowing observed in this patient.

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Make way for tomorrow

TO THE EDITOR: The buzz words continue to toss around on the turbulent sea of health care. Health care tradeoffs, medical industrial complex, quality, access, and cost are but a few of the terms. Unions are watching their hard-won health care benefits disappear in a bewildering whirlpool of give-backs. Physicians are constantly being pressured to do more with less.

The question that is being whispered about is, "What if these cost containment strategies don't work?" The murmurs are getting louder that it will be the elderly who will be asked to make sacrifices. In a sense, they will be encouraged to "make way for tomorrow," to step aside and let the benefits of technology be enjoyed by the younger generation. The popular age to start custodial care appears to be 80. The emphasis seems to be that children should not be made to continue sacrificing for their parents. Ignored in this presentation are the sacrifices that the elders have made for their children and the over-utilization of resources that all segments of the society enjoy. The elderly will thus be asked to accept the realities of rationing unless they can pay for high-tech procedures and treatment. Attention will be drawn to the largesse that they enjoy, Medicare. The fact that they made substantial contributions to Social Security and Medicare and that the "run on the Social Security bank" was partly fueled by payouts to unemployment insurance will be forgotten.

Both the seniors and physicians suffer from the misguided efforts to contain costs at the expense of caring. It will not be easy but these two camps with common interests will have to learn to work together if we hope to emerge intact from bureaucratic encounters. There will, of course, be times when we disagree but these differences must not be misconstrued as stark ideological positions.

We have already discovered that well meaning regulations which become punitive toward physicians only serve to shatter the relationship between physicians and seniors. The result is further isolation and mistrust. If we are to achieve meaningful legislation, the architects of this endeavor should consider convening a consensus development panel with pertinent participants from all levels of involvement before a program is designed. It is unproductive to have regulatory agencies develop a plan and then ask physicians and their representatives to rubber stamp it. The agencies need to seek this input at the beginning of the process and not at the end. Physicians have to be stake holders in health care planning, not obedient servants. The policies formulated would then more accurately reflect the concerns of all rather than document that which often satisfies none of the constituencies. This will not be a speedy process, but it might well avoid some of the chaos that currently occurs.

In our talks with seniors, we must continue to stress that we are just as much victims of the system as they are. We must strive to get beyond their panic and fear and seek a dialogue that considers the realities and the opportunities. Together we can fashion a collaborative relationship which seeks equity and fairness for our seniors and ourselves.

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The use of free phenytoin levels in averting phenytoin toxicity

TO THE EDITOR: A paradoxical increase in seizure frequency[1] is a dose-related neurotoxicity effect of phenytoin and is generally reversible with decreased dosage. There is little information on the use of free phenytoin levels to better monitor serum drug concentrations.

Case Report. A 29-year-old woman with Type I diabetes mellitus and longstanding epilepsy (and no history of status epilepticus) relatively well controlled with phenytoin, 300 mg/day, and phenobarbital, 90 mg/day, presented to the emergency room after suffering a seizure that abated spontaneously.

Laboratory studies revealed a phenobarbital level of 22 μg/mL (therapeutic range 15–40 μg/mL) and a phenytoin level of 18.5 μg/mL (therapeutic range, 10–20 μg/mL). The glucose level was 175 mg/dL, nonketotic. Magnesium and corrected calcium levels were normal. The cause of the seizure was undetermined and the patient was admitted to the hospital for observation without any modification in her anticonvulsant regimen.

The next day, the patient underwent a seizure characterized by unresponsiveness and sustained leftward gaze and nystagmus. The glucose level was 38 mg/dL. The seizure was aborted with the administration of dextrose and phenytoin, 500 mg intravenously. Later that day, the patient went into status epilepticus. She received more than 1 g of intravenous phenobarbital and required mechanical ventilation. The phenytoin level at this time was 22 μg/mL and after the status episode, it was 29 μg/mL; the free phenytoin level was 4.5 μg/mL (therapeutic range, 1–2 μg/mL); and the cerebrospinal fluid (CSF) phenytoin level was 4.2 μg/mL. CSF studies were otherwise normal. The patient underwent several more seizures and required boluses of intravenous phenobarbital. An interictal electroencephalogram showed diffuse slowing without epileptiform activity.

A possible relationship between this patient's seizure and her phenytoin therapy was not initially suspected since the first total phenytoin level was within the accepted therapeutic range. Later, when the seizures recurred and proved refractory to aggressive treatment with phenytoin and phenobarbital, despite correction of the hypoglycemia, an underlying factor was suspected. Based on the subsequent finding of hypoalbuminemia (2.5 g/dL) and the known binding of phenytoin to plasma proteins, phenytoin toxicity was implicated as the etiology of the intractable seizures and phenytoin was thus discontinued. As serum phenytoin levels fell, the patient's seizure frequency decreased. Ultimately her seizures were controlled on phenobarbital at 120 mg/day orally, with serum levels of 30–40 μg/mL.

Discussion. In healthy patients with epilepsy receiving phenytoin alone, the binding of phenytoin to plasma protein is about 90% and the free (unbound) pharmacologically active fraction (about 10%) is nearly constant.[2] Free phenytoin levels of 1.0–2.0 μg/mL are generally equivalent to the accepted therapeutic range of total phenytoin of 10–20 μg/mL. Interestingly, CSF levels of phenytoin approximate the free plasma levels.[3]

Impairment of protein binding to phenytoin may occur in neonatal or elderly patients; during the late phase of pregnancy; in patients with hyperbilirubinemia, hepatic disease, uremia, nephrotic syndrome, burns, surgery, malnutrition, or other conditions associated with hypoalbuminemia; as well as during combination therapy with valproic acid, aspirin, sulfonamides, and tolbutamide, by displacing phenytoin from its albumin binding sites.[4] The unbound fraction of phenytoin also increases with increasing temperature.[2] In these clinical settings of impaired protein binding, the total plasma concentration of phenytoin may result in underestimation of the free fraction and inadvertent phenytoin toxicity. Thus measurement of the free phenytoin fraction is the preferred index of available anticonvulsant activity. Free phenytoin levels ≤ 2.1 μg/mL typically do not lead to phenytoin-induced neurotoxicity.[5]

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Capgras' syndrome

TO THE EDITOR: In 1923 Capgras and Reboul-Lachaux reported the case of a 53-year-old woman suffering from the delusion that persons significant to her had been replaced by imposters.[1] This basic feature is involved in the present case report. Soon after the appearance of the report by Capgras and Reboul-Lachaux, publications used the term Capgras' syndrome to describe this entity. The vast majority of reports have been of single cases appearing in the British and French psychiatric literature.

There are several variants of the syndrome. One type, for example, is autoscopic. Patients see doubles of themselves in persons or objects nearby.[2] Atwal and Khan described other variants.[3] The syndrome has often been associated with schizophrenia and affective psychoses, and has also been described in patients with varying types and degrees of brain pathology. Lewis' report involved a 19-year-old girl in whom magnetic resonance imaging revealed bilateral subcortical lesions in the occipitotemporal and frontal regions.[4]

Case Report. A 55-year-old woman was visited by her sister from another state, who was obliged to return home after several hours instead of remaining a week as planned. The two sisters had been viewing photos of siblings, and took two drinks of whiskey. Soon thereafter the patient suddenly became verbally abusive, kicked her apartment door in anger, and shouted repeatedly that her sister was an imposter. She ran to a neighbor who heard the commotion and the patient yelling that the person with her was a stranger, not her sister. She asked the neighbor to get rid of this stranger, and then went to sleep upon the advice of the neighbor. She awoke at 3:00 AM after the sister was gone. The patient had amnesia for the entire episode. The neighbor confirmed the events as revealed by the sister who telephoned later that morning, telling the patient that she had behaved in a bizarre fashion. She said she had been frightened and feared for her own life. The patient, however, resented what she felt was abandonment, claiming she would have remained to help had their roles been reversed. At her psychiatric session the next day, the patient felt embarrassed, angry about the abandonment, and believed people on the street looked at her as if she were crazy. The paranoid reaction disappeared in two days. She had had similar paranoid feelings in the past when under great stress.

The patient believed that the misidentification was related to childhood suspicions of having been adopted. One of several brothers and sisters, older and younger, she differed temperamentally from them. She felt they were superficial, materialistic, and favored by both parents with gifts and financial assistance whereas she profited by neither. She left home at age 16 to educate herself, never forgiving her father for allegedly unwarranted beatings she received in contrast to better treatment of the other children. She asked older brothers and sisters more than once whether she had been adopted but met with denials. Her past history and suspicions meshed with the imposter delusion.

The patient recalled a similar event ten years earlier when she was involved in a turbulent marriage. After having some alcoholic drinks which apparently triggered her delusion, she became abusive and insisted her husband was an imposter resembling her husband. He tried to reassure her, and telephoned the psychiatrist in distress. The patient's delusion rapidly disappeared after sleep. It apparently involved an attempt to deny the validity of her marriage which eventually ended in divorce. In the more recent episode, the delusion was an attempt to deny the existence of valid filial relationships owing to persistence of long-standing unresolved grievances.

Discussion. Koritar and Steiner,[5] noting that there are differences of opinion regarding the etiological role of organic versus psychological factors in Capgras' syndrome, attempted a synthesis of various viewpoints and presented two patients diagnosed as paranoid schizophrenics in whom general physical and neurological examinations revealed normal findings. Pelletier, Bartolucci, and Wallace[6] regarded the syndrome as a nonspecific symptom seen in both functional and organic conditions. Lewis' report of the 19-year-old patient with brain lesions, mentioned above, noted that a diagnosis of "schizophreniform psychosis" was also made during a six-month follow-up.[4]

A combination of psychopathological and neuropathological etiologies may be present. An either-or approach seems unnecessary. The patient reported here was apparently not schizophrenic and had no affective psychosis. Her work-related annual physical examinations for the past 25 years had revealed normal findings. She was in treatment mainly because of problems involving interpersonal relations. Of interest is that her two demonstrations of Capgras' syndrome were triggered by alcohol intake, which apparently released inhibitions resulting in an upsurge of long-standing grievances. Thus the chemical effect on brain function must be taken into consideration. Of additional interest is that the psychodynamics could be somewhat explicated. The findings are consistent with the "ambivalence theory." Fishbain and Schiffman[7] claimed that this is the most widely accepted psychodynamic hypothesis in the evaluation of the Capgras delusion. The patient harbors strongly ambivalent feelings toward a person significant to him which are not adequately repressed, and the anxiety resulting from this conflict induces "a psychotic solution based on denial and displacement."

Psychotherapy for patients with Capgras' syndrome deals with psychopathological functioning in general. It may be combined with pharmacotherapy when necessary. The Capgras delusion is not dealt with in isolation and disappears with general improvement. There may be relapses under special circumstances as in the patient described here. The prognosis is favorable so long as overall improvement is maintained.

This report of a patient with Capgras' syndrome supplements the relatively few reports which have been published in this country in comparison to those in the British and French medical literature.

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FROM THE LIBRARY

UNTOWARD EFFECTS OF THE NEWER DRUGS

Prior to the current era of medical science the physician was largely dependent upon naturally occurring botanicals and minerals for his dispensable therapeutic agents. Bromides were employed for convulsions, colchicum for gouty arthritis, quinine for malaria and other fevers, iron salts for anemia, iodine for thyroid disorders, foxglove for heart disease, opium for pain, mercury for syphilis, ergot for contraction of the uterus, caffein for diuresis, and a variety of herbs and minerals for purgation. Each therapeutic agent was effective in selected instances; sometimes the efficiency was high, at other times it was low. Most of these preparations are still in use and continue to accomplish great therapeutic good. On the other hand, and without exception, new and improved drugs, which have come largely from the chemical laboratory, have been introduced either to enhance or to replace each of the above-noted agents. No well-informed physician will deny the value of these newer drugs. Some small loss has accompanied a tremendous gain, however, and the untoward reactions of certain of the preparations in a percentage of the patients receiving accepted therapeutic amounts has led to serious or even fatal results. . . .

In most instances the side-effects are inconvenient and not serious and subside with cessation of the use of the drug. On the other hand, irreparable and mortal changes have followed the use of some of the substances in an occasional instance. Prophylactic and precautionary measures should be observed with each drug that has been shown to be potentially harmful. Careful watch should be maintained in each patient even though there is no visible evidence of toxicity. To know when to stop a drug is frequently as important as to know when to prescribe it. Finally, it is the duty of every physician who is prescribing either new or established preparations to observe carefully untoward reactions and to report them either to the manufacturer or in a medical publication. As the number of new drugs increases, and we know that this will be a fact, continued vigilance should be practiced. . . .

JOHN H. TALBOTT, MD
(*NY State J Med* 1948; 48:280–286)

Country doctor

House Calls: Memoirs of a Country Doctor. By Marvin Brown, MD. 214 pp. Buffalo, NY, Prometheus Books, 1988. $19.95 (clothcover)

There is an apocryphal story circulating through the halls of the modern medical school teaching hospital that goes something like this: The resident physician admitted the patient last night and now, on attending rounds, presents the "case" in classic, complete fashion. After listening attentively, from the introductory "this is a 47-year-old white male who presents to University Hospital with a . . . " to the closing "and our diagnosis and plans are . . . ," the attending physician turns to the resident and says, "Now tell me, what does this patient do for a living, where does he live, and what is his community like?" The resident says, somewhat sheepishly, "I don't know." The attending physician says, "Go back and find out. What this man needs is a doctor!"

What that resident needs is to read Marvin Brown's *House Calls: Memoirs of a Country Doctor*. Dr Brown, a family practitioner in upstate New York since 1938, has written a personal and moving portrait of his love affair with profession and family that serves as a poignant reminder to us of the joys of being a personal physician, a family doctor to his community. Especially timely in this period of great unrest, discontent, and uncertainty in the medical profession, the public, and third-party payers, this simple, straightforward, and eloquent memoir reminds us of how good we can be if we just remember "one central fact . . . patients will always need care, and physicians will always be there to provide it." The author uses these 214 pages to provide a blueprint of how today's physicians might best accomplish this care for the patient.

This memoir is not just for primary care practitioners; it speaks to a wider audience, both lay and medical, with clarity and simplicity. His medical career spanning the end of the Great Depression to the 1980s, Marvin Brown writes as a witness to the wonders of the antibiotic era as he treats cellulitis, pneumococcal pneumonia, and puerperal fever with sulfa and penicillin. He describes home-delivering three babies in one long day and making house calls by car, sleigh, and snowshoes in the bitter winter of 1947–1948. He speaks of "weather" as being an important rate-limiting step in his practice of rural medicine.

Dr Brown should help both a wider medical audience and the public at large to understand what family doctors are and why they are still so important. His memoir is replete with phrases like "knowing the limits of my expertise" and "the patient is first" and "the family doctor is not treating an ailment but a whole person." Marvin Brown means what he says and he lived, professionally, what he preaches. House calls were as matter-of-fact for him as CT scans are for headaches today. In the January 1943 influenza epidemic he made more than 50 house calls in one day. He realizes the time he could not spend with his family was a loss (wonderfully told as a tribute to his dear wife, "Bee"), but that this was a course agreed upon by both husband and wife as they first set out in 1938 to find an agreeable practice location in the country. Dr Brown saved many lives, counseled countless others, and even played the violin at a patient's funeral (a directive from the patient!).

Dr Brown's descriptions of Cleveland, New York, and the surrounding north shore towns around Lake Oneida are particularly interesting. One gets impressions of small town American life circa 1938–1950s not unlike earlier depictions from Sherwood Anderson's *Winesburg, Ohio* or even the Frank Capra film *It's a Wonderful Life*. This book stands on its own merits as a depiction of American culture from this era. The author's social commentaries concerning poverty, governmental bureaucracy, and the transformation of the medical profession are not new but are honest, conservative, wizened opinions.

Marvin Brown is a country family doctor who has won many medical and community awards and practiced family medicine for almost 50 years. As is evident from the last section of this memoir, he was, more importantly, also married to a wonderful lady for almost 50 years. This wealth of experience in medical practice and in marriage is movingly told here in anecdotal form, and should reinspire the reader, as it did for me, to remember what our professional and personal lives can be all about.

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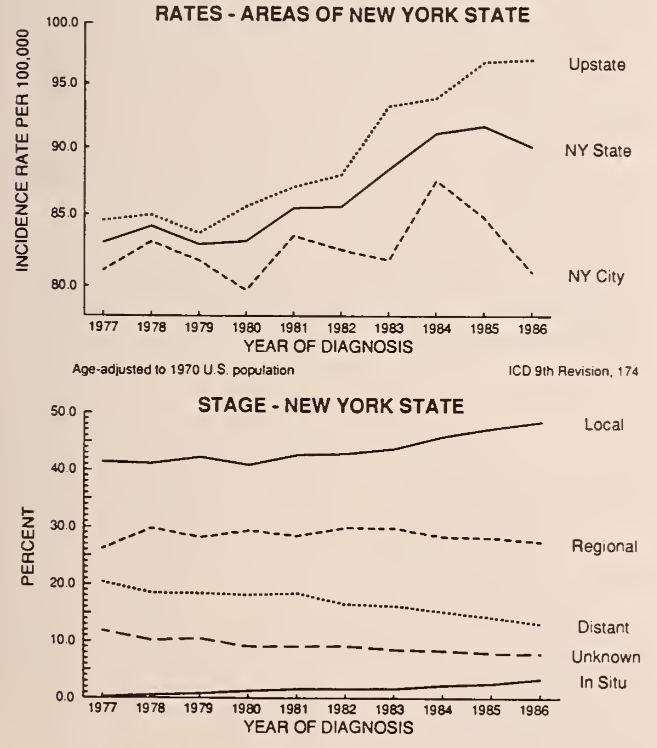
Time trends in breast cancer incidence

Breast cancer is the leading cancer among women in New York State. Nearly 11,000 cases were diagnosed in 1986, up from 9,300 a decade ago.[1] Several risk factors have been identified, but to date no single factor or combination of factors can be used to predict breast cancer occurrence. The major risk factors are age, white race, never married, family history of breast cancer, no children or first child born after age 30, early onset of menstruation or late menopause, obesity, history of benign breast disease, high socioeconomic status and urban residence.[2]

Patterns of breast cancer incidence are illustrated here for New York State women 1977–1986. Figure 1 shows age-adjusted incidence rates for New York State, New York City and New York State exclusive of New York City (upstate). Breast cancer incidence in the state increased from 83.0 per 100,000 in 1977 to 90.1 in 1986. Upstate New York's increase was dramatic, rising from 84.6 to 96.9 over the decade. Rates for New York City were variable, increasing from 81.1 in 1977 to 87.5 in 1984, but dropping back to 80.9 by 1986.



Source: New York State Cancer Registry

FIGURE 1. Breast cancer incidence, 1977–1986: New York State, New York City, Upstate New York.

The patterns of increasing incidence in New York State and upstate are consistent with increasing incidence reported by the Surveillance, Epidemiology, and End Results (SEER) program. From 1977 to 1986 breast cancer incidence increased in the United States from 83.4 to 104.9 per 100,000, a 26% increase.[3] The trend for New York City is less defined and its lower rates are likely due to the large population of blacks, Hispanics and other minority women for whom breast cancer rates are lower overall.

New emphasis nationally on early detection of breast cancer through mammography appears to account for at least some of the observed increases. As shown in the lower graph of Figure 1, there were steady increases in the percent of cases in New York State diagnosed as *in situ* or localized to the breast, notably since about 1983. A corresponding decrease occurred in the percent of cases with distant metasteses and in the percent of cases with unknown stage. Cases with regional spread to nearby organs remained unchanged. Changes in stage at diagnosis were similar for New York City as well as upstate.

Similar trends by stage of disease have also been reported for the San Francisco-Oakland Metropolitan Statistical Area.[4] In that highly urban area, 11% of breast cancer cases diagnosed in 1986 were *in situ*. New York State had 3.4% in 1986 and New York City had 3.1%. Both San Francisco-Oakland and New York State experienced relatively stable breast cancer rates until the 1980s. Beginning about 1983, both areas experienced rising rates. Table I compares the relative rates for various stages of breast cancer since 1983 to the more stable period of prior years. Both areas experienced similar trends of increasing

TABLE I. Incidence Rates of Breast Cancer by Stage In New York State, 1977–1986, and in the San Francisco-Oakland Metropolitan Statistical Area,* 1974–1986

		Early-Stage Cancers		Relative Rate According to Stage				
Period/ Year	No. of Cases	In Situ	Local	In Situ	Local	Regional	Remote	Unknown
		(Percent of cases)		*(Percent of base period mean)*				
New York State								
1977–1982	57,174	1	42	100	100	100	100	100
1983	10,318	2	44	185	113	113	95	93
1984	10,778	2	46	259	123	112	94	96
1985	11,023	3	47	299	130	113	89	92
1986	10,968	3	48	381	132	110	82	90
San Francisco-Oakland								
1974–1982	16,956	5	49	100	100	100	100	†
1983	2,144	6	47	137	98	108	89	†
1984	2,362	8	52	190	118	101	95	†
1985	2,657	11	52	274	128	105	90	†
1986	2,720	11	55	296	135	92	90	†

* Source: Reference 4
† Not given

in situ and localized cancers and decreasing metastatic cancers.

A New York State Department of Health publication, *Time Trends in Cancer Incidence 1977–1986*, for New York State, New York City and upstate New York will soon be available from the New York State Cancer Registry. In addition to the numbers of cases and rates by sex, age and year, a number of important cancer sites are highlighted. Featured are brief discussions and graphical illustrations of time trends for smoking-related cancers, gastrointestinal cancers, uterine corpus and uterine cervix cancers, female breast cancer, malignant melanoma, lymphomas and Kaposi's sarcoma. Single copies may be ordered from the New York State Department of Health, Cancer Registry, Room 536, Corning Tower, Empire State Plaza, Albany, NY 12237.

References

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Poison control

The New York State Poison Control Network Act, enacted May 12, 1986, established a statewide network of regional poison control centers to reduce poisonings, educate people about hazardous exposures, ensure statewide emergency coverage by poison control facilities, provide expert information to professionals and the public, collect data and conduct research.

Regional poison control centers can reduce hospital costs by handling nontoxic and mildly toxic poisoning emergencies through telephone consultation. Statewide savings could total $35 million annually. Costs are reimbursed through insurance payments.

In 1955, 27 deaths from poisoning of children under age five in New York City led to the first poison control center in New York State. Integrated with the New York City Health Department, the first center was a collaborative effort of the American Academy of Pediatrics, New York Academy of Medicine and local medical societies.

Over the next two decades, additional regional programs provided poison control services. The scope of these programs varied depending on the needs of the community and the available funding. To determine the level of poison control services available, the State Health Department conducted a 1979 survey of all poison control services in the state. Results illustrated that only 50% of New York State's population received any level of poison control services and there was a lack of service uniformity in those areas.[1]

In 1981, Health Commissioner David Axelrod established an advisory council on poison prevention and control to advise the department on the further development of statewide standards for poison control services. In 1984, the council and department developed the comprehensive *Administrative Guidelines for the Operation of Poison Control Centers*, subsequently used in developing state regulations.

Network Implementation

The law requires the health commissioner to designate and establish minimum standards for regional poison control centers, report annually to the legislature and establish an advisory committee.

- On September 4, 1986, the department sent a notification letter to all New York State hospitals about designating regional poison control centers. On April 28, 1987, a regional poison control center was designated in each of the state's eight health service areas. Subsequently, consolidation of two regions resulted in the present complement of seven centers.
- The statutory advisory committee, formed in 1987 by the health commissioner, meets regularly to counsel the Health Department on poison prevention, treatment and organizing regional poison control activities. The advisory committee includes directors of regional poison control centers, consumers, industrial representatives and representatives of state and federal agencies involved in poison safety.
- The managerial activities are overseen by the Injury Control Program and include review and approval of program plans and budgets, monitoring each center's progress toward network achievements and documenting the operations of each center and the network.

The network's performance is evaluated through narrative and statistical reports and onsite visits to the regional poison control centers. Following the implementation of the funding mechanism during 1988, most centers have hired additional staff, prepared outreach materials and acquired necessary equipment. Medical directors and administrators have been appointed and poison information specialists and health educators hired. Procedures for patient management, follow-up and referral have been adopted. A standard data collection system was adopted January 1990.

During 1987, an estimated 184,000 calls were handled by the regional poison control centers, for a rate of ten calls per 1,000 population, as shown in Table I. Within

TABLE I. Estimated Number of Calls to Regional Poison Control Centers, New York State Department of Health, Injury Control Program, January 1, 1987–December 31, 1987

HSA	Hospital	Estimated No. of Calls	Population	Calls per 1,000 Population
1	Children's, Buffalo	16,673	1,585,800	10.5
2	University of Rochester	15,789	1,192,900	13.2
3	SUNY Syracuse Health Science Center	11,647	1,411,600	8.3
4	Binghamton General	4,921	310,400	15.9
5*	Ellis, Schenectady	6,934	1,364,300	5.1
6	Nyack	22,587	1,969,600	11.5
7	New York City	73,041	7,282,200	10.0
8	Nassau County Medical Center	32,792	2,629,500	12.5
Total		184,384	17,746,300	10.4

* *Effective 1/1/90, operations handled by Nyack Hospital*

each HSA, poison information calls and services were more heavily concentrated within the vicinity of the center than in the outlying counties. Improved outreach to the outlying areas is expected during 1990, as the centers expand their staff and advertise their accessibility. The addition of a health educator to each center's staff will facilitate a greater focus on targeted activities for prevention of poisoning episodes among high-risk and underserved populations.

Adapted from New York State Poison Control Network Act, 1988–1989 Report to the Legislature. Single copies are available from the New York State Department of Health, Injury Control Program, Room 621, Corning Tower, Albany, NY 12237.

Reference

1. Fisher L: The next five years: The goals and objectives of poison control systems in New York State. *J Vet Hum Toxicol* 1981; 23:103–107.

REGIONAL POISON CONTROL CENTERS

There are seven regional poison control centers in New York State.
In an emergency, contact the regional poison control center serving your county.

REGION/COUNTIES			POISON CONTROL CENTER	
Region 1				
Allegany	Cattaraugus	Chautauqua	Children's Hospital	
Erie	Genesee	Niagara	219 Bryant Street	1-800-888-7655
Orleans	Wyoming		Buffalo, NY 14222	(716) 878-7654
Region 2				
Chemung	Livingston	Monroe	University of Rochester Medical Center	
Ontario	Schuyler	Seneca	601 Elmwood Ave., Box 321	1-800-333-0542
Steuben	Wayne	Yates	Rochester, NY 14642	(716) 275-5151
Region 3				
Cayuga	Cortland	Herkimer	SUNY Syracuse Health Science Center	
Jefferson	Lewis	Madison	750 East Adams Street	1-800-252-5655
Oneida	Onondaga	Oswego	Syracuse, NY 13210	(315) 476-7529
St. Lawrence	Tompkins			
Region 4				
Broome	Chenango	Tioga	Binghamton General Hospital	
			Mitchell Street	
			Binghamton, NY 13905	(607) 723-8929
Regions 5 and 6				
Albany	Clinton	Columbia	Nyack Hospital	
Delaware	Dutchess	Essex	North Midland Ave.	1-800-336-6997
Franklin	Fulton	Greene	Nyack, NY 10960	(914) 353-1000
Hamilton	Montgomery	Orange		
Otsego	Putnam	Rensselaer		
Rockland	Saratoga	Schenectady		
Schoharie	Sullivan	Warren		
Washington	Westchester	Ulster		
Region 7				
Bronx	Kings	New York	New York City Poison Control Center	
Richmond	Queens		455 First Avenue	(212) POISONS
			New York, NY 10016	(212) 340-4494
Region 8				
Nassau	Suffolk		Nassau County Medical Center	
			2201 Hempstead Trnpk	
			East Meadow, NY 11554	(516) 542-2323

OLD MEN OF THE BOWERY: STRATEGIES FOR SURVIVAL AMONG THE HOMELESS

By Carl I. Cohen, MD, and Jay Sokolovsky, PhD. 248 pp, illustrated. New York, Guilford Publications, Inc, 1989. $17.50 (paperback)

> " . . . stranded by time on the bleak shore of a forgotten or friendless old age."
> —Shaftesbury, 1839

"Skid row" is a term derived from Seattle's 19th century Skid Road, down which loggers dragged their loads to a mill. This street attracted cheap bars and housing. *Skid row*, or *skid road*, has assumed striking metaphorical qualities, a term standing for a downhill course of life without hope, on the skids.

This eloquent book is written by two investigators with authority in the field earned through years of service on New York City's skid row, the Bowery. The volume fills an important need in clarifying the characteristics of older Bowery men, and offers cogent proposals for their survival. Homeless men age early. Few survive to advanced age. Studies of more than 220,000 homeless persons examined at shelter clinics in 19 cities across the country between 1985 and 1987 show that 3% to 4% are age 65 and older, while the figure for the general population is about 12%. Premature death is the likely cause of this differential. Lack of shelter has a markedly harmful effect on all persons, but the elderly homeless are in double jeopardy.

Twenty-five years ago a homeless woman was a rarity, and homeless children essentially nonexistent. Hallucinating schizophrenics were not prominent on the streets. Runaway youths were not recognized as a major problem. Skid row men, mostly middle-aged white alcoholics, were the homeless of the 1960s. Today, homeless women and children are common, many homeless persons are severely disabled by chronic mental illness, alcoholism is relatively less common and abuse of other drugs is much more so. The homeless are younger, and many more are minorities. Now, skid row men constitute perhaps one-fifth of the total.

This book, then, concerns a modest proportion of the homeless. Rather than minimizing the importance of these men, however, we should acknowledge that, in their vulnerability, they need enhanced attention. They stand for all of us in their frailty and humanity.

This comprehensive work considers the history of homelessness; means of survival on the streets and in flop houses; social supports; physical and mental health; alcoholism; and strategies for intervention. Cohen and Sokolovsky have the skill and passion to bring the skid row world to life. The authors describe themselves, their own work, and motives, an enhancing personal touch. Also, the entire text is exemplified and realized by the running life histories of three prototypical skid row men, each known well to the authors.

Caveats are minor. The study, conducted in 1982–1983, is a bit dated. There are several typographical errors; and there is one inanity: the authors attempt to blame traditional homelessness on "the full development of capitalism." However, any well-read person is aware that some members of society will sink to the bottom, no matter what the ruling economy, as this quote from a memoir illustrates: " . . . winos dotted the pavements or staggered through the crowds and accosted passersby, as is the custom throughout the whole of Russia" (*Russian Doctor*, 1984).

This work—clear, accurate, and of high quality—is a worthy addition to the long and resonant field of skid row studies.

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MENTAL HEALTH CARE FOR ALLIED HEALTH AND NURSING PROFESSIONALS

Edited by H. Steven Moffic, MD, Pedro Ruiz, MD, and George L. Adams, MD. 271 pp. St Louis, Mo, Warren H. Green, Inc, 1989. $27.50 (softcover)

This softcover book is divided into three sections: "Evaluation," "Treatment," and "Mental Health of the Care Giver." The assumed goal of the title is monumental. The topics are numerous and represent a compilation of the work of 21 contributors.

As in the case with most books with numerous contributors, the style of writing is inconsistent and the subject matter is uneven. There is no preface, introduction, or foreword, thus clouding the purpose of the book. Later, however, chapter 13 tries to more clearly explain the book's purpose by differentiating mental health nursing skills from psychiatric nursing. Since the book is addressed to a wide variety of health care providers with different levels of expertise and/or exposure, the contents fall short in some areas while being steadfastly detailed in others. Although the issues involved in dealing with patients in mental distress have remained fairly consistent over the past decade or two, much of the presented material is outdated. The most recent reference used in the text was from 1983, with "additional annotated references" attached, as if by afterthought. An example in chapter 6 cited "recent research contributors" (1974, 1973, 1968, and 1965). Furthermore, there were many distracting typographical and grammatical errors, beginning with the first sentence. These were quite annoying and difficult to overcome. As the chapters progressed, thankfully, fewer errors were found.

Few, if any, medical references have tried to introduce mental health care to allied health and nursing professionals. However, there are other texts written exclusively by either nursing personnel or specific allied health professionals which are much more comprehensive and easier to utilize as a reference.

This book is not without some highlights. Chapters 2, "Interviewing Techniques," and 9, "Psychotherapy and Counseling Skills," are excellent, with many useful pearls of information. Also noteworthy are the case examples interspersed in some of the chapters. The majority of the chapters, however, are dry with some interesting but not very useful information. For example, chapter 10, "Behavior Therapy: An Overview and Application for Sexual Dysfunction," provides a good overview of behavior therapy, but fails to provide information on how it can be applied to more common problems. It familiarizes the reader with techniques but without the background expertise, the professional would be taking on much more than he or she is capable of.

In summary, this 271-page softcover book is outdated in some of its information, unclear in its purpose, too generalized in its target market, and full of typographical and grammatical errors. It is not without some saving chapters, but the shortcomings far outweigh the positive aspects. The $27.50 price for this book would be better spent for a more profession-specific or comprehensive text that can be found in the literature.

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MAKE SURE YOU DO NOT HAVE BREAST CANCER

By Philip Strax, MD. 118 pp, illustrated. New York, St Martin's Press, 1989. $15.95 (clothcover)

This small, hardcover book is the kind of book that leaves me uncomfortable after reading it. While it is an excellent treatise for health-conscious women on one of their most terrifying concerns—so good, in fact, that I wish it could be supplied to every woman graduating from high school—my discomfort arises from the fact that readership will most likely be low.

Most medical books written for laymen by physicians are not widely advertised and thus have relatively small markets. Books also lag far behind other methods of getting educational material into the hands of those for whom they are intended. Nearly always, the language used to transmit this information is considerably above the reading level of those for whom it would be most helpful.

Despite these generic drawbacks, this is a fine little book. In essence, it is an update of a 1974 book by the author, *Early Detection: Breast Cancer Is Curable*. Who is better qualified than Dr Philip Strax to prepare this material? He was one of the pioneers of breast health, and has been a leader and prime investigator since its importance became evident more than a quarter of a century ago. The book is logical and well thought out and it covers all the information which is important. Chapter 6, entitled "What Constitutes a Breast Examination," is especially good. Four times in this chapter there is a statement that breast self-examination (BSE) monthly *for life* is important.

The book is full of reassurances, noting that most women do not get breast cancer, and it speaks of breast cancer as "The Dragon In Their Midst!" It manages to assure that the chances of getting the disease are small indeed, which is such an important message. There are refreshingly few "doctor speak" passages, but the level of language is still above average for the group of women most at risk and those who need it most—the disadvantaged, minorities, and the poorly educated.

There is a section on the importance of motivation which is important for doctors to instill in their patients. However, as Dr Strax notes in his preface, "Even more, a large proportion of the medical profession have yet to be convinced, and motivated to, in turn, urge women to follow through."

Another very important point is made with regard to the emerging trend for women to select the therapy they feel would be best in their particular case. Dr Strax stresses that women have no real scientific basis for making this decision. The presence of intense anxiety and lack of knowledge of the disease makes it extremely unlikely that a valid decision can be made. The real decision and the only one a woman can reliably make is that of choosing which physician will be responsible for her treatment. She must rely upon the good judgment of the physician in whom she has confidence.

The one thing that would markedly increase the influence of this little book would be ensuring wide availability, possibly via paperback, through the American Cancer Society, a foundation, or other philanthropic association so that the message could be widely disseminated.

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BABY TIME: A GROWNUP'S HANDBOOK TO USE WITH BABY

By Laurie Krasny Brown and Marc Brown. 48 pp, illustrated. New York, Alfred A. Knopf, Inc, 1989. $12.95 (hardcover)

It is refreshing to find a new approach to parental education among the many baby care books available. Laurie Krasny Brown seeks to instill new parents with confidence in their role as nurturers. *Baby Time* is a picture book which is designed to be read by the parent and child together. In addition to captioned illustrations of active children, each page has a short text for the parent discussing the developmental significance of the activities. The book also gives practical suggestions for further enhancing a baby's awareness of his environment. The content is divided into discussion of each of the five senses. Dr Brown, an educational psychologist, provides the reader with simple explanations of developmental concepts such as object permanence and the acquisition of fine and gross motor skills. The text is too elementary for persons with a background in child development, but would be ideal for most first-time parents (especially very young parents), babysitters, and older siblings. Marc Brown, the author's husband, is to be commended for his nonsexist and expressive illustrations of parents and children.

While Dr Brown does not attempt to create an encyclopedia of child care, she does stress the importance of safety and childproofing the home. However, one error in this realm is made on the page illustrating various finger foods for babies. Whole grapes and berries are included as examples. These items should never be given to children under three years of age unless cut into quarters because of the danger of choking and aspiration.

As a pediatrician, I will recommend this book to parents as a tool for understanding and enjoying their child's development. I support the overall message that parents should relax and trust their own instincts in creating a stimulating environment for the child. As a parent, I will also use the book with my son as a means of encouraging language skills and an appreciation of books.

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THE CHILD IN OUR TIMES: STUDIES IN THE DEVELOPMENT OF RESILIENCY

Edited by Timothy F. Dugan, MD, and Robert Coles, MD. 240 pp, illustrated. New York, Brunner/Mazel Publishers, 1989. $25.00 (hardcover)

The word "resiliency" in the title of this book refers to the capacity of children to undergo grave psychological, physical, and environmental stresses and yet somehow in the face of such adversity have the capacity to grow toward becoming competent and mentally healthy adults. Those of us working in medicine and the mental health field observe this phenomenon repeatedly in our professional lives. This volume is a comprehensive attempt to study why this occurs, and as Timothy Dugan, MD, one of the authors, so aptly states in the preface, "it seems to me that if we are interested in elucidating a truly general developmental psychology, then we must complement our highly evolved thinking about pathology-producing variables with a like measure of thinking about development-sustaining variables. Only in a synthesis of these two forces can we then feel assured that we are indeed understanding development."

Drs Dugan and Coles have approached their subject by assembling an excellent array of contributors in this relatively new area of study. Both physicians are eminently qualified for editing such a book. Dr Coles has spent 25 years adding to our knowledge on how poor and often quite vulnerable children struggle against the vicissitudes of their life circumstances in an attempt to survive and in his words, "demonstrate certain qualities of mind and spirit which strike an observer from another world as astonishing." Dr Dugan brings to the book a psychoanalytic understanding of child and adolescent development which permeates the book and helps it to make contributions beyond sociological or medical epidemiology.

The book adds to our knowledge in three areas regarding resiliency. The first is documentation and description of the unexpected capacity of some children to overcome harsh realities in life. These stresses may be psychological, as in the case of children from alcoholic and other dysfunctional families; physical, as in the case of children with severe and chronic illnesses, such as diabetes; or social, for example, children who grow up in poverty or even as street urchins. The second contribution has to do with presenting various types of designs for studying resiliency in childhood along with reviewing the literature, so that any readers who wish to pursue this fascinating area of study can use this book for a ready reference. Third, and most interesting to this reviewer, are those aspects of the articles that have to do with the precursors for the development of resiliency in childhood; that is, the intrapsychic and interpersonal experiences of infants and children that seem to make them less vulnerable and more adaptable. In these discussions there are really no surprises, but the importance of the documentation that infants and children who are raised by parents who are empathic, supportive, flexible, authoritative but not authoritarian, etc, develop from infancy on the characterological strengths to meet adversity and survive. And they survive not only in the concrete sense of the word, but also in their greater capacity to live a life that might be described as scientific. They are able to develop a mind that is inquiring, open, and not self-deluding.

The book is important reading for those physicians who interact with infants, children, and adolescents as a part of any treatment plan. Such persons must take into account and assess the capacity of the child to react to the stress of illness in a constructive rather than destructive way. Further, it provides for the physician a framework of understanding and, to some extent, predictability as to how an individual child will react when it is at risk.

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DRY ALL NIGHT: THE PICTURE BOOK TECHNIQUE THAT STOPS BEDWETTING

By Allison Mack. 176 pp, illustrated. Waltham, Mass, Little, Brown & Co, 1989. $17.95 (hardcover)

As the title indicates, the purpose of this beautifully illustrated and designed little book is to help the enuretic child overcome this troublesome and disturbing condition. The author, Allison Mack, who previously wrote *Toilet Training: The Picture Book Technique for Children and Parents*, has done her homework extremely well. She has researched the subject of enuresis extensively. As pointed out in the acknowledgments, Ms Mack has discussed the subject with and listened to a great many medical authorities on bed-wetting.

Dry All Night is divided into two main sections. Part 1 is directed to the parents and offers a ten-step program to help the parent help the child remain dry all night. The suggestions in this section all make very good sense, starting with the use of underpads and draw sheets, the realization that bed-wetting is a common problem, and the advice to parents never to tease, criticize, or punish the child, and dealing with any contributing factors to the problem. Further steps include helping the child make a dry-all-night scoreboard and communicating supportively with the youngster.

Part 2, "Soon I'll Be Dry All Night," is for the child to read. Since studies have shown that bed-wetting stops faster when the child takes on the responsibility for ending it, the author outlines just such a program for the enuretic child. This step-by-step method is presented in the form of a wonderful picture story that allows and encourages the child to assume complete responsibility for the bed-wetting. This section is the core of the book. Its purpose is to build up the child's confidence with one positive reinforcement after another, page by page. This program is presented in a most appealing format. It appears to be easy to implement. It does not require any special equipment nor does it make complicated demands on the rest of the family.

Since enuresis is such a common problem and since it often is associated with significant psychological problems, such as loss of self esteem, embarrassment, and peer rejection, along with being a cause of increased tension between parents and child, a book such as *Dry All Night* is an important contribution, and a most welcome and helpful addition to the literature.

I would question the author's statement that "parents often tell me that their sleep-wetting child takes the bottle to bed." Since by definition enuresis does not begin until the child is at least five or six years old, I do not understand why taking a bottle to bed is discussed at all. With rare exceptions, the bottle is long gone by the time a child is considered to be enuretic.

The author's stated objective is to help the child stop bed-wetting. I believe the author has succeeded. The book follows sound and accepted principles, and is easy to read and to follow. But I would have been even happier if the author had actually tested the effectiveness of her system before publication. Had 100 enuretic children and their parents followed Ms Mack's program and it resulted in a significant percentage of success (taking into account the fact that 15% stop their bedwetting each year without any treatment at all), I would be even more enthusiastic in recommending this book to the parents of my enuretic patients.

I believe that *Dry All Night* meets a significant need. Before considering the use of drugs or various urine alarms for treating a bed-wetter, I now will advise that parents get a copy of this book. Even in cases when it does not succeed in stopping the bed-wetting, it still puts the whole subject in its proper perspective, both for the parents and for the child. And this in itself is very important.

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ESSAYS ON THE RISE AND DECLINE OF BEDSIDE MEDICINE

By Mark D. Altschule, MD. 458 pp, illustrated. Philadelphia, Pa, Lea & Febiger, 1989. $29.50 (hardcover)

As a solo medical practitioner and a voluntary teacher for more than 25 years, the title of this book and my admiration for its author made me grasp the opportunity to read and review it for the *Journal*. The writing is excellent and obviously required a great deal of historical research. The introduction and epilogue alone are worth reading. Dr Altschule makes a very strong case in favor of the overwhelming importance of the patient-physician relationship in the practice of medicine. He deplores the lack of communication and the overuse of testing, as many of us do, but goes too far in his criticism of the role of the "basic sciences."

Part I (220 pages) is very heavy reading and I found it close to boring. The important contributions of the English, French, and Irish are described in great detail. Part II stirred my interest. The author recounts the familiar stories of the outstanding medical centers in Vienna and Germany in the late 19th and early 20th century and then takes the reader up to and through the famous Flexner Report of 1910.

I particularly enjoyed the last five chapters, but as a clinician, hoped to read more on such outstanding bedside teachers as Peabody, Osler, Harrison, Pickering, Dock, Keefer, and others—a breed that is fast becoming extinct. Overall, I enjoyed this book and recommend it to all those interested in clinical medicine.

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AIDS is affecting women at increased rates

Based on current indications, researchers at the Centers for Disease Control (Atlanta, Ga) predict that acquired immunodeficiency syndrome (AIDS) will be one of the five leading causes of death among women of childbearing age by 1991 (*Wall Street Journal*, July 11, 1990). This increasing incidence of AIDS among women will also have a direct effect on the number of babies born who are seropositive for the human immunodeficiency virus (HIV) and at risk of developing AIDS. In 1989, for example, of the almost 2,000 infants born with HIV infection, about 80% had acquired the virus from their mothers.

In 1980, at the beginning of the AIDS epidemic, 18 women aged 15 to 44 years died of AIDS. This figure rose to 1,430 in 1988, at which time HIV infection was responsible for 3% of deaths among this population of women. The deadly spread of AIDS among women is especially evident in New York and New Jersey, in which AIDS is already the primary cause of death among young black women.

Synthetic protein binds AIDS virus and prevents its spread

CPF, a small man-made molecule composed of the amino acids proline and phenylalanine, can block the site on the surface of the AIDS virus that enables the virus to stick to and penetrate healthy T cells (*The Morning Call*, July 20, 1990, p A3). CPF's antiviral activity has been impressive in the test tube, and animal tests are now underway. The synthetic molecule is easy and inexpensive to produce, according to researchers at the Dana-Farber Cancer Institute (Boston, Mass).

The researchers have found that CPF sticks so tightly to the gp120 molecule on the surface of the human immunodeficiency virus (HIV) that it cannot be washed off. When CPF attached to HIV, the virus was unable to infect healthy T cells in vitro. In subsequent tests, the researchers demonstrated that when CPF is put in a test tube with HIV-infected T cells and uninfected T cells, the AIDS virus did not spread from the infected to the healthy immune cells.

In vivo studies will help determine whether CPF is stable and effective in the body, whether it is specific for the gp120 molecule or will stick to other things, and whether it can be tolerated at therapeutic doses.

Incidence of toxic shock syndrome drops dramatically over ten-year period

In the ten years since toxic shock syndrome (TSS) was first recognized, 3,295 cases have been reported to the Centers for Disease Control (CDC) in Atlanta (*MMWR* 1990; 39:421-423). The nationwide public health initiatives aimed at identifying and eliminating risk factors for TSS have helped to reduce the number of cases from 890 in 1980 to 61 in 1989.

Of the 55 cases of TSS first reported to the CDC in May 1980, 95% occurred in women, and the illness began during menstruation in 95% of the women who provided a menstrual history. The subsequent national surveillance program led to a correlation between TSS and tampon use, and the knowledge that risk of TSS increased with increased tampon absorbency.

In 1980, tampon absorbency—measured in vitro—ranged from 10.3-20.5 g, and 42% of tampon users chose very high absorbency brands. By 1986, use of very high absorbency tampons had dropped to only 1% of tampon users. In 1990, the FDA required standardized absorbency labeling of tampons, which ranges from 6 g to 15 g. Manufacturers have also altered tampon composition.

The association between tampon use and toxic shock syndrome is still not well understood, yet awareness of the increased risk of menstrual TSS with tampon use has led to a substantial decrease in cases during the past ten years, while the number of cases of nonmenstrual TSS has remained virtually unchanged.

New prenatal test yields results in one day

Prenatal diagnosis of chromosomal abnormalities such as Down's syndrome typically requires at least two weeks while the fetal cells, which are obtained through amniocentesis, grow in culture. Using DNA probe technology, researchers will be able to test for many chromosomal abnormalities in as little as 24 hours (*Science* 1990; 248:121). The DNA probe, which is specific for a certain segment of DNA on a specific chromosome, binds to the chromosome and can then be identified using a fluorescent dye. Abnormal numbers of or portions of chromosomes can be easily recognized.

A new drug to treat osteoporosis increases bone mass and reduces fractures

Etidronate, a biphosphonate compound which inhibits osteoclast-mediated bone resorption, has shown promise in clinical trials as a therapy for postmenopausal osteoporosis. A two-year, prospective, controlled study of etidronate in 423 women with postmenopausal osteoporosis demonstrated the drug's ability to increase spinal bone mass and reduce the incidence of new vertebral fractures (*N Engl J Med* 1990; 323:73-79). Current therapies available in the United States include oral estrogen and parenteral calcitonin, neither of which are safe or effective in all women.

The subjects in this study received intermittent cyclical phosphate and etidronate therapy or placebo, with the cycle of administration repeated every 91 days for a two-year period. In the treatment groups, which received etidronate, there were "significant increases from base line in the bone mineral density of the spine after 12, 18, and 24 months ($p < 0.017$)." The etidronate-treated groups also had fewer patients with new vertebral fractures. The authors reported a 50% decrease in the rate of new vertebral fractures. "These beneficial effects were greatest in the women who had the lowest spinal bone mineral densities at the beginning of the study."

Reported adverse effects of etidronate, including nausea and diarrhea, were in general mild and infrequent. "Intermittent cyclical therapy with etidronate should be a

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NEWS BRIEFS are compiled and written by Vicki Glaser.

welcome addition to the therapeutic options for osteoporosis," conclude the authors of this study.

Growth hormone therapy may reverse some age-related bodily changes

The decrease in lean body mass and increase in adipose tissue mass that occur with aging are due in part to reduced secretion of human growth hormone. Rudman et al support this conclusion with a study of 12 men aged 61 to 81 in whom treatment with biosynthetic human growth hormone produced significant increases in lean body mass, the average density of the lumbar vertebrae, and the sum of skin thicknesses at four sites, as well as a significant decrease in the mass of adipose tissue (*N Engl J Med* 1990; 323:1-6).

The 12 subjects were healthy men who had greatly reduced levels of circulating growth hormone, as measured indirectly based on insulin-like growth factor I (IGF-I) concentrations. IGF-I is synthesized and released in the body in response to growth hormone. The subjects all had IGF-I concentrations below 350 U/L (normal range for healthy men aged 20–40 years, 500–1,500 U/L). The beneficial effects of growth hormone therapy demonstrated in this study could only be generalized to the approximately one third of men aged 60–80 years who have plasma IGF-I concentrations below 350 U/L.

As the American population ages, the findings of this study hold promise for reversing some of the effects of aging. "The effects of six months of human growth hormone on lean body mass and adipose-tissue mass were equivalent in magnitude to the changes incurred during 10 to 20 years of aging."

Low-dose cyclosporin reduces joint inflammation in rheumatoid arthritis

In earlier studies, cyclosporin administered in doses of 5 mg/kg or more was able to lessen joint inflammation in patients with rheumatoid arthritis. However, the nephrotoxicity of the drug necessitated the cessation of treatment. In a recent issue of the *Lancet* (1990; 335:1051–1055), Tugwell et al reported the results of a placebo-controlled trial with low-dose cyclosporin.

The 144 subjects received either placebo or cyclosporin—the latter at an initial dose of 2.5 mg/kg/day given in two doses every 12 hours. The dose was increased by 25% each week to achieve final serum trough levels of cyclosporin of 75–150 ng/mL. Serum creatinine levels were monitored, and cyclosporin doses were moderated if serum creatinine levels rose by 50% or more from baseline. The mean dose of cyclosporin administered was 3.79 mg/kg/day. Following the six-month drug treatment period, the cyclosporin dose was tapered off for a period of eight weeks.

The clinical evaluation included a study of active joints, "as determined by pain on passive motion and tenderness on pressure, or inflammatory joint swelling," an assessment of pain, and a subjective questionnaire in which patients describe problems related to their arthritis.

The authors reported a 23% improvement for the joint count, 24% for pain, and a 16% improvement in patient functional status for the treatment group. "The blinded assessor rated 62% and 35% of cyclosporin and placebo patients, respectively, as improved globally at 6 months ($p = 0.001$)." Only one patient experienced problematic renal dysfunction that was not ameliorated by altering the dose of cyclosporin.

The authors recommend starting cyclosporin therapy at low doses, even though improvement is slower to appear, increasing doses slowly, and monitoring renal function via serum creatinine levels. They encourage further testing of cyclosporin in patients with rheumatoid arthritis, as it "improves the patients' ability to carry out their daily activities in addition to the improvement seen in the traditional clinical measures."

New Merck manual addresses geriatrics

Recognizing the growing need for knowledge about geriatric medicine, with more Americans now over the age of 65 years than under age 25, Merck & Co (Rahway, NJ) recently published *The Merck Manual of Geriatrics*. The 1,267-page book explains the physiologic changes that naturally accompany aging and details diseases that affect the elderly, as well as special medical, social, ethical, and legal issues surrounding the treatment of geriatric patients.

David Irving Arbuse, MD, New York. Died May 25, 1990; age 90. Dr Arbuse was a 1923 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the New York Neurological Society, the American Academy of Neurology, the American Psychiatric Association, the Pan American Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

George C. Armistead, MD, Pelham. Died June 9, 1990; age 74. Dr Armistead was a 1941 graduate of the University of Virginia School of Medicine, Charlottesville, Virginia. He was a Diplomate of the American Board of Internal Medicine. His memberships included the American Occupational Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Leslie Paxton Barker, MD, New York. Died June 5, 1990; age 89. Dr Barker was a 1927 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Dermatology, Inc. His memberships included the New York Academy of Medicine, the New York State Dermatological Society, the American Dermatological Association, the American Academy of Dermatology, the Society for Investigative Dermatology, the New York County Medical Society, and the Medical Society of the State of New York.

Annelies Bergmann, MD, Williamsville. Died May 30, 1990; age 55. Dr Bergmann was a 1958 graduate of the Medizinische Fakultaet der Freien Universitaet Berlin, Berlin, Germany. Her memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Henry Brill, MD, Islip. Died June 17, 1990; age 84. Dr Brill was a 1932 graduate of the Yale University School of Medicine, New Haven, Connecticut. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a Fellow of the American Psychiatric Association. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Lynn R. Callin, MD, Fort Myers, Florida. Died June 8, 1990; age 82. Dr Callin was a 1936 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. He was a Diplomate of the American Board of Orthopedic Surgery. He was a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the American Academy of Orthopaedic Surgeons, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Ralph V. Chester, MD, Syracuse. Died June 5, 1990; age 73. Dr Chester was a 1943 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a Fellow of the American College of Obstetricians and Gynecologists. He was a member of the Onondaga County Medical Society and the Medical Society of the State of New York.

Edward Lyman Coffey, MD, Port Washington. Died June 19, 1990; age 56. Dr Coffey was a 1960 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Pathology and a Fellow of the American College of Pathologists. His memberships included the New York Pathological Society, the American Society of Clinical Pathologists, the International Academy of Pathology, the Nassau County Medical Society, and the Medical Society of the State of New York.

Alfred Deutsch, MD, New York. Died May 22, 1990; age 105. Dr Deutsch was a 1909 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

William C. L. Diefenbach III, MD, Southampton. Died June 26, 1990; age 67. Dr Diefenbach was a 1947 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Internal Medicine. His memberships included the American Geriatrics Society, the Pan American Medical Association, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Seymour Fuchs, MD, Bay Shore. Died May 30, 1990; age 62. Dr Fuchs was a 1956 graduate of the Medizinische Fakultaet der Universitaet Basel, Basel, Switzerland. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a Fellow of the American College of Obstetricians and Gynecologists and a Fellow of the American College of Surgeons. His memberships included the American Fertility Society, the Suffolk County Medical Society, and the Medical Society of the State of New York.

David Green, MD, Troy. Died May 13, 1990; age 69. Dr Green was a 1951 graduate of the University of Melbourne, Faculty of Medicine, Parkville, Victoria, Australia. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Academy of Neurology, the Association for Research in Nervous and Mental Disease, the Medical Society of the County of Rensselaer, and the Medical Society of the State of New York.

James Louis Green, MD, Floral Park. Died June 5, 1990; age 70. Dr Green was a 1943 graduate of the Cornell University Medical College, New York. He was a Diplomate of the American Board of Urology. His memberships included the New York Academy of Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Henry Greenbaum, MD, Forest Hills. Died December 6, 1989; age 82. Dr Greenbaum was a 1935 graduate of the Universite de Paris VI, Paris, France. His memberships included the American Psychiatric Association, the American Academy of Psychoanalysis, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Franklyn Alvin Huber, MD, Winter Park, Florida. Died May 30, 1990; age 84. Dr Huber was a 1933 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Aron Klibanski, MD, Bay Shore. Died February 21, 1990; age 93. Dr Klibanski was a 1928 graduate of the Medizinische Fakultaet der Universitaet Basel, Basel, Switzerland. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the American Geriatrics Society, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Teologos Logaridis, MD, Buffalo. Died June 1, 1990; age 70. Dr Logaridis was a 1945 graduate of the Bursa Tip Fakultesi Istanbul Universitesi, Bursa, Turkey. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Niagara, and the Medical Society of the State of New York.

Moisey Lutrin, MD, Brooklyn. Died June 7, 1990; age 69. Dr Lutrin was a 1963 graduate of the Doneckij Medicinskij Institut, Doneck, Ukrainian SSR, Union of Soviet Socialist Republics. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Mary R. Headlee Markham, MD, New York. Died June 6, 1990; age 82. Dr Markham was a 1940 graduate of the Ohio State University College of Medicine, Columbus, Ohio. She was a Diplomate of the American Board of Otolaryngology. Her memberships included the New York Academy of Medicine, the American Academy of Otolaryngology, the American Academy of Legal and Industrial Medicine, the American Medical Women's Association, the New York County Medical Society, and the Medical Society of the State of New York.

Bertrand Latimer New, MD, New York. Died June 13, 1990; age 64. Dr New was a 1954 graduate of the University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a Fellow of the American Psychiatric Association. His memberships included the American Academy of Child & Adolescent Psychiatry, the New York County Medical Society, and the Medical Society of the State of New York.

Shirley Rubler, MD, Great Neck. Died May 30, 1990; age 67. Dr Rubler was a 1948 graduate of the Medical College of Pennsylvania, Philadelphia, Pennsylvania. She was a Diplomate of the American Board of Internal Medicine. She was a Fellow of the American College of Physicians, the American College of Cardiology, and the American College of Angiology. She was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Lewis Alfred Scheuer, MD, New York. Died May 26, 1990; age 88. Dr Scheuer was a 1928 graduate of the Yale University School of Medicine, New Haven, Connecticut. His memberships included the American Public Health Association, the New York County Medical Society, and the Medical Society of the State of New York.

Edward Ludwig Schwabe, MD, Sykesville, Maryland. Died November 1989; age 91. Dr Schwabe was a 1942 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a Fellow of the American Academy of Family Practice. His memberships included the New York Academy of Medicine, the American Geriatrics Society, the Medical Society of the County of Chautauqua, and the Medical Society of the State of New York.

Harry L. Segal, MD, Rochester. Died June 4, 1990; age 90. Dr Segal was a 1924 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Diplomate of the American Board of Internal Medicine. He was a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the American Gastro-Enterological Association, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Joseph Charles Serio, MD, Buffalo. Died May 11, 1990; age 78. Dr Serio was a 1937 graduate of the St Louis University School of Medicine, St Louis, Missouri. He was a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Robert Turell, MD, New York. Died May 19, 1990; age 88. Dr Turell was a 1928 graduate of the University of Wisconsin Medical School, Madison, Wisconsin. He was a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Harold T. Vogel, MD, Rockville Centre. Died May 19, 1990; age 90. Dr Vogel was a 1924 graduate of the Yale University School of Medicine, New Haven, Connecticut. He was a Diplomate of the American Board of Pediatrics. He was a Fellow of the American Academy of Pediatrics. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Stephen Bennett Yohalem, MD, New York. Died May 12, 1990; age 74. Dr Yohalem was a 1941 graduate of the New York University School of Medicine. He was a Diplomate of the American Board of Internal Medicine. He was a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Guidelines for authors

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The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1988; 88:456-462). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925-927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337-338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

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ACQUIRED IMMUNODEFICIENCY SYNDROME

Current Issues and Scientific Studies

Edited by Pascal James Imperato, MD

With a foreword by
George J. Lawrence, Jr., MD, Chairman
AIDS Task Force
Medical Society of the State of New York

The epidemic of acquired immunodeficiency syndrome has presented Americans with a number of social, medical, political, and economic issues that are now the focus of much public discussion and, in some instances, heated debate. The chapters in this volume reflect a range of social and philosophical challenges posed by the epidemic and provide some stimulating insights from authors who have given these issues serious thought. Topics covered include the epidemiology of AIDS in the northeastern United States, an overview of the problem of AIDS in Africa, health education intervention strategies, the impact of the epidemic on the health-care system, issues related to HIV antibody testing, and common and unusual clinical complications encountered in the disease. This book consists of important papers on AIDS published in the *New York State Journal of Medicine* in 1987 and 1988.

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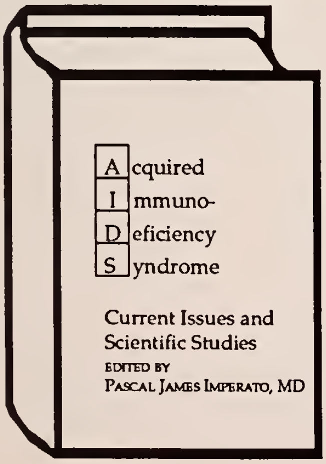

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References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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(*continued from p 5A*)

ARIZONA

Oct 12–14. **Advances in Drug Therapy.** 14 Cat I Credits. Arizona Inn, Tucson. Contact: Continuing Medical Education Office, 1501 N. Campbell Ave, Tucson, AZ 85724. Tel: (602) 626-7832.

Oct 16–21. **41st Annual Workshops and Scientific Program of the Society for Clinical and Experimental Hypnosis.** 43 Cat I Credits. Tucson Hilton East, Tucson. Contact: Marion Kenn, Administrative Director, SCEH, 128A Kings Park Drive, Liverpool, NY 13090. Tel: (315) 652-7299.

CALIFORNIA

Oct 11–12. **No Crystal Stair: Models for Successful Development of Afro-Americans.** Laurel Heights Campus of the University of California, San Francisco. *Oct 12–14.* **8th Postgraduate Course in Medical Mycology (Dermatomycology).** 16 Cat I Credits. University of California San Francisco. *Oct 20–21.* **The Skin From A to Z.** University of California Laurel Heights Campus. *Oct 26–27.* **The Aftermath of Suicide: Counseling Survivors.** University of California, San Francisco. Contact: Extended Programs in Medical Education, University of California, Room C-124, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

Oct 20–21. **Advanced Imaging of the Musculoskeletal System.** 12 Cat I Credits. *Oct 22–26.* **15th Annual San Diego Postgraduate Radiology Course.** 27 Cat I Credits. *Oct 26–28.* **Tenth Annual Interventional and Imaging Radiology.** 17½ Cat I Credits. Hotel del Coronado, San Diego. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

FLORIDA

Oct 4–7. **Second Annual Meeting and Convention of the American Association of Office Nurses.** Grosvenor Resort Hotel, Walt Disney World Village, Lake Buena Vista. Contact: Mary Ann Jones, RN, MA, President, The American Association of Office Nurses, 109 Kinderkamack Rd, Montvale, NJ 07645. Tel: (201) 391-2600.

HAWAII

Oct 6–13. **Annual International Body Imaging Conference.** Hyatt Regency Maui Hotel. Contact: Annual Interna-

(*continued on p 21A*)

CONTROL ACID RAIN

with once-a-night h.s. therapy for active duodenal ulcers

Zantac 300

ranitidine HCl/Glaxo 300 mg tablets

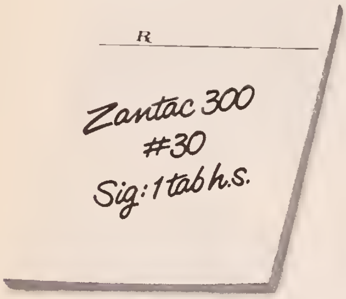
Glaxo/ROCHE

See adjacent page for Brief Summary of Product Information.

In active duodenal ulcers

Once-a-night h.s. therapy controls acid rain

Zantac® 300
ranitidine HCl/Glaxo 300 mg tablets



Zantac® 150 Tablets
(ranitidine hydrochloride)
Zantac® 300 Tablets
(ranitidine hydrochloride)
Zantac® Syrup
(ranitidine hydrochloride)

BRIEF SUMMARY

The following is a brief summary only. Before prescribing, see complete prescribing information in Zantac® product labeling

INDICATIONS AND USAGE: Zantac® is indicated in:

1. Short-term treatment of **active duodenal ulcer.** Most patients heal within four weeks.
2. **Maintenance therapy** for duodenal ulcer patients at reduced dosage after healing of acute ulcers
3. The treatment of **pathological hypersecretory conditions** (eg, Zollinger-Ellison syndrome and systemic mastocytosis).
4. Short-term treatment of **active, benign gastric ulcer.** Most patients heal within six weeks and the usefulness of further treatment has not been demonstrated.
5. Treatment of **gastroesophageal reflux disease (GERD).** Symptomatic relief commonly occurs within one or two weeks after starting therapy. Therapy for longer than six weeks has not been studied.

In active duodenal ulcer; active, benign gastric ulcer; hypersecretory states; and GERD, concomitant antacids should be given as needed for relief of pain

CONTRAINDICATIONS: Zantac® is contraindicated for patients known to have hypersensitivity to the drug.

PRECAUTIONS: General: 1. Symptomatic response to Zantac® therapy does not preclude the presence of gastric malignancy

2. Since Zantac is excreted primarily by the kidney, dosage should be adjusted in patients with impaired renal function (see DOSAGE AND ADMINISTRATION). Caution should be observed in patients with hepatic dysfunction since Zantac is metabolized in the liver.

Laboratory Tests: False-positive tests for urine protein with Multistix® may occur during Zantac therapy, and therefore testing with sulfosalicylic acid is recommended.

Drug Interactions: Although Zantac has been reported to bind weakly to cytochrome P-450 *in vitro*, recommended doses of the drug do not inhibit the action of the cytochrome P-450-linked oxygenase enzymes in the liver. However, there have been isolated reports of drug interactions that suggest that Zantac may affect the bioavailability of certain drugs by some mechanism as yet unidentified (eg, a pH-dependent effect on absorption or a change in volume of distribution).

Carcinogenesis, Mutagenesis, Impairment of Fertility: There was no indication of tumorigenic or carcinogenic effects in lifespan studies in mice and rats at doses up to 2,000 mg/kg/d.

Ranitidine was not mutagenic in standard bacterial tests (*Salmonella, Escherichia coli*) for mutagenicity at concentrations up to the maximum recommended for these assays.

In a dominant lethal assay, a single oral dose of 1,000 mg/kg to male rats was without effect on the outcome of two matings per week for the next nine weeks.

Pregnancy: ***Teratogenic Effects: Pregnancy Category B:*** Reproduction studies have been performed in rats and rabbits at doses up to 160 times the human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Zantac. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers: Zantac is secreted in human milk. Caution should be exercised when Zantac is administered to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established

Use in Elderly Patients: Ulcer healing rates in elderly patients (65 to 82 years of age) were no different from those in younger age groups. The incidence rates for adverse events and laboratory abnormalities were also not different from those seen in other age groups

ADVERSE REACTIONS: The following have been reported as events in clinical trials or in the routine management of patients treated with Zantac®. The relationship to Zantac therapy has been unclear in many cases. Headache, sometimes severe, seems to be related to Zantac administration.

Central Nervous System: Rarely, malaise, dizziness, somnolence, insomnia, and vertigo. Rare cases of reversible mental confusion, agitation, depression, and hallucinations have been reported, predominantly in severely ill elderly patients. Rare cases of reversible blurred vision suggestive of a change in accommodation have been reported

Cardiovascular: As with other H_2-blockers, rare reports of arrhythmias such as tachycardia, bradycardia, atrioventricular block, and premature ventricular beats.

Gastrointestinal: Constipation, diarrhea, nausea/vomiting, abdominal discomfort/pain, and rare reports of pancreatitis.

Hepatic: In normal volunteers, SGPT values were increased to at least twice the pretreatment levels in 6 of 12 subjects receiving 100 mg qid intravenously for seven days, and in 4 of 24 subjects receiving 50 mg qid intravenously for five days. With oral administration there have been occasional reports of hepatitis, hepatocellular or hepatocanalicular or mixed, with or without jaundice. These were usually reversible.

Zantac® 150 and 300 (ranitidine hydrochloride) Tablets
Zantac® (ranitidine hydrochloride) Syrup

Musculoskeletal: Rare reports of arthralgias.

Hematologic: Blood count changes (leukopenia, granulocytopenia, thrombocytopenia) have occurred in a few patients. These were usually reversible. Rare cases of agranulocytosis, pancytopenia, sometimes with marrow hypoplasia, and aplastic anemia have been reported.

Endocrine: Controlled studies in animals and man have shown no stimulation of any pituitary hormone by Zantac and no antiandrogenic activity, and cimetidine-induced gynecomastia and impotence in hypersecretory patients have resolved when Zantac has been substituted. However, occasional cases of gynecomastia, impotence, and loss of libido have been reported in male patients receiving Zantac, but the incidence did not differ from that in the general population.

Integumentary: Rash, including rare cases suggestive of mild erythema multiforme, and, rarely, alopecia

Other: Rare cases of hypersensitivity reactions (eg, bronchospasm, fever, rash, eosinophilia), anaphylaxis, angioneurotic edema, and small increases in serum creatinine.

OVERDOSAGE: Information concerning possible overdosage and its treatment appears in the full prescribing information.

DOSAGE AND ADMINISTRATION: (See complete prescribing information in Zantac® product labeling.)

Active Duodenal Ulcer: The current recommended adult oral dosage is 150 mg or 10 ml (2 teaspoonfuls equivalent to 150 mg of ranitidine) twice daily. An alternate dosage of 300 mg or 20 ml (4 teaspoonfuls equivalent to 300 mg of ranitidine) once daily at bedtime can be used for patients in whom dosing convenience is important. The advantages of one treatment regimen compared to the other in a particular patient population have yet to be demonstrated

Maintenance Therapy: The current recommended adult oral dosage is 150 mg or 10 ml (2 teaspoonfuls equivalent to 150 mg of ranitidine) at bedtime.

Pathological Hypersecretory Conditions (such as Zollinger-Ellison syndrome): The current recommended adult oral dosage is 150 mg or 10 ml (2 teaspoonfuls equivalent to 150 mg of ranitidine) twice a day. In some patients it may be necessary to administer Zantac® 150-mg doses more frequently. Doses should be adjusted to individual patient needs, and should continue as long as clinically indicated. Doses up to 6 g/d have been employed in patients with severe disease.

Benign Gastric Ulcer: The current recommended adult oral dosage is 150 mg or 10 ml (2 teaspoonfuls equivalent to 150 mg of ranitidine) twice a day.

GERD: The current recommended adult oral dosage is 150 mg or 10 ml (2 teaspoonfuls equivalent to 150 mg of ranitidine) twice a day.

Dosage Adjustment for Patients with Impaired Renal Function: On the basis of experience with a group of subjects with severely impaired renal function treated with Zantac, the recommended dosage in patients with a creatinine clearance less than 50 ml/min is 150 mg or 10 ml (equivalent to 150 mg of ranitidine) every 24 hours. Should the patient's condition require, the frequency of dosing may be increased to every 12 hours or even further with caution. Hemodialysis reduces the level of circulating ranitidine. Ideally, the dosage schedule should be adjusted so that the timing of a scheduled dose coincides with the end of hemodialysis.

HOW SUPPLIED: Zantac® 300 Tablets (ranitidine hydrochloride equivalent to 300 mg of ranitidine) are yellow, capsule-shaped tablets embossed with "ZANTAC 300" on one side and "Glaxo" on the other. They are available in bottles of 30 (NDC 0173-0393-40) tablets and unit dose packs of 100 (NDC 0173-0393-47) tablets.

Zantac® 150 Tablets (ranitidine hydrochloride equivalent to 150 mg of ranitidine) are white tablets embossed with "ZANTAC 150" on one side and "Glaxo" on the other. They are available in bottles of 60 (NDC 0173-0344-42) and 100 (NDC 0173-0344-09) tablets and unit dose packs of 100 (NDC 0173-0344-47) tablets.

Store between 15° and 30° C (59° and 86° F) in a dry place. Protect from light. Replace cap securely after each opening.

Zantac® Syrup, a clear, peppermint-flavored liquid, contains 16.8 mg of ranitidine hydrochloride equivalent to 15 mg of ranitidine per 1 ml in bottles of 16 fluid ounces (one pint) (NDC 0173-0383-54).

Store between 4° and 25° C (39° and 77° F). Dispense in tight, light-resistant containers as defined in the USP/NF.

January 1990

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DIVISION OF GLAXO INC.
Research Triangle Park, NC 27709

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ZAN325R

Printed in USA

September 1990

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PHYSICIANS WANTED—CONT'D

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MISCELLANEOUS

PROFESSIONAL CONDUCT EXPERT. Robert S. Asher, J.D., M.P.A., in health, former Director Professional Conduct, N.Y.S. Board of Regents, now in private legal practice. 15 years health law experience concentrating on professional practice, representation before government agencies on Disciplinary, Licensure, Narcotic Control, Medicaid, Medicare of Third-Party Reimbursement matters and professional business practice. Robert S. Asher, Esq., 295 Madison Ave., Suite 700, NYC. (212) 697-2950 or evenings (914) 723-0799.

WRITERS' SYMPOSIUM FOR PHYSICIANS—October 27, 1990, American Society of Journalists and Authors (ASJA) and NYU Medical Center present the third annual Writers' Symposium for Physicians. How to communicate with the lay public through books, magazines, and other media. How to find and work with co-authors agents, editors. The program features three panels: Communicating Through Magazines and Newspapers; How To Sell Your Book; and Collaboration: Working With Your Co-Author. Panelists include Gina Kolata, Science and Medical Reporter for The New York Times; Edward Edelson, Science Editor, New York Daily News; Carol Cohen, Vice President and Associate Publisher, Harper & Row; literary agent Barbara Lowenstein, and others. Special luncheon speaker: Susan Hester, Associate Producer, Booking, ABC's "Good Morning America." Tuition (which includes lunch): $140 for reservations received by Oct. 12th; $150 for those received by Oct. 26th; $175 at door. For information, contact: ASJA, 1501 Broadway, Suite 1907, New York, N. Y. 10036. (212) 997-0947.

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AIR FORCE

(*continued from p 16A*)

tional Body Imaging Congress, c/o Medical Seminars International, Inc, 9800 D Topanga Canyon Blvd, Suite 232, Chatsworth, CA 91311. Tel: (818) 700-9821.

NEBRASKA

Oct 1–6. **Emergency Medicine Review.** Lincoln. Contact: Continuing Education, University of Nebraska Medical Center, 42nd and Dewey Ave, Omaha, NE 68105. Tel: (402) 559-4152.

NEW MEXICO

Oct 19–21. **Annual Scientific Meeting of the Western Neurological Society.** Eldorado Clarion Hotel, Santa Fe. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

RHODE ISLAND

Oct 8–12. **16th Annual Education Conference of the American Association of Pathologists' Assistants.** Providence Marriott, Providence. Contact: Doris Schuler-Mahoney, MS, Mercy Hospital Medical Center, Department of Pathology, 6th and University, Des Moines, IA 50314. Tel: (515) 247-4478.

TEXAS

Oct 6–8. **AAFP Congress of Delegates.** Loews Anatole, Dallas. *Oct 7–11.* **AAFP Annual Scientific Assembly.** Loews Anatole, Dallas. Contact Beth Paulsen, Manager, Public Relations Department, The American Academy of Family Physicians, 8880 Ward Pkwy, Kansas City, MO. Tel: (800) 274-2237, ext 4220.

AROUND THE WORLD

CANADA

Oct 14–16. **Canadian Congress on Utilization Management.** The Westin Hotel, Edmonton, Alberta. Contact: Canadian College of Health Service Executives, 201,17 York St, Ottawa, Ontario K1N5S7. Tel: (613) 235-7218.

FRANCE

Oct 29–Nov 3. **Fourth U.S. Phlebology Workshop in Paris.** Paris, France. Contact: Walter de Groot, MD, 801 Broadway, #401, Seattle, WA 98122. Tel: (206) 292-6265. □

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MISCELLANEOUS—CONT'D

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DECEMBER 3–5, 1990: 25th Anniversary Program: Interpretation and Treatment of Cardiac Arrhythmias. Philadelphia, Pennsylvania. Sponsored by the American College of Cardiology and Likoff Cardiovascular Institute, Hahnemann University. 22 credit hours in Category 1. Inquiries: Registration Secretary, Extramural Programs Department, American College of Cardiology, 9111 Old Georgetown Rd., Bethesda, MD 20814. Tel. 1-800-253-4636, ext. 228. FAX 301-897-9745.

DECEMBER 14–16, 1990: Changes, Choices, Challenges in the Management of Cardiovascular Disease. New York, New York. Sponsored by the American College of Cardiology. 19.5 credit hours in Category 1. Inquiries: Registration Secretary, Extramural Programs Department, American College of Cardiology, 9111 Old Georgetown Rd., Bethesda, MD 20814. Tel. 1-800-253-4636, ext. 228. FAX 301-897-9745.

PROFESSIONAL MISCONDUCT ADVOCACY with dignity and concern. George Weinbaum, Esq. former deputy director of the New York State Attorney General's office for Medicaid Fraud Control and also former Director of Medicare and private insurance investigations for Empire Blue Cross/Blue Shield is pleased to announce the opening of his office for the practice of health law. Attention is devoted to misconduct avoidance, disciplinary proceedings, reimbursement maximization, controlled drug issues and medical staff representation among other physician needs. George Weinbaum, Esq., 3 Barker Avenue, White Plains, New York 10601. Telephone (914) 686-9310 office hours, (212) 621-7776-24 hours.

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MISCELLANEOUS—CONT'D

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Index to Advertisers

Burroughs Wellcome 9A, 10A, 11A, 12A, 13A

Classified Advertising 4A, 20A, 21A, 22A

Glaxo Pharmaceuticals 17A, 18A

Knoll Pharmaceuticals 3A, 4A

Eli Lilly and Company 7A

Medical Directors Advancement Council 5A

Med Pro .... 2nd Cover

Palisades Pharmaceuticals 16A

Roche Laboratories .... 3rd & 4th Cover

Searle Pharmaceuticals 1A

Self-Marketing Management 19A

U.S. Air Force 21A

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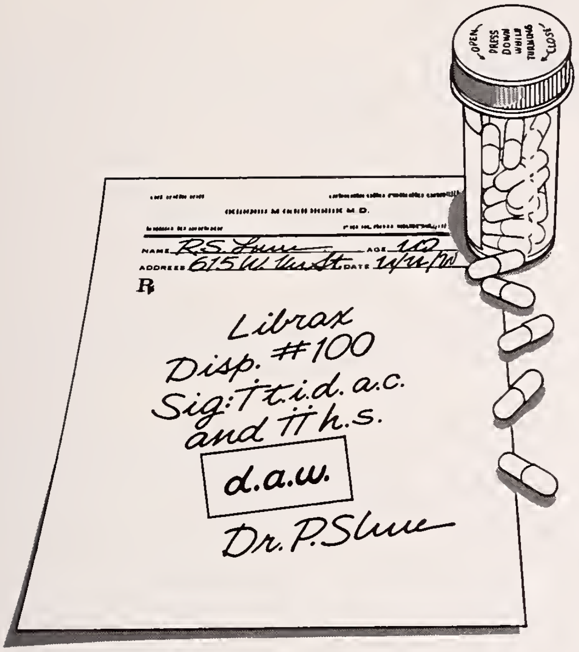

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As with all anticholinergics, inhibition of lactation may occur.

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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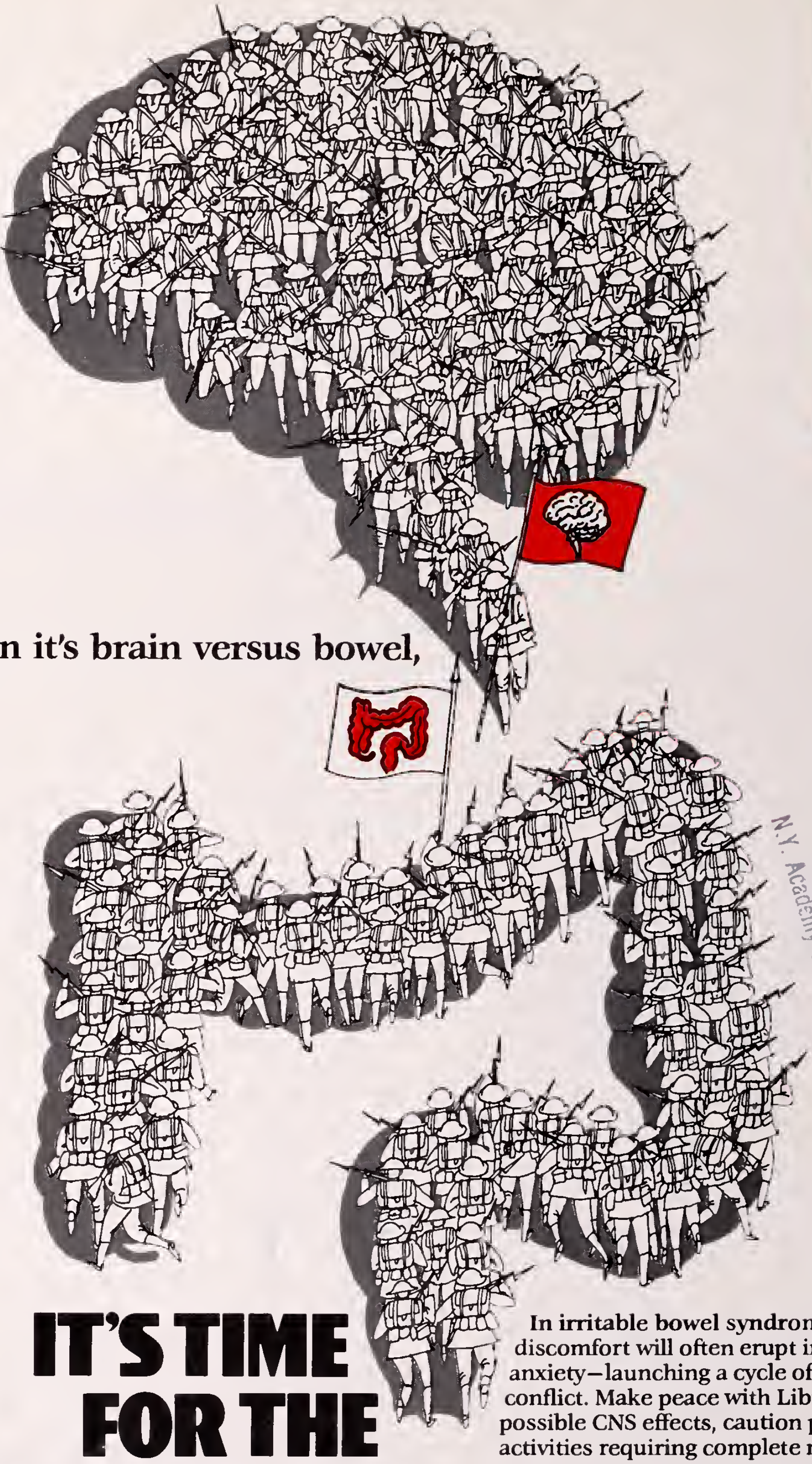

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VOL 90
NO 10
OCTOBER
1990

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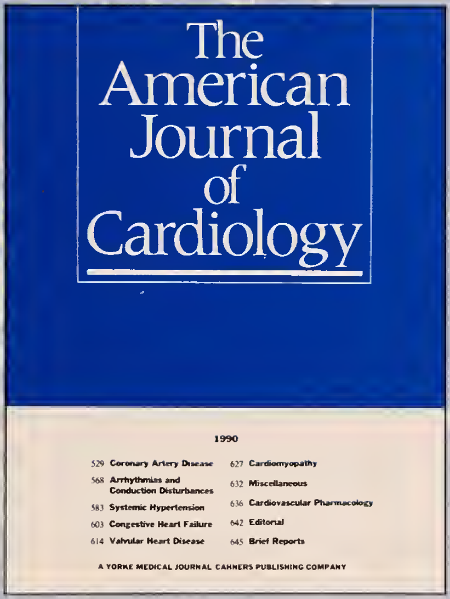

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529 Coronary Artery Disease
568 Arrhythmias and Conduction Disturbances
583 Systemic Hypertension
603 Congestive Heart Failure
614 Valvular Heart Disease
627 Cardiomyopathy
632 Miscellaneous
636 Cardiovascular Pharmacology
642 Editorial
645 Brief Reports

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New York State Journal of Medicine

OCTOBER 1990 / Volume 90, Number 10

Contents

COMMENTARIES

485 **The rising incidence of congenital syphilis: Back to the future**
LAURENE MASCOLA, MD, MPH

487 **Crack cocaine: Ripples of a rock in a pond**
KENNETH J. KRAJEWSKI, MD

RESEARCH PAPERS

488 **Syphilis among parturients at an inner city hospital: Association with cocaine use and implications for congenital syphilis rates**
DEEPAK NANDA, MD; JOSEPH FELDMAN, DrPH; ISAAC DELKE, MD; SHOBHA CHINTALPALLY, MD; HOWARD MINKOFF, MD

491 **The prevalence of marijuana (cannabis) use and dependence in cocaine dependence**
NORMAN S. MILLER, MD; ARYEH L. KLAHR, MD; MARK S. GOLD, MD; KENNETH SWEENEY; JAMES A. COCORES, MD

REVIEW ARTICLE

493 **The public health consequences of crack cocaine**
LaCLAIRE GREEN BOUKNIGHT, MD

POSITION PAPER

496 **Position paper of the Associated Medical Schools of New York on HIV testing and access to treatment**

HISTORY

499 **Gas warfare: Military-medical responsiveness of the Allies in the Great War, 1914–1918**
JOHN S. HALLER, JR, PhD

(*continued on page 6A*)

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1. Brantly ML, Paul LD, Miller BH, et al: Clinical features and history of the destructive lung disease associated with alpha-1-antitrypsin deficiency of adults with pulmonary symptoms. *Am Rev Respir Dis* 1988;138:327-336.
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Journal of Medicine

CASE REPORTS

511 **Crack abuse and asthma: A fatal combination**
A. NAVEEN RAO, MD; PETER G. POLOS, MD, PhD; FREDERICK A. WALTHER, MD

512 **Eosinophilia-myalgia syndrome and L-tryptophan**
RICHARD L. GOLDEN, MD

513 **Neurocutaneous manifestations of neurofibromatosis-2**
EMILIO D. SORIA, MD; EDWARD J. FINE, MD; MICHAEL M. CANDARAS, MD

LETTERS TO THE EDITOR

517 **Defining brain death**
PETER ROGATZ, MD

517 **Trade and generic names of drugs**
GERTIE F. MARX, MD

517 **A neglected resource**
RANDALL D. BLOOMFIELD, MD

518 **Reversible hypoxic cerebral edema in severe hemolytic crisis**
WILLIAM J. DAVID, MD; PREMLATA WADHERA, MD; SUDHA NARASIMHAN, MD; PARTHAS NARASIMHAN, MD

519 LEADS FROM EPIDEMIOLOGY NOTES

523 BOOK REVIEWS

526 NEWS BRIEFS

528 OBITUARIES

530 GUIDELINES FOR AUTHORS

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References
1. USP DI Update, September/October 1988, p 120.
2. Br J Clin Pharmacol 1985;20:710-713.
3. Data on file, Lilly Research Laboratories.
4. Scand J Gastroenterol 1987;22(suppl 136):61-70.
5. Am J Gastroenterol 1989;84:769-774.

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Carcinogenesis, Mutagenesis, Impairment of Fertility—A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

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In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of varying durations included almost 5,000 patients. Among the more common adverse events in domestic placebo-controlled trials of over 1,900 nizatidine patients and over 1,300 on placebo, sweating (1% vs 0.2%), urticaria (0.5% vs <0.01%), and somnolence (2.4% vs 1.3%) were significantly more common with nizatidine. It was not possible to determine whether a variety of less common events was due to the drug.

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Hepatic—Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to three times the upper limit of normal, however, did not significantly differ from that in placebo patients. Hepatitis and jaundice have been reported. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS—Rare cases of reversible mental confusion have been reported.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with equal frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic—Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental—Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity—As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Because cross-sensitivity among this class has been observed, H_2-receptor antagonists should not be administered to those with a history of hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance by approximately 84%.

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NOVEMBER 1990

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(*continued on p 13A*)

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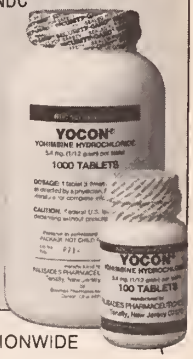

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New York State
Journal of Medicine

COMMENTARIES

The rising incidence of congenital syphilis: Back to the future

It will probably come as no surprise, especially to the medical practitioners of the State of New York, that one of the 11 objectives established for the nation in 1979 for the year 1990 is unlikely to be met. That objective was as follows: "By 1990, the reported incidence of primary and secondary syphilis should be reduced to a rate of seven cases per 100,000 population per year, with a reduction in congenital syphilis cases to 1.5 cases per 100,000 children under one year of age."[1] As the article by Nanda et al[2] in this issue of the *Journal* highlights, congenital syphilis is on the rise at their inner New York City hospital, with rates of positive cord rapid plasma reagin tests increasing more than threefold from 1985 to 1988. A more extensive review of congenital syphilis cases occurring in all of New York City reported a fivefold increase of reported congenital syphilis cases from 1986 to 1988.[3] No health department wants to admit that cases of congenital syphilis are occurring in its locality since its occurrence is a sentinel health event marking the failure of both the syphilis control program and the prenatal care system.[4] It is also a marker for drug abuse and poverty.

The State of New York is not alone in its congenital syphilis problem. Recent papers by Ricci[5] in Miami and Cohen[6] in Los Angeles also describe tragic rises in incidence. A recent phenomenon, the precipitous rise of "crack" cocaine use, appears to be associated with a concurrent increase in cases of congenital syphilis. As described by Nanda and his group at the local hospital level[1] and at the city-wide level by the New York City Health Department,[2] women who deliver congenitally affected infants are more likely to have used drugs (crack/cocaine) during their pregnancy and less likely to have received prenatal care. Both of these factors contribute substantially to pregnant women with syphilis receiving inadequate or no therapy for their disease.

It would be tempting to place all the blame on crack. Crack, a smokable form of cocaine, has been reported to acutely enhance sexual desire, unlike other addictive drugs. Accordingly, the number of mothers who use crack/cocaine in New York City has increased from 1/1,000 live births in 1981 to 21/1,000 live births in 1988.[3] Moreover, because of limited employment opportunity, lack of education, and limited access to other established channels for obtaining income, women increasingly exchange sex for drugs. These interactions are frequent as the crack highs are intense and short lasting. In fact, a whole new set of terminology for interactions such as these has been developed. Women who exchange sex for drugs are degradingly called "tossups." One man, when asked to define "tossup," said, "It's just that, you know, you use her and then you just toss her to your buddy or the next guy."[7] Obviously these frequent sexual encounters are behaviors that increase women's risks of becoming pregnant and contracting syphilis.

If one were to develop a formula to describe factors contributing to the recent rise of congenital syphilis, *Treponema pallidum* would not appear as the sole cause of this recent epidemic. Neither would the use of crack/cocaine. Both of these major "players" would be surrounded by a web of societal, economic, and demographic factors, such as the rising rates of teenage pregnancy, a changing urban environment, and the higher incidence of sexually transmitted diseases (STDs) in minority populations.[8] The widespread use of crack makes it more likely that inner city young women will continue to engage in high risk behaviors that will cause them to be infected not only with syphilis but with the human immunodeficiency virus (HIV) as well. A recent study has established links between intravenous cocaine use and retroviral (HIV-1) infection.[9]

Addressing these undercurrent issues requires a long term approach and sufficient political resolve. However, what aspects of this epidemic do we as health care practitioners have control over and what solutions can we offer to alleviate this problem? First, we must screen drug abusing women for syphilis, especially those who are pregnant. As demonstrated by Nanda et al,[2] pregnant, drug-abusing women do not seek prenatal care. However, a recent Los Angeles study demonstrated that women who might not seek prenatal care often access the local health care system to find out if they are pregnant (D. Cohen, personal communication, April 1990). Therefore, all women should be screened for syphilis at pregnancy test clinics.

Obstacles to prenatal care, such as financial barriers and long waiting times, should be removed for all women, addicted and nonaddicted. Providing evening clinics to pregnant women who might be working during the day would be one such approach. A mobile van providing prenatal care and other services in high risk areas would be another. If we are successful, women will enter the prenatal care system early enough to allow the screening for syphilis in the first trimester and subsequent rescreening for syphilis in the third trimester.

We must provide pregnant addicts with priority access to drug treatment programs. Communities do not have enough drug treatment centers to deal with the crack epidemic. A recent survey of 78 New York treatment programs in 1989 found that 54% excluded pregnant women, 67% excluded those on Medicaid, and 87% excluded crack addicts.[10] While we are trying to provide more drug treatment slots for these women, we also need to teach women, especially those addicted to crack, how to have safe sex. How about outreach programs to specific high risk groups to deliver such health aids as contraceptives or condoms? Former addicts could be trained and used as community workers, encouraging women to practice safe sex.

A recent development illustrating some groups' frustrations in dealing with this escalating drug abuse problem in pregnancy has been the attempt of some advocates to indict women who use crack on child abuse charges.[11] I believe that we cannot create the fear of criminal prosecution in women who use drugs during pregnancy. This would provide an added reason for addicted women to avoid medical and prenatal care and to conceal their drug histories from medical providers.

What we really need to do is to empower women, especially those of lower socioeconomic status, to take control of their lives. Education against crack use must be introduced early in our children's educative experience. A recent poll by the National Association of Elementary School Principals found that 58% of schools began drug education in kindergarten, 30% by third grade and 12% between fourth and sixth grades.[12] In addition, safe sex education must be included early in the curriculum. It is unrealistic, however, to expect teenagers who are high on crack to practice safe sex. Somewhere in this massive wish list, our society has to eliminate crack/cocaine.

Except for the acquired immunodeficiency syndrome (AIDS), syphilis is the most damaging disease sexually transmitted to the fetus. In the third edition of *Modern Clinical Syphilology*, published in 1944, 46 years ago, the authors state that the "proper treatment of the syphilitic pregnant woman could virtually eliminate from practical consideration the whole field of congenital syphilis."[13] They also state that "it is hard to find anywhere in present-day medicine, a more effective demonstration of disease prevention or in public health, a field more likely to show dramatic satisfactory results of the reduction of disease incidence in prevalence as in the field of congenital syphilis."[13]

If I were a lawyer, I would put society and health departments on trial for allowing the escalation of congenital syphilis cases in the era of penicillin. I would tell the jury of the magnitude of the offense, of the high case-fatality rate, and of the misery and suffering caused. But I am just an epidemiologist; I can only manipulate numbers, summarize data, and provide hypotheses and discussions. We need to react to the numbers provided by Nanda et al[2] and other researchers, and our actions have to supplement traditional resources used for disease and drug abuse control. We need to enroll the private medical community and we need to motivate people as they have never been motivated before. We can no longer wait passively for this epidemic to disappear; the time to act is now.

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Crack cocaine: Ripples of a rock in a pond

A review of my medical school pharmacology notes, taken during the early 1970s, reveals that I was taught, "Cocaine is an illegal drug, is a sympathomimetic like an amphetamine, and is not addicting; however, for some users, there is a risk of psychological addiction. Its use is limited to people such as musicians and artists. You probably won't see much of it. Next drug . . . ."

By the beginning of the 1980s, epidemiologic evidence indicated that cocaine use had reached a plateau from its previous significant upsurge in use. However, during this period our university-based alcohol treatment program split into an alcohol service and a drug abuse service, and my department chairman asked me to direct a four-bed inpatient sub-program of the alcohol treatment service. The chairman, a man of wisdom and foresight, expressed his belief that medical students and residents would someday benefit from the training and education obtained from this clinical experience. Seemingly overnight, outpatients' chief complaints became cocaine: free base, rock, crack. The university's nonprofit inpatient substance abuse beds number 46, with a waiting list of more than 200 potential patients.

As a rock tossed into a pond produces ripples that spread until they reach the banks, the effects of crack cocaine use have penetrated all biopsychosocial aspects of society, including medical practice.

Alcohol and marijuana have often been considered "gateway" drugs to "harder" substances. It is common knowledge among those specializing in substance abuse treatment that at least 75% of crack users have smoked marijuana more than 100 times.[1] Illegal drug traffickers first introduced crack in marijuana cigarettes, called "primo." In time, crack replaced marijuana as a drug of choice because, as some patients commented, crack was easier to buy than marijuana, and pot dealers gradually changed their product lines exclusively to crack.

The case of an asthmatic patient succumbing to substance abuse, and described in this issue of the *Journal* by Rao et al, exemplifies the pervasive nature of this societal phenomenon.[2]

On admission to the emergency room, the patient presented a history of both asthma and crack use. Left unanswered is whether the patient's clinical history of chronic asthma had been affected by gateway drugs prior to his initial presentation to the emergency room. If the patient were a "typical" crack user, it is likely that he had abused other substances before his first use of crack. An important clinical question, though only speculation, is whether diagnosis of a substance abuse disorder and effective intervention would have altered the patient's clinical course. Should clinicians who are managing an asthmatic on an outpatient basis order a urine drug screen with the same regularity as a complete blood count, urinalysis, electrocardiogram, or chemical profile? Though the outpatient management of this patient is not included in and is not a focus of this discussion, that clinical history must dictate laboratory investigation remains the rule.

This case reported by Rao et al[2] also raises many disturbing, although scarcely investigated, possibilities of drug interactions. What are the significant clinical ramifications of interactions between cocaine and routinely employed asthma medications; between cocaine and cardiac medications or anesthesia? Is the cocaine abusing patient at a higher risk for even minor medical or surgical procedures?

Perhaps had the patient's cardiac or pulmonary pathology been greater, hospital admission would have been warranted, thus protecting him from his behavioral pathology, behavior that resulted in his terminal presentation to the emergency room.

This case report adds to the growing body of knowledge of the effects of cocaine on medical disorders. The ripples of cocaine use have affected or produced psychiatric, cardiac, pulmonary, neurological, renal, gastrointestinal, obstetrical-gynecological, infectious, and surgical disorders.[3]

Clearly, clinical investigation (history, physical examination, and laboratory data) and medical treatment of substance abuse disorders are called for. The patient's history of a substance abuse disorder is a significant clinical finding, and substance use cannot be easily dismissed as within normal limits without further exploration.

Substance abuse disorders are a chronic, progressive disease—a primary disease—and not a symptom of an underlying emotional or physical problem. A generation of American history and the case report of a cocaine-dependent patient who was also an asthmatic attest to this fact.

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Syphilis among parturients at an inner city hospital: Association with cocaine use and implications for congenital syphilis rates

DEEPAK NANDA, MD; JOSEPH FELDMAN, DrPH; ISAAC DELKE, MD; SHOBHA CHINTALAPALLY, MD; HOWARD MINKOFF, MD

ABSTRACT. The frequency of positive cord blood rapid plasma reagin (RPR) tests among newborns at an inner city hospital and associations with maternal cocaine use, prenatal care, and adequacy of syphilis therapy were retrospectively assessed. The incidence of positive cord blood RPRs increased from 1.1% of all live births in 1985 to 3.4% in 1988. In 1987, 98 babies were born with positive cord blood RPRs; 86 of their charts were available for review. Four infants had false positive RPRs, and one patient delivered twins, leaving 81 mothers who could be evaluated. Almost 37% of these patients had had no prenatal care. More than 55% had inadequate or no therapy for syphilis. Of these, only 17.4% had prenatal care. Slightly more than 40% of patients acknowledged using drugs during pregnancy, 87.9% of whom used cocaine. Among the patients who used drugs, 75.8% received no prenatal care, in contrast to 10.4% of mothers who did not use drugs ($p < 0.001$). It appears that drug use, particularly use of cocaine, is associated with low levels of utilization of prenatal services and inadequate therapy for syphilis. This may lead to increased risk of congenital syphilis in newborns.

(NY State J Med 1990; 90:488–490)

Syphilis continues to grow as a public health problem despite national efforts to control its spread. Although syphilis is easily diagnosed and treated, recent reports have documented a progressive increase in its incidence, particularly among minority women.[1-3]

The increased incidence of syphilis among women of child-bearing age has been reflected in a concurrent rise in the incidence of congenital syphilis. The number of reported cases of congenital syphilis in the United States increased four-fold between 1985 and 1987,[2] with 681 cases being reported to the Centers for Disease Control (CDC) in 1987. Reports of congenital syphilis have appeared from many other parts of the industrialized world.[4,5]

Congenital syphilis is linked to factors associated with both sexually transmitted diseases and underutilization of medical care services by women. Among these factors are youth, race (black), and residence in areas endemic for congenital syphilis such as New York City, Florida, Texas, and California.[3] Drug use may also be linked to syphilis in women, and the CDC has made determination of the relationship of syphilis to illegal drug use a research priority.[6]

Many patients receiving care at the Kings County Hospital Center (KCHC), a 1,200-bed municipal hospital in Brooklyn, NY, are poor minority women who receive late or no prenatal care. A high incidence of "crack" (alkaloidal cocaine) use among women with no prenatal care has recently been documented in this population.[7] Accordingly, we hypothesized a high prevalence of syphilis in pregnant women as well as a high risk for congenital syphilis among newborns at KCHC, and we undertook a preliminary evaluation of the association between illicit drug use and syphilis as suggested by the CDC.

The objectives of this cross-sectional study were to estimate the prevalence of syphilis among parturients at an inner city hospital, to determine how often syphilis had been adequately treated, and, most importantly, to ascertain the relationship between syphilis and the use of cocaine. Finally, the utilization of prenatal care services by patients with syphilis was evaluated and an attempt was made to determine the implications for congenital syphilis rates.

PATIENTS AND METHODS

A cross-sectional study of the medical records of live born infants with positive cord blood rapid plasma reagin (RPR) tests was done to estimate the prevalence of congenital syphilis. All the infants were born at Kings County Hospital in 1987 (the last year from which most charts were available at the time the study was undertaken). Since cord blood RPR tests detect antibodies that have crossed the placenta, and these tests are routinely obtained on all patients in labor, they were used as surrogate markers of syphilis among parturients. It should be noted that some infants with negative serologic results may have mothers with positive RPRs who would be missed with this entry criteria (ie, false negatives). Only cord blood, not mother's blood, however, was routinely sent at the time of delivery for RPR tests during the time of the study. The mothers' charts were reviewed for the prenatal course, drug use history, and adequacy of maternal syphilis prenatal treatment. Infants with congenital syphilis were classified according to the CDC criteria.

All positive RPR tests were confirmed by a fluorescent trepo-

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nema antibody absorption test (FTA-ABS). All mothers who received treatment less than that recommended by the CDC for syphilis in pregnancy—single-dose treatment in mothers with syphilis of unknown duration[8]—were considered to have received inadequate care, and their children therefore fulfilled the criteria for CDC category 2, "compatible case of syphilis."

The association of intravenous and crack drug use and syphilis was determined by a case controlled comparison in which drug use among controls was estimated from medical records. It is policy to inquire routinely about drug use at the time of delivery. Similarly, among mothers with syphilis, drug use was determined by history at the time of admission. Among both case and control patients, in instances in which a negative history was obtained but drug use was strongly suspected (eg, a symptomatic newborn), urine toxicology studies were done.

The number of children born with positive cord blood RPRs in each year from 1985 to 1988 was examined to assess recent trends. Data on stillbirths were collected, but the information available was incomplete, and as a result they were not included in the analysis. Although five mothers who had stillbirths did have positive serologies, there was no way to confirm if syphilis was the cause of stillbirth.

All statistical tests were exactly estimated for sample size less than 40. The Wilcoxan rank sum test adjusting for ties was used to assess the significance of the trend in syphilis over time. The Fisher exact test was used to assess the significance of contingency tables. Odds ratios were computed based on hypersegmentric probabilities. The actual computations were done on a microcomputer using StatXact (statistical software for exact nonparametric inference; Cytel Software Corp, Cambridge, Mass).

RESULTS

The number of live births at the study site between 1985 and 1988 and the percentage with positive cord RPRs is shown in Figure 1. There was an increasing trend in the percentage of positive cord RPRs from 1.1% in 1985 to 3.4% in 1988 ($p < 0.001$, Wilcoxan rank sum test). In 1987 (the study year), 98 infants were born with positive cord serologic findings. Medical records were available for review for 86 patients. Of these, four patients had negative fluorescent titer antibody tests and were excluded from analysis. One mother had delivered twins, leaving 81 mothers and 82 babies for final analysis.

Of the 81 mothers, 30 (36.58%) had no prenatal care, and 38 (46.34%) had had at least one prenatal visit, either at KCHC, its affiliated satellite institution, or at an unaffiliated institution. In the remaining 13 cases (17.07%), information on care status was not available from the charts.

In the case comparison regarding drug use, 33 of the 81 study patients (40%) acknowledged using drugs during pregnancy (crack cocaine/intravenous drugs) compared to 6% of the 4,920 patients who delivered at KCHC in 1987 ($p < 0.001$). Table I shows that the likelihood of a mother with positive RPR using drugs was more than ten times greater than for other mothers. Twenty-nine of 33 (87.9%) mothers who acknowledged drug use used crack or other forms of cocaine, and four patients admitted to current or past intravenous drug use.

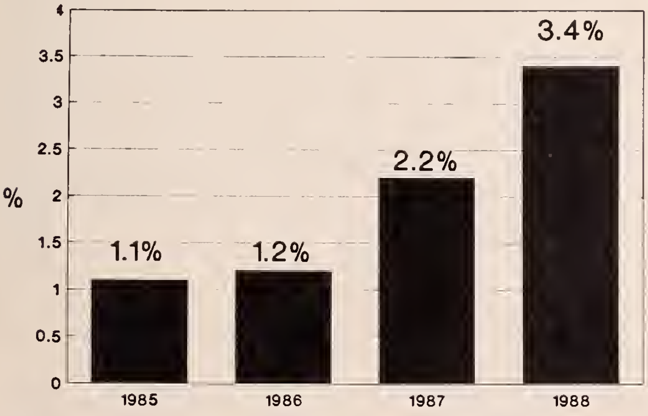

FIGURE 1. Incidence of positive cord serologies in infants, Kings County Hospital Center, New York City.

TABLE I. Mothers with Positive Rapid Plasma Reagin Test in Association with Drug Use, Kings County Hospital Center, New York City, 1987

Drug Use	RPR Positive Number	RPR Positive (%)	Control Number	Control (%)	Odds Ratio	95% Confidence Interval
Yes	33	40.2	295	6.0	10.8	6.8–17.1
No	48	59.8	4,625	94.0		
Total	81		4,920			

Among the patients who admitted to using drugs (N = 33), 25 (75.8%) received no prenatal care, compared to only 10% of 48 mothers who did not use drugs ($p < 0.001$, Table II). A non-drug user was more than 30 times (95% confidence interval, 8.2–122.9) more likely to have received prenatal care than a drug user. Even the minimal estimate showed an eight-fold difference in likelihood of prenatal care between drug users and nonusers. Similarly, drug usage was four times more likely among parturients (N = 38) with either no therapy or inadequate therapy for syphilis ($p < 0.001$, Table II), compared to the 31 patients with adequate therapy (therapy described below). All 24 of the drug-using patients with inadequately treated syphilis admitted to using crack or other forms of cocaine during pregnancy. Among parturients with no prenatal care whose infants' diagnosis was "compatible with congenital syphilis," 80% admitted to using drugs during pregnancy.

Adequate information was available from charts to assess treatment in 69 cases. Of these, 38 mothers (39 infants) received either no therapy or inadequate therapy. Using these mothers as a minimal estimate, at least 0.8% (38/4,920) of all infants born at KCHC in 1987 were born with the potential of having congenital syphilis due to inadequate maternal treatment. Twenty-six of the 38 mothers had positive serologic findings with no prenatal care or treatment, while the other 12 patients had some prenatal care but received inadequate or suboptimal treatment. Five of these 12 had syphilis of indeterminate duration and had received inadequate therapy (one dose of bezathine penicillin). These five patients had had only one prenatal visit late in the third trimester. An additional patient was treated with oral erythromycin because of penicillin allergy. Of the remaining six patients, two had non-reactive RPRs in clinic, were not retested later in pregnancy, and had positive tests at the time of delivery; two had rising titers despite treatment; one had prenatal care at another institution but had no history of syphilis or treatment during pregnancy; and one failed to register a drop in titer despite treatment in the second trimester and delivered an infant with a maculopapular rash. For thirty-one (44.9%) infants, the available data suggested that the mother had received adequate therapy.

TABLE II. Effect of Drug Use, Prenatal Care, and Maternal Treatment for Syphilis, Kings County Hospital Center, New York City, 1987

Prenatal Care	Drug Use (N)	Drug Use (%)	No Drug Use (N)	No Drug Use (%)	Total	Odds Ratio	95% Confidence Interval
Yes	5	15.2	32	66.7	37	32.0	8.2–122.9
No	25	75.8	5	10.4	30		
Treatment for Syphilis							
Yes	9	29	22	71	31	4.2	1.5–11.8
No	24	63.2	14	36.8	38		

The 39 children of the 38 mothers with inadequate or no therapy fulfill CDC criteria for cases "compatible with congenital syphilis" (a reactive serologic test for syphilis in an infant whose mother had syphilis during pregnancy and was not adequately treated regardless of symptoms in the infant). Eight of the 39 infants with a diagnosis "compatible with congenital syphilis" had syphilitic stigmata (three had maculopapular rash, one had keratitis which in the absence of other possible etiologic causes was presumed to be syphilitic, three had hepatosplenomegaly, and two had periostitis). No confirmed cases of congenital syphilis were reported, as dark-field examination and special tissue stains were not performed.

DISCUSSION

This study demonstrates a marked increase (>300%) in the incidence of positive cord blood tests for syphilis among infants born in an inner city hospital in New York City over the last four years. This has occurred at a time when several areas of the country are in the midst of an epidemic of primary and secondary syphilis.[1,3] Although the study design limits our ability to estimate a specific untreated, infectious syphilis rate (retrospective review with incomplete documentation of information), a large number of infected, untreated parturients have been noted. Excluding missing records and infected mothers of children with negative RPRs, it was still found that 0.8% of live births occurred among women with untreated or inadequately treated syphilis. This compares to a national incidence of syphilis of 0.015%.[6] Historically, each surge in the incidence of syphilis among women is succeeded by an increase in the incidence of congenital syphilis. Since confirmed cases of congenital syphilis are associated with perinatal mortality rates as high as 40%,[1] and since a rapid increase in the number of exposed infants is occurring, the data presented in this study suggest that attention and resources may need to focus once again on syphilis.

Previous studies have shown that a lack of prenatal care is linked to congenital syphilis.[9,10] One earlier study noted that 40% of patients who delivered babies with congenital syphilis had had no prenatal care,[9] and Mascola et al[10] reported that 62% of mothers of children with congenital syphilis in a Texas hospital had had no prenatal care. Sixty-eight percent of mothers whose children had a diagnosis "compatible with congenital syphilis" in the study presented here had had no prenatal care. Overall, among women whose children had positive cord blood RPRs, only 44.9% of the patients evaluated had received adequate treatment for syphilis.

Perhaps the most significant and worrisome finding is the association between the epidemic of congenital syphilis and the use of crack and other forms of cocaine which are becoming epidemic in the nation's inner cities. Previous studies have shown that 4% of parturients at KCHC acknowledge using crack cocaine.[7] In this study, 63.1% of mothers at the same institution whose infants had a diagnosis "compatible with congenital syphilis," admitted to the use of crack or other forms of cocaine. Most importantly, of those mothers who had had no prenatal care, 80.8% acknowledged using either crack or other forms of cocaine and since urine toxicologies were not routinely obtained, that number may understate the actual incidence of illicit drug use. Previous data from this institution have shown that 65% of parturients acknowledging crack use had had no prenatal care.[7] Therefore, it is reasonable to conclude that drug use is contributing to the failure to provide adequate therapy to pregnant women with syphilis.

It has also been suggested that crack use contributes to sexual behavior that increases the possibility of contracting syphilis. In January 1988, the CDC reported that anecdotal reports of interviews with prostitutes indicated that prostitution involving the exchange of sex for drugs might be linked to outbreaks of syphilis.[11] Thus, the increasing use of cocaine, with its dual effects on syphilis (increased acquisition, decreased care and opportunity for treatment), has ominous prognostic significance for congenital syphilis rates.

Twelve of the patients in this study had had prenatal care and still delivered infants who were classified as "compatible with congenital syphilis." As stated above, five patients had only one prenatal visit and received only one dose of benzathine penicillin just prior to delivery, which is not the recommended treatment regimen. Further, it has been reported that even adequate therapy late in the third trimester is associated with treatment failures.[12] Again, inadequate utilization of health care services contributed to the infants' risk. In the remaining cases, care providers' error may have contributed to the outcomes, since there was a failure to follow established re-screening protocols for syphilis in pregnancy.

However, even with the rigorous application of prenatal screening programs in early and late pregnancy, only a minority of cases of congenital syphilis will be prevented, since only a minority of infected mothers have prenatal care. Given the current increase in drug use, which reduces utilization of prenatal services and which may be associated with high risk behavior, a continued rise in the incidence of syphilis among parturients in New York City and an increasing risk of congenital syphilis among neonates may be anticipated.

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The prevalence of marijuana (cannabis) use and dependence in cocaine dependence

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ABSTRACT. In a retrospective and prospective study using DSM-III-R criteria for substance dependence in 232 inpatients and 51 outpatients, the clinical suspicion that cocaine addicts use other drugs including marijuana was confirmed. As many as 53% of cocaine addicts diagnosed by DSM-III-R criteria for cocaine dependence qualified for a diagnosis of cannabis dependence. The reports regarding marijuana and other drug dependence among cocaine addicts have been few and inconclusive. The diagnosis of other drug use and dependence in cocaine dependence has importance with regard to prognosis and treatment. This study found a high prevalence of marijuana (cannabis) dependence in patients with cocaine dependence.

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Cocaine addicts diagnosed by DSM-III-R criteria for cocaine dependence may qualify for other substance dependence diagnoses. The reports regarding marijuana (cannabis) and other drug dependence among cocaine addicts are few and inconclusive.[1–3] Moreover, the diagnosis of other substance use and dependence in cocaine dependence has an important impact on prognosis and treatment.[4]

Since most cocaine addicts use marijuana prior to initiating cocaine use, some may qualify for a DSM-III-R diagnosis of marijuana dependence prior to cocaine use. Few reports have provided data to confirm the prevalence of DSM-III-R diagnosis of marijuana dependence in cocaine addicts.[3] Any investigations into the etiology, natural history, and treatment of cocaine use and dependence and related consequences may be severely handicapped by the lack of inclusion of information on marijuana use and dependence.

The treatment of only a single drug dependence reduces the probability of success. Many addicts need to abstain from all potentially addictive drugs. This study examines the rate of DSM-III-R diagnosis of marijuana dependence among persons with a diagnosis of cocaine dependence. We used both retrospective and prospective methods to evaluate 232 inpatients and 51 outpatients.

METHODS

Three separate methods of study consisting of retrospective analysis of inpatients, a prospective analysis of inpatients, and a prospective analysis of outpatients were incorporated. One hundred fifty patients selected for the retrospective portion of the study were consecutively discharged in a six-month period from July to December 1986. The patients had received treatment in two different inpatient drug units with similar programs. All of these patients had received the diagnosis of cocaine dependence. The prospective inpatient study included 82 patients consecutively admitted for evaluation and treatment in an inpatient drug treatment unit during a six-month period from April through September 1987. The prospective outpatient study included 51 patients who were consecutively admitted for outpatient treatment of cocaine dependence.

All diagnoses in this study were derived from DSM-III-R criteria in a structured diagnostic interview by a psychiatrist for substance dependence. Other diagnoses in addition to the diagnosis of cocaine dependence were recorded for all 283 patients. Age, sex, employment status, marital status, medical problems, and prescribed medications were noted. The age of onset of first use of cocaine and marijuana was determined when possible. The data were collected and analyzed by individuals other than the psychiatrist who determined the diagnoses.

RESULTS

In the retrospective study of 150 consecutive patients selected with a DSM-III-R diagnosis of cocaine dependence, 15 (17%) were diagnosed as having cocaine dependence only. An additional 79 (53%) patients with cocaine dependence fulfilled DSM-III-R criteria for cannabis dependence (Table I). The age of onset of marijuana

TABLE I. Current Diagnoses

Study Group	N	Mean Age (yr)	Cocaine Dependence Alone N	Cocaine and Cannabis Dependence N
Retrospective inpatients				
Male	118 (79%)	28.3	15	66
Female	32 (21%)	26.0	10	10
Total	150		25 (17%)	76 (53%)
Prospective inpatients				
Male	65 (79%)	36.5	4	15
Female	17 (21%)	39.2	2	11
Total	82		6 (7%)	26 (46%)
Prospective outpatients				
Male	39 (76%)	31.9	2	21
Female	12 (24%)	26.8	1	5
Total	51		3 (6%)	26 (51%)

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use was earlier than for cocaine use by six to seven years on the average for both sexes (Table I).

In the prospective inpatient study, of the 82 consecutive admissions to the inpatient drug unit, 65 (79%) were male and 17 (21%) were female. When the index diagnosis was taken as cocaine dependence (36 patients), 15 (42%) of the patients with cocaine dependence fulfilled DSM-III-R criteria for cannabis dependence. The age of onset of first drug use was 15.8 years for marijuana and 19.9 years for cocaine (Table I).

In the prospective outpatient study, there were 51 consecutive admissions to the outpatient treatment program with the diagnosis of cocaine dependence; three (6%) had a diagnosis of cocaine dependence alone, and 26 (51%) of the patients with the diagnosis of cocaine dependence fulfilled DSM-III-R criteria for cannabis dependence (Table I).

The inpatient and outpatient populations were similar for patterns of drug use. The majority of the patients had administered the cocaine intranasally (90%); smoking freebase cocaine was much less common (9%); and the intravenous route was used by only 1%. The average amount and frequency of cocaine used was 1 to 2 grams per day for three to four days per week. The average amount and frequency of marijuana used was one to three cigarettes (joints) per day for three to four days per week.

The reasons most commonly given for the use of marijuana were to "come down" or "sedate" cocaine-induced anxiety, to relieve the cocaine-induced "depression," to use as a substitute when cocaine was not available, and to use marijuana as the "drug of choice" or primary drug.

The majority of the patients were white (90%), employed, and middle class. About 50% were married.

DISCUSSION

The retrospective inpatient, prospective outpatient, and prospective inpatient methods of study yielded rates of marijuana (cannabis) dependence of 53%, 42%, and 51%, respectively, among patients who had received the diagnosis of cocaine dependence. A majority of the patients had entered the hospital with some insight and acceptance of a cocaine problem but tended to deny a marijuana problem. The diagnosis of marijuana dependence was often obtained in the structured interview and was not volunteered by the patient prior to admission for a cocaine problem.

The patient population is representative of a suburban, middle-class, predominately white, intranasal-using substance abuser. Further research is needed to determine if this prevalence rate of cannabis use applies to a population of urban, black, freebase-using patients. Other survey data suggest that cannabis use is high among young, urban cocaine addicts.[1,2]

The need to identify marijuana use and dependence among cocaine addicts is crucial. Cannabis dependence has its own medical, psychiatric, and social consequences, as well as a natural history and prognosis that require treatment.[6,7] Cocaine addicts may relapse to cocaine use if marijuana is used, whether in an addictive or nonaddictive manner. The treatment for cocaine dependence can be similar for cannabis dependence. Abstinence, group and individual therapy, and 12-step programs are effective in the treatment of both cocaine and cannabis dependence.[4,8]

More studies are needed to confirm the high prevalence of cannabis dependence coexistent with cocaine dependence. Marijuana may also be used prior to and/or concurrently with other drugs such as alcohol. The diagnosis of cannabis dependence is common among heroin addicts.[9] The diagnosis of alcohol dependence among cocaine addicts may be high.[9] The large number of cocaine addicts who have the additional diagnoses of alcohol and drug dependence is an important finding.[10] This multiple drug dependence in the same addict may suggest that the vulnerability to one drug dependence extends to other drug dependencies, and that the treatment of all forms of drug dependence is critical to outcome. Further support for this finding could affect the implementation of drug treatment programs.

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REVIEW ARTICLE

The public health consequences of crack cocaine

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The 1980s ushered in a new era in the world of illicit drug use across America. Cocaine use, which had grown steadily in popularity since the late 1960s, exploded in the mid-1980s with the introduction of "crack"—smokable cocaine.

HISTORY

The history of cocaine goes back at least to 500 AD, when the Peruvians chewed the leaves of the *Erythoxylon coca* plant for energy. Its use later spread to Europe, where it was added to wine and other beverages. Subsequently, in the 1800s, cocaine was brought to the United States, where it was added to patent medicines. After restrictive legislation was passed regarding the use and sale of cocaine, the drug vanished from mainstream America to the dens of the underworld. Amphetamines became the stimulant of choice and remained so until their use became tightly regulated. Cocaine re-emerged in the 1960s with a new, glamorous image as the drug of wealthy jet-setters. "Snorting" (nasal inhalation) was the most popular route of administration, but "freebasing" cocaine to a smokable form quickly spread. Freebasing, however, required a volatile solvent (ether), making the process hazardous, until the discovery of another smokable form of cocaine, crack, which could be safely made using baking soda.[1] Widely available now, small crack packets may be purchased for as little as three dollars.

THE SEDUCTION OF COCAINE

Cocaine's allure is its powerful euphoric effect which occurs within seconds after smoking or intravenous use and minutes after snorting. The user feels omnipotent, as though he/she had accomplished a wonderful feat, such as winning several Olympic gold medals. There is a feeling of increased competence and elevated self-esteem. Cocaine, especially smokable cocaine, is highly addicting and particularly attractive to women and teenagers.[1,2]

Unfortunately, due to cocaine's stimulant qualities and its post-euphoric depression, cocaine use is accompanied about 68% of the time by other drugs, primarily sedatives such as alcohol, marijuana, and heroin. Conversely, cocaine is used by heroin addicts to treat their withdrawal symptoms. The simultaneous injection of cocaine and heroin is called "speedballing."[1]

MORBIDITY AND MORTALITY

Cocaine use has had staggering effects on morbidity and mortality in urban areas across the country. The Drug Abuse Warning Network (DAWN) records the number of emergency room visits precipitated by drug use. Each such visit is referred to as a "mention." Cocaine mentions in Detroit, Michigan, increased 1,400% from 1982 to 1987 (Fig 1), and speedball emergency room mentions rose 480% during the same time period. Treatment admissions for cocaine in Detroit rose 4,000% between 1982 and 1987, with a steep rise in 1985 when crack hit the streets (Fig 2). During similar time periods, cocaine deaths in Detroit increased by 1,400% (Fig 3), and deaths from speedballing increased 900% (Fig 3). These dramatic increases have been accompanied by a 5.5-fold increase in homicide victims testing positive for cocaine (Fig 4), as well as the finding that 53% of those arrested for felonies have cocaine in their urine. In 1987, one fifth of automobile accident victims and one fourth of suicide victims tested positive for cocaine.[3]

SEX FOR DRUGS

Marked increases in the incidence of sexually transmitted diseases (STDs) parallel widespread crack use across the country. This phenomenon is occurring in rural as well as in urban areas.[2,4] Between 1983 and 1987, Michigan experienced an 80-fold increase in penicillin-resistant cases of gonorrhea. These cases were traced to "crack" house sexual activity.[5] Increasing numbers of cases of pri-

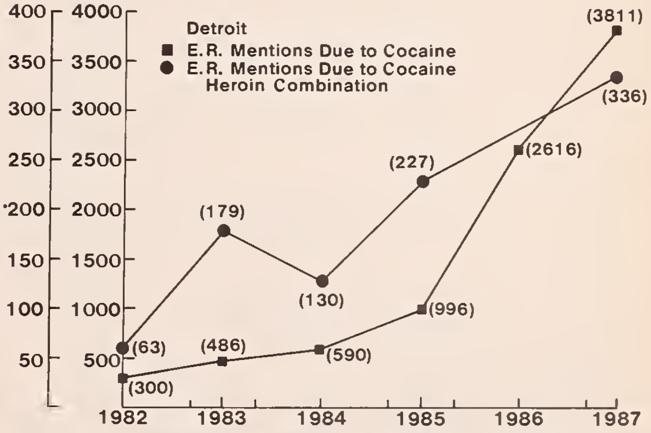

FIGURE 1. Cocaine emergency room mentions in Detroit, Michigan, 1982–1987.

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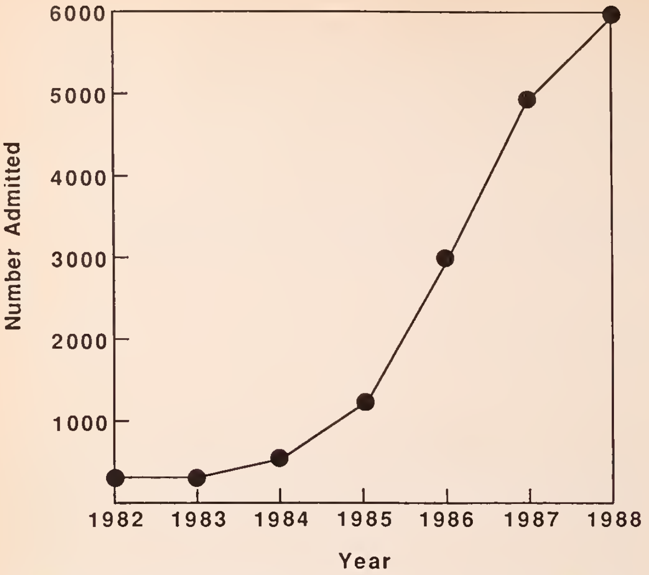

FIGURE 2. Detroit drug treatment admissions, 1982–1988 (1988 figures projected from six-month data).

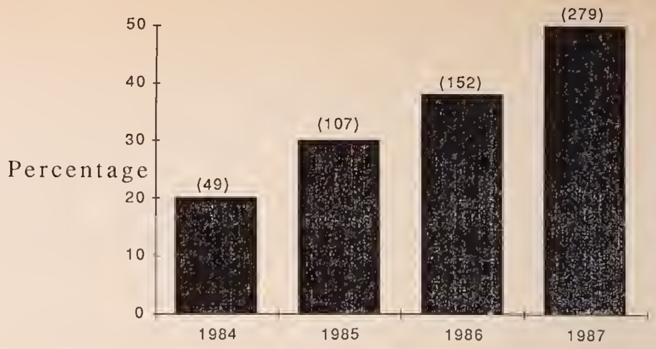

FIGURE 4. Percent of homicide victims 40 years of age and under testing positive for cocaine, 1984–1987.

mary and secondary syphilis nationwide in the heterosexual population are clearly having an impact on the skyrocketing numbers of cases of congenital syphilis. In New York City, the incidence of primary and secondary syphilis in women rose from 541 cases in 1986 to 1,814 in 1988, while the number of congenital syphilis cases rose from 57 to 357 during the same time period.[6] In 1989, New York City had 950 cases of congenital syphilis (personal communication, New York City Department of Health, March 1990). A case study done in New York City found that 28% of the mothers who had babies with congenital syphilis used cocaine, versus 6% in the control group.[6] Detroit has seen an increase of congenital syphilis cases from zero to 20 between 1984 and 1989. All of the mothers of the 20 babies with congenital syphilis born in 1989 were drug users, and virtually all admitted to using crack cocaine (personal communication, Michigan Department of Public Health, February 1990). Unfortunately, congenital syphilis causes significant mortality among affected infants and this only compounds the infant morbidity and mortality associated with perinatal crack use.[7,8]

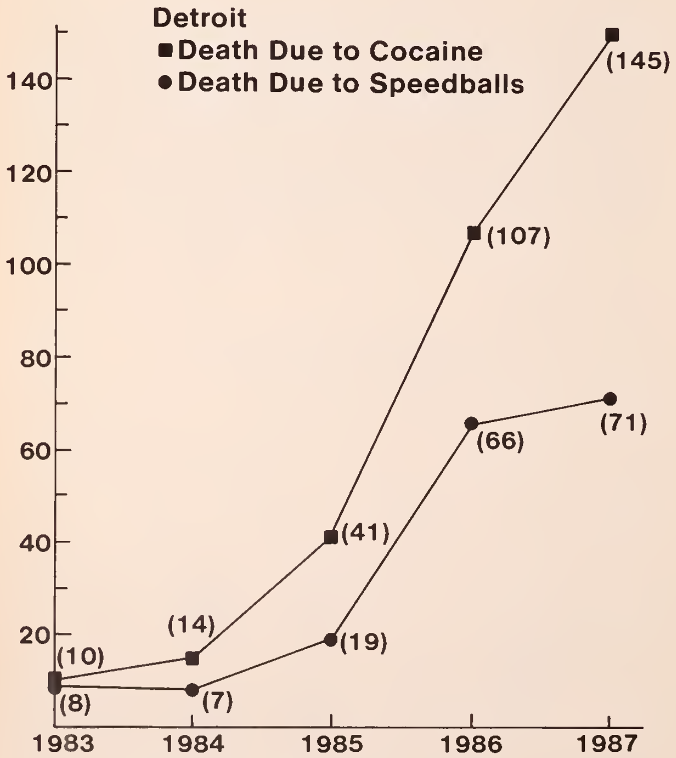

FIGURE 3. Deaths due to cocaine and "speedballs."

The association of increased STDs in crack users is due in part to the phenomenon of trading sex for drugs. Among middle class adolescents who were heavy cocaine users, 60% said that they traded sex for cocaine.[9] Another study of adolescents in Oakland and San Francisco, California, found that 25% of the subjects who used crack admitted to exchanging sexual favors for drugs or money.[10] Another factor in the association of STDs and cocaine is that cocaine, particularly in lower doses, is a libido stimulant or aphrodisiac causing prolonged erection in men.[11]

THE COCAINE-AIDS CONNECTION

Crack use appears to be associated with human imunodeficiency virus (HIV) infection due to the same factors that contribute to increased STDs among crack users. One study of 109 women in New York City hospitalized for pelvic inflammatory disease showed that of the HIV-positive women, 87% were crack users. The crack users also had twice as many sexual partners as noncrack users.[12] Studies have shown that the risk of HIV transmission rises in association with genital ulcers found in syphilis, herpes, and chancroid. These infections are increasing rapidly among cocaine users.[2]

CONCLUSIONS

The current nationwide epidemic of crack cocaine use is having a major impact on public health. In particular, California, Florida, New York, Pennsylvania, and Michigan have noticed significant rises in crack cocaine related morbidity and mortality.[4] Increases in the incidence of STDs and congenital syphilis are most striking. The latter trends will require physicians and public health officers to rethink STD prevention and treatment strategies. For example, innovative methods for helping women to avail themselves of prenatal care are clearly needed. Obstetricians need to increase STD surveillance, testing for syphilis during the second and third trimesters and at delivery in high-risk populations.[13] Most essential, however, is available and accessible long term treatment for crack cocaine addiction.

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REPORT ON DRUG ADDICTION II

In 1955 The New York Academy of Medicine issued a comprehensive report on drug addiction. Its main themes set forth the two most potent reasons for the growth and spread of drug addiction: profit and punishment.

To appreciate fully the powerful influence of profit on narcotics addiction, it is necessary to examine the economics of narcotics. Whether recognizable readily or less obviously, most things have their financial aspect. It is equally true that many useful, even noble things are corrupted. With this perversion their financial aspect may take a disproportionate and dominant position. Narcotics are a conspicuous example. Appropriately used, they serve important medical purposes. Except for these purposes, possession or purveyance of them is illegal. Under these restrictions they have become debased into the product of a vicious and nefarious business.

Drug addiction is a complex condition with several phases: medical, sociologic, psychologic, and economic. In this constellation it would be a mistake to overlook or underestimate the powerful economic force operating in drug traffic. Between drug addiction and drug traffic the relation is an interaction. In a circular arrangement addiction invites and supports traffic; in turn, traffic enables addiction to endure. It is obvious that if there were no addiction there would be no traffic, and vice versa.

Profit. Throughout the transactions in illegal narcotics, economic factors are at work. Here has grown up a sizable enterprise with several levels of employees whose combined duty is to purvey narcotics illicitly at a price, an exorbitant price. Consequently, many consumers under the compulsion of their habit are driven to engage in shady, degrading, illegal, even criminal activities to obtain money or its equivalent, to pay for their supply.

Why should anyone want to operate this sordid, illicit business in narcotics? For the same reason that persons conduct any legitimate business—profit. Of all the economic elements, profit is the most impelling.

There are several features of narcotics traffic, some unique, that make the prospect alluring. Illicit business usually yields extraordinarily high profits. In the illicit sales of narcotics, these already swollen profits on occasion expand further. The more vigorous the police action against illicit traffic, with the consequently greater risk, the higher the price of the product and the greater the profit. In addition, the very nature of narcotics' effects virtually ensures endless repeat sales. Finally, whether by accident or design, another development has added a particularly vile and despicable, but again highly profitable, note to an already sordid and contemptible racket. Cultivation of juveniles and teenagers or pandering to their inclinations opens a "dream" market of lifetime sales.

Thus, profit is one of the most potent influences that perpetuate addiction and allow it to spread. If purveyance of narcotics were not profitable, it is doubtful that the syndicates would remain in the business. With no supply, addiction would cease. Thus, traffic and addiction maintain each other because of one very important economic factor—profit. . . .

THE NEW YORK ACADEMY OF MEDICINE
Committee on Public Health
(*NY State J Med* 1963; 63:1977–2000)

Position paper of the Associated Medical Schools of New York on HIV testing and access to treatment

Through its committee on the acquired immunodeficiency syndrome (AIDS), the Associated Medical Schools of New York (AMS) has monitored this unfolding epidemic and has conferred with public health officials to determine how the state's medical schools can serve as a resource for the state and New York City. In 1988, AMS contributed to the public dialogue on the responsibility of physicians to treat AIDS patients when it became the only major medical organization to take the position that dismissal should be considered as a sanction in the event a medical school faculty member, resident, or student refused to treat an AIDS patient. The organization has also actively supported state legislation assuring confidentiality for those testing positive for HIV and its members have been in the forefront of those urging the federal government to continue to provide assurance that no one would be denied treatment for AIDS because of financial barriers.

In this statement, AMS addresses the difficult and emotional issues of testing and reporting of AIDS information, along with access to care and confidentiality. Undergirding the AMS position are data on the continued acceleration of the epidemic and growing evidence of the efficacy of treatment modalities, most notably zidovudine (AZT) and pentamidine. The New York City Department of Health has estimated the number of infected individuals in New York City at 200,000. The actual cumulative caseload in New York City is 21,000. The New York City AIDS Task Force estimates that this will triple to about 64,000 by the end of 1993, with as many as 50,000 succumbing to the disease.

Of growing concern is the impact of AIDS on infants and newborns. The New York City Health and Hospitals Corporation (HHC) provides data on what it perceives to be the increasing impact of AIDS on women and children. Noting that the majority of pediatric AIDS cases arise from infection passed from mother to infant, HHC estimates that, of 200,000 intravenous (IV) drug users in New York City, approximately 60,000 are women of childbearing age. An estimated 40–60% of IV drug users are HIV infected. HHC further estimates that women, infected as IV drug users or as partners of infected men, will account for an increasing share of new AIDS cases as well, and the New York City Department of Health projects an increase in the share from 16% in 1988 to 21% in 1993.[1]

Clearly, the potential for a frightening acceleration in the number of babies born HIV positive is clear. For that reason, evidence of the effectiveness of treating AIDS in infancy and early childhood takes on major significance. In studies reported by Dr Philip Pizzo of the Pediatric Branch of the National Cancer Institute, treatment with AZT resulted in significant improvement in several AIDS-related symptoms in children.[2] Underscoring the potential problem in children, Dr Pizzo cites projections by some authorities that in coming years AIDS will be detected in as many as 10,000 to 20,000 children.[2] Much still needs to be learned about the most appropriate time to begin treatment and about the optimal regimen.

Recent attention has been paid to the increasing prevalence of AIDS among adolescents. Recently, Dr Jody W. Zylke cited 1988 data from the Centers for Disease Control showing that the proportion of AIDS attributed to heterosexual activity was higher than for other age groups.[3] She quoted Dr Karen Hein, Director of the Adolescent AIDS Program at Montefiore Medical Center, who cited statewide data showing that the number of HIV-positive infants born to adolescent mothers has skyrocketed, going from ten infants recorded in a recent 12-month period to 88 during the following 12 months. In addition, noting the difference in the male-to-female ratio among adolescents in New York City, she notes that for adults it is 7:1 and for adolescents, 2.9:1.[3] Finally, she noted that the HIV positive rate among 1,500 homeless and runaway young people at Covenant House in New York City in a recent study was 7% among adolescents and 16% among 18–20-year-olds.[3]

These data make it clear that the ability to identify high risk mothers and infants and to diagnose the presence or absence of HIV infection accurately is an extremely important goal.[3]

Data on the implications of early intervention for all age groups were reported by Arno et al.[4] The authors concluded as follows:

> The development of early diagnostic and therapeutic care for asymptomatic HIV disease has already begun, and we believe the trend will continue. The need to think about the implications of early intervention on the provision of care should not wait until research has achieved its goal, but rather should proceed with the expectation that greater pharmacologic success will eventually be with us.
>
> Despite its promise, the epidemiology of HIV infection suggests that there is a limited window of opportunity in which to provide early intervention to many of those carrying the virus. Data from the Hepatitis B Cohort Study in San Francisco indicate that cumulative progression to AIDS increases from 15% to 39% to 54% among those infected for five, eight and 10 years, respectively. . . . The implication of this assessment is clear: time is running out.[4]

These and other data provide a powerful case for public policy that calls for aggressive outreach to high risk populations aimed at convincing those at risk to be tested and diagnosed so that treatment might begin as early as possible. Efforts to reach contacts must also be pursued as vigorously to slow the spread of the disease and increase the numbers of those under treatment.

In this light, the current initiatives being undertaken by the AIDS Institute of the state health department merit broad support. Specifically, the institute has begun to reach out to at-risk women by intensifying its efforts to ensure that HIV counseling and voluntary testing become a routine part of gynecological and prenatal care. Recognizing that many at-risk women receive little or no prenatal care, the institute has begun a pilot effort to assist hospitals in developing in-hospital HIV counseling and voluntary testing programs. On a broader level, the American College of Obstetricians and Gynecologists and the Medical Society of the State of New York have joined with the Department of Health in appealing to physicians to incorporate active HIV counseling into their standard of practice. To further strengthen access to primary care, the Department of Health has developed enhanced Medicaid reimbursement rates for HIV related primary care visits. In addition, grants have been made available to community health centers and AIDS designated centers for a variety of outreach, prevention, and provider education programs and $1.5 million of state funding has been dedicated to the development of integrated community based primary care and drug abuse treatment. AMS strongly supports these initiatives.

A critical need in the state's response to the AIDS crisis is additional personnel with the training and expertise to understand all dimensions of the disease. To address these needs at the physician level, the Department of Health recently instituted a fellowship program for physicians and nurses to provide training in the management of HIV infection and substance abuse. AMS supported this proposal and is prepared to participate in the development of such fellowships.

Clearly, the federal government must also back up its public expressions of deep concern about the epidemic with a recognition that the high prevalence areas merit special consideration in federal grants.

In terms of federal legislation, AMS endorsed the recently enacted Ryan White Act, which provides emergency assistance in areas of high HIV prevalence, and the Americans With Disabilities Act, which extends protection against AIDS-related discrimination to employment and public accommodations.

While outreach and service initiatives are being developed, we must not lose sight of the absolute necessity of intensifying research into therapeutic procedures for HIV infection and of integrating the research into patient care. Well designed clinical trials are essential, and access to clinical trials by IV drug users and their contacts should be improved. An encouraging development is the pilot testing of community based clinical trials in San Francisco, in which private practitioners will have an opportunity to enroll HIV-infected patients. No one argues the need for intensified efforts to make treatment available to as many as possible. What is at issue are the pragmatic and logistical questions involved in such policy implementation.

Routine testing is perhaps the most controversial of the AIDS policy issues. Yet it cannot be separated from the issues of confidentiality, counseling, availability of treatment, and financing. The testing controversy revolves around the degree to which traditional public health procedures involving routine testing and follow-up should be applied to the AIDS epidemic.

Although these concerns are similar to those once prevalent in the early stages of the epidemics of syphilis and other highly communicable diseases which, at one time, had no cure, the fear of discrimination towards those infected with HIV appears far more pronounced than in those earlier epidemics. Sadly, there is evidence that those fears are well grounded, given the number of known incidents of outright discrimination.

The issue of possible discrimination remains by far the strongest argument against routine testing for HIV. This eminently justifiable concern then must be balanced against the reality that large numbers of at-risk individuals will not be in a position to receive therapy because they have not been tested and diagnosed, nor, in the case of sexual partners, even informed that they are at risk. The latter concern becomes increasingly significant with the evidence cited above that AZT and possibly other drugs can indeed delay the onset of symptoms and prolong life.

The AMS view is that the improving therapeutic modalities for AIDS have changed the nature and the focus of the public policy dialogue regarding testing and have led to this reconsideration of the positions taken by AMS regarding testing two years ago. Unfortunately, these developments have not reduced the complexity of the dialogue; if anything, they have intensified it. More than ever, the justified high priority accorded to the need to protect those with AIDS from discrimination must be weighed against the question: Are we, in fact, allowing opportunities for early intervention and treatment to slip past us?

The essence of the AMS position is as follows: HIV infection is a sexually transmitted communicable disease with an exceedingly long incubation period and a nearly uniformly fatal outcome. Evidence that early intervention can be effective in at least delaying the onset of the disease is compelling, as is the evidence that the transmission of this disease is now cutting across previously designated at-risk population groups. An effective and professional approach to this public health problem demands maintenance of utmost confidentiality while allowing all those at risk to be so notified.

Discussions of testing must not detract from the immediate priorities of providing basic primary care to HIV infected individuals. At a time of fiscal crisis, the staggering cost of this burden on those states with high volume of AIDS must become a major national priority. Governor Cuomo has called for a meeting of public policy officials from those high volume states to recommend a more equitable and precisely targeted use of federal funds. Such a conference would be a valuable step in confronting the intensifying financial implications of the AIDS epidemic.

In the meantime, efforts to convince high risk patients to be tested and also to contact partners must be intensified. Time is indeed running out, and we cannot wait for the published research reports before developing the logistics and the financing for dealing with the enormous demand for this care that will inevitably result from these technological developments.

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COOPERATION BETWEEN PATIENT AND PHYSICIAN

The Essence of Good Medical Care, Individual or Collective

The educational policies, methods, subject matter, and teaching in the schools of medicine are the undertakings of physicians or nonmedical representatives of the basic sciences.

This education of physicians before they can be licensed to practice among the people, and the subsequent training of the recent graduate in hospitals, and the still later postgraduate study by physicians for higher and specialty qualifications is all done under the control of physicians and their colleagues in the basic sciences.

The people, voluntarily and through taxation, have supplied the financial resources for schools of medicine with their related hospitals, laboratories, and research institutions, and, yet, the cost of the medical student's education is higher than that of any other profession or occupation, not only to the institution of learning, but in the matter of tuition fees to the student and in his years of life.

Once the physician is licensed to practice medicine he may live and work wherever he finds conditions to his liking, socially, economically, culturally, or professionally, and he is thereafter under no compulsion of law to pass examinations as to his competence or to alter his place of residence, so far as the latter is within the jurisdiction of the state or national board of examiners which passed upon his original qualifications.

Briefly, then, the people, having set up what they consider sufficient formal requirement to protect themselves against grossly incompetent practitioners of medicine, have trusted to the wisdom of medical faculties and to the ambition and self-interest of physicians the education and distribution of practitioners of medicine to meet the needs of the sick.

The experience of the people of the United States has been, on the whole, favorable under the present conditions which cannot be properly described as a system, physicians being now better educated, more widely distributed, and in higher ratio to the population in our states than has been the case heretofore with any similar number of people in other lands or under other methods of control, education, and distribution of physicians. We can note properly in this connection that every index of health, or relative freedom from preventable diseases, and of longevity reveals a state of affairs in the United States superior to that of any such aggregation of people or of races elsewhere under single or multiple government.

Two major complaints have been brought against the present medical care of the sick in our country, namely, the unevenness of its availability geographically for some portions of our population, and the cost of services of physicians and of medical institutions which are beyond the day-by-day resources of a large number of the sick to meet out-of-pocket, or at the time of sickness and the receipt of the medical services.

It is to the correction of these that our first efforts should be directed to the end that sickness may be diagnosed correctly, and humane and skillful treatment be applied, wherever people are, regardless of the ability of the sick person to pay on the spot for the medical care required. This is a high ambition and worthy of the best brains and spirit of our times. . . .

HAVEN EMERSON, MD
(*NY State J Med* 1947; 47:1501–1506)

Gas warfare: Military-medical responsiveness of the Allies in the Great War, 1914–1918

JOHN S. HALLER, JR, PHD

The debut of chemical warfare as a weapons system dates to the fifth century BC during the wars between Athens and Sparta (431–404 BC). At the siege of the walled city of Plataca in 429 BC, the Spartans built large mounds of earth close to the southern wall of the city and on these mounds lit fires of faggots soaked in pitch, and on which they placed brimstone. They intended for the prevalent southerly winds to carry the sulphurous fumes over the city wall. The Athenians responded by tunnelling under the wall and into the mounds, causing their collapse. Five years later, Thucydides recorded the use of poisonous fumes in the siege of Delium, a fortified post held by the Athenians and besieged by the Boetians. According to his accounts of the siege, the Boetians hollowed out a large tree and, using a bellows, forced sulphur dioxide through the "tree pipe" into the garrison at Delium. The Athenian garrison took flight and the fort was taken.[1(p413),2(pp233-234)]

In the following century, Aeneas Tacitus described a new technique in sea warfare in which sailors lit wooden pots filled with sulphur, pitch, charcoal, and tow, and threw them on the decks of enemy ships. Aeneas also described similar fire compositions thrown burning from the walls of besieged towns onto and under the testudo and buildings which sheltered the besiegers.[2(p234)]

"Greek fire," invented by the Greek architect Callinicus in 678 AD, was a combination flame-thrower, smoke-cloud, and poison gas, the chemical composition of which was jealously guarded by the Emperor of Constantinople. Naphtha and quicklime were its principal ingredients, with a certain amount of sulphur and pitch. Armies projected Greek fire by means of a wooden siphon, pointing the siphon tube (which took the shape of dragons and other monsters) towards the enemy, and at the same time applying the hose pipe of a water engine at the rear. The heat generated by the interaction of the water on quicklime fired the mixture and generated clouds of smoke and soot, along with sulphurous acid gas. The Byzantine Greeks used the "Greek fire" in their wars against the Saracens and, later, the Saracens used the same weapon in their defense against the Crusaders in the 13th century.[2(p235),3(p23)]

Examples of gas or chemical warfare extended into the 19th century as well. Admiral Lord Dundonald was so impressed by the destruction of animal and vegetable life near the Sulphur Kilns in July 1811 that he considered the phenomenon applicable to military and naval purposes. A commission consisting of Lord Keith and Lord Exmouth reported favorably on his suggestion. With the outbreak of the Crimean War in 1854, Dundonald suggested using sulphur fumes to expel the Russians from Sebastopol by rendering the Matakoff and Redan works untenable. He estimated that 400 or 500 tons of sulphur and 2,000 tons of coke could accomplish the task. The committees that reviewed Dundonald's proposal concluded that the effects from the sulphur would undoubtedly occur; nevertheless, they recommended against its implementation because they considered the effects so horrendous that "no honorable combatant could use the means required to produce them."[1(pp413-414),4]

Also during the Crimean War, Lord Playfair suggested the introduction of a hollow brittle shell containing phosphorus dissolved in bisulfide of carbon to produce "conflagration of the enemy's stores or property." Another suggestion from Playfair involved the use of a hollow brittle shell containing cyanide or cacodyl which, if fired between decks of a ship, "would render the atmosphere irrespirable, and poison the men if they remained at the guns."[4(p22)]

During the American Civil War, one inventive Yankee suggested capturing Petersburg by firing shells containing snuff into the city in order to set its defenders to violent sneezing.[5(p413)] Although the Union army never tested this novel approach, it did employ chemical warfare during the siege of Charleston by setting fire to wood saturated with sulphur and placing it under the parapets about the city. On another occasion, General Gilmore ordered shells containing "Greek fire" sent into the town.[6(p245)] Less than a decade later, during the Franco-Prussian war, a French chemist prepared a fulminate of picrate of potash intended for use against the German army.[5(p413)]

Gas poisoning became an unintended by-product of the naval race at the turn of the 20th century and the accompanying development of high explosives. The 15-inch guns of the super-dreadnoughts, with charges of 400 pounds of cordite, gave off 2,500 cubic feet of carbon monoxide each time they were fired. Shells that exploded in close spaces such as cellars or the interior of ships sent out nitre compounds such as picric acid and trinitrotoluol to form nitric oxide gas and carbon monoxide. During the Anglo-Boer War, General Joubert protested to General White against the use of the lyddite shell, which he claimed had caused

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inhumane suffocation. Actually, the carbon monoxide poisoning was incidental to combustion and not the intended method of destruction.[7(p110),8(p383)] During the Battle of Tsushima in the Russo-Japanese War, Lieutenant Semenoff of the Russian Navy recounted vivid descriptions of asphyxiating yellow fumes from the Japanese explosive "Shimose" which enveloped the Russian ships. He also recorded the effects of smoke from below-deck fires and of funnel smoke which entered pierced ventilators and incapacitated the ships' crews.[9(p461)]

In the years before the Great War, Germany became the unchallenged master of the chemical industry, producing chlorine on a tremendous scale for bleaching powder and a thousand other uses. The great chemical works such as the Badische Anilin und Sodafabrik at Ludwigshafen, the firm of Meister Lucius und Bruning at Hochst-am-Main, the Bayer Company at Leverkusen, and the Griescheim-Elektron Chemische Fabrik were unrivalled in their ability to meet the needs of German industry and, eventually, the needs of the German General Staff. Even the gold industry required huge quantities of chlorine in its reduction processes, while chlorine's value in purifying water was known to every water engineer. German industry understood chlorine; it was deadly, easily liquified, produced in large quantities from salt, and readily transportable. When scientists discovered ways to liquify gases, the potential for chemical warfare increased precipitously. This was particularly the case with liquid sulphurous acid, chlorine, bromine, nitrous bioxide, and carbon dioxide.

Not surprisingly, the signatories to the Hague Peace Conference in 1899 agreed to abstain from the use of "asphyxiating or deleterious gases." At the Hague Conference of 1907, signatories reaffirmed the earlier statement and widened the definition to include the use of "poison or poisoned weapons" (Article 23). Clearly, the signatories to the two conventions understood the intent of the article and, while the only force for the prohibition of gas warfare was a moral one, its significance could not be ignored. Thus, when General Erich von Falkenhayn and the German High Command chose to employ chlorine at the second battle of Ypres in April 1915, they breached the conventions and offended the sense of honor and fair play held by the belligerents.[10(pp119,121,174,196)]

THE BATTLE OF YPRES

The Great War began as a war of movement, with German armies sweeping across Belgium and sending shock waves through the capitals of Europe. By September, however, the German advance had ground to an unexpected halt following the battle of the Marne. A race to the sea quickly followed, with the German and French armies attempting to outflank each other. By the end of the first battle of Ypres (October 30–November 24), the western front entered into a stalemate, confined largely to trench warfare which endured for nearly four years. Eventually, more than 15,000 miles of trenches stretched in zig-zag fashion from the North Sea to the borders of Switzerland and became the killing ground for armies on the western front.

In their attempt to break through to the coastal ports, the Germans employed chlorine gas clouds over an eight-mile front. They made their first attack at 5:30 PM on April 22, 1915, against trenches in the vicinity of Langemarck, northeast of Ypres in an area from Bikschote to Poelkapelle. Weeks before the attack, prisoners as well as a German deserter had informed the French that the Germans were preparing to introduce gas from cylinders installed in their trenches. However, French intelligence gave little notice to the information and, while the British were more cautious with the information, the attack nevertheless caught officers and men totally unprepared and unprotected.[11(pp31-32)] The whole of the line manned by French, African, and Territorial troops fell back, leaving a wide gap and exposing the left side of the Canadian Division. German infantry followed up the attack by occupying ground up to the Yser canal between Steenstraate and Het Saas. The Canadians moved to fill the gap but at a tremendous cost.[12(pp363-364,420)] The Germans delivered a second cloud attack on April 24, directed primarily against the Canadians, who, though unprotected, held their ground and even launched a counter-attack. This was followed by other gas attacks in May, all in the Ypres sector. Following the attack on May 24, there was a lull until December 1915.[13(p234)]

Despite the element of surprise, the Germans did not foresee the tremendous impact that gas would have and were understandably unprepared to exploit their success. Moreover, they had provided insufficient protection for their own men. Thus, when the Canadians rallied and closed the line, Germany lost one of its major opportunities for breaking the stalemate on the western front. In fact, the Germans did not attempt to follow up their success or to attack in that sector for another day and a half. Had the Germans been able to penetrate the gap, there would have been nothing to stop them between Langemarck and Dixmude.

At the time of the attack, the Allies did not know the type of gas used except that, from its effect on metals, it was obviously an acid agent. Confirmation of this came when the British captured a German respirator which consisted of cotton-waste soaked in a hyposulphite solution.[14(p52)] From this information, the Allies began the task of finding appropriate protection for their soldiers in the trenches. They also proceeded to consider the most effective organization for effecting their offensive and defensive measures. Initially, the British directed that the Ministry of Munitions assume responsibility for retaliatory measures. At the same time, protective responsibilities fell to the Army Medical Department which set up laboratories in the Royal Army Medical College at Millbank and also established a new section, the Anti-Gas Department. The Ministry of Munitions relied for assistance on the Chemical Advisory Committee of the Royal Society, and the Anti-Gas Department had a similar group of scientists from the fields of chemistry, physiology, and pharmacology to serve its purposes. Because many problems were, in fact, common to both groups, in October 1917 the government transferred to the Ministry of Munitions the Anti-Gas Department which, along with the offensive organization, became a section of the Chemical Warfare Department.[15(p309)] "Every development in the use of gas was foreseen and provided for," commented Major W. R. Galwey, "so that our gas defence in a short time equalled and then surpassed that of the enemy."[15(p67)] From 1916 on, the British and French gas services maintained a close liaison, and inter-Allied conferences were held in Paris at which "all possible information on every aspect of the subject was confidentially placed before the representatives of each of the allied armies."[11(pp139-175),16(pp249,324-328)]

On April 23, in an effort to counteract the effects of the gas cloud, the Director-General Medical Services, General Headquarters, issued a circular directing troops to maintain a solution of bicarbonate of soda in the trenches with instructions to dip a handkerchief or cloth into the solution, wring it out, and cover the nose and mouth during a gas attack.[15(p66)] In the meantime, troops at the front had improvised protective devices, including the use of a limited number of Vermorel sprayers for cleansing trenches and dugouts. Men devised pads made from handkerchiefs, torn strips of shirting, and socks to cover the mouth and nose. Many of these masks were simply strips of gauze and cotton moistened with water or urine (which contained the alkali ammonia to combat the acid fumes). Some found protection by placing their faces in the mud, the wet earth acting as a filter. Others broke off the bottoms of bottles, filled them with earth and water, and then breathed through the bottle.[17(p3)] The chemist Henry Brunner recommended that soldiers breathe through a cloth moistened with whiskey, brandy, rum, or other spirituous liquor.[18(p880)]

Within 48 hours of the initial attack at Ypres, the French had supplied their troops with masks; and only a few days elapsed before the British soldier was similarly equipped. Unfortunately, many of these early masks proved worthless, since they consisted of little more than a cotton pad fastened over the mouth and nostrils. Only when combined with hyposulphite of soda or common washing soda did they neutralize the gas. Washing soda (sodium carbonate) proved an efficient absorbent for most of the acidic gases such as chlorine, bromine, and oxide of nitrogen. Three to four tablespoonsful in a pint of water produced a 20% solution that worked well in most situations. The Academy of Medicine in Paris recommended a combination of sodium hyposulphite, sodium carbonate, glycerin, and water.[5(p421)]

METHODS OF GASSING

Opposing armies delivered gases in a number of different ways. During the early period of gas warfare, the Germans discharged drift, or cloud gas (generally chlorine or phosgene) from cylinders at night or in the early morning, when a steady wind of 4 to 12 miles an hour blew toward the enemy lines. Since chlorine and phosgene were heavier than air, they quickly sank into trenches, shell-pits, mine-craters, cellars, and dugouts. In some instances in which the gas penetrated into the trenches, it caused the buttons on the soldiers' uniforms to turn green and their bayonets to blacken.[18(p875)]

The cloud gas cylinders were heavy and unwieldy, requiring considerable time and effort to bring sufficient numbers of the cylinders up to the front line. Placing a 90-pound cylinder every yard or so along the front required extensive preparation. To prevent discovery, the Germans buried the 4-foot cylinders in short trenches under a parapet and then covered them with a quilt of moss containing potassium carbonate solution, and then with sand bags. In preparation for an attack, gas personnel removed the sand bags and connected each cylinder to a lead pipe which extended eight feet over the top of the parapet. They then placed a sand bag over the nozzle to prevent the pipe from kicking back and throwing gas into the trench.

A successful gas cloud depended on thorough preparation, including excellent knowledge of the countryside, the layout of trenches, and the direction and velocity of the wind. Heavy rain destroyed the effectiveness of a gas attack, but slight dampness, with ground fog, worked well because it kept the gas low and provided the opportunity for surprise. Armies preferred to emit gas at night for purposes of surprise and because cold air currents moved down and kept the gas low and effective. The most suitable terrain consisted of ground that sloped gently away from the emission area. The Germans, given the nature of their defensive positions, were usually in the best position to employ gas. With a favoring wind blowing toward the enemy trenches, they opened the valves and the liquid gas ejected under pressure of 76 pounds into the air. On exposure to the atmosphere, the chlorine quickly volatilized, forming a greenish mist which drifted into enemy lines. A Canadian officer gave the following description of the gas as it came toward his line at Ypres.

> On Thursday, April 22nd, when I was in a support trench about 600 yards from the German lines, I saw first of all a white smoke rising from the German trenches to a height of about three feet; then in front of the white smoke appeared a greenish cloud, which drifted along the ground, not rising more than seven feet from the ground when it reached our first trenches. Men in these trenches were obliged to leave and a number were killed by the effects of the gas.[5(p416)]

Although gas clouds operated best in a steady breeze from 4 to 12 miles per hour, attacks were made with winds as little as 2 and as great as 20 miles per hour. Sometimes, the receiving side noticed the effects as far back as 20 miles from the point of emission. However, the distance for serious casualties extended 5 to 6 miles from the point of emission. One cloud in the Champagne district caused casualties 30 miles behind the front line, catching large numbers of French civilians unaware and without protection. The British were equally successful in putting gas over south of Bethune just prior to Messines. From German prisoners, the British learned that the gas had drifted in effective concentrations 10 miles past Douayu.[19(p222)]

Eventually, both armies concluded that the gas cloud had too many disadvantages. In the first place, its success depended on fairly precise weather conditions, with the slightest change causing disastrous results. If too strong, the gas whirled about and dissipated before reaching its intended target or, with a change in direction, wafted back into the lines, creating havoc. The Germans experienced this situation at Hulluch, Flanders, in the spring of 1916, when a change in the wind caused the gas to blow back into their own trenches, causing 1,100 casualties. Then, too, from a tactical point of view, the greatest concentration of the gas cloud occurred at the wrong place, ie, directly in front of the trenches of the attacking force. Consequently, if an attack followed the gas cloud, offensive troops often had to endure more gas than the defending troops. If, on the other hand, they chose to delay the attack until the gas had dissipated, they lost the opportunities created by the gas.[20(pp3-5)]

As the science of gas warfare evolved, military strategists focused on several purposes for which the weapon was used: to inflict death or incapacitation in front lines and support trenches just prior to an attack; to render positions temporarily untenable in areas prior to an attack or where an attack was expected; and to disorganize the enemy, reduce its fighting efficiency by necessitating the use of respirators, and affect morale.[9,15(pp64-66),20]

Given these objectives, armies on both sides of the No-Man's-Land developed the gas shell, which delivered several quarts of poisonous liquified gas in a single shot. However, the quantity of gas sent over in a single shell was small. The typical gas shell weighed approximately 6 pounds, whereas the gas cylinder contained 40 to 45 pounds of gas. Thus, armies had to put the shells on specific targets rather than attempt to imitate the effect of a gas cloud. With 50 or more guns firing approximately ten shots per minute, an army could direct gassing to particular positions at or behind the lines with a precision unknown with the former gas cloud method. Gas shells soon became the most important method of using deploying gas on the western front.

The use of the gas projectile had a number of advantages. Unlike the gas cloud, it was not dependent on the wind. Moreover, it was not necessary to make elaborate preparations which might be subject to enemy observation. Targets could be picked with greater accuracy, and gas shells often succeeded in immobilizing a target for which other artillery shells proved ineffective. Finally, forcing a soldier to wear a mask—whether at the front line or in reserve or support areas—diminished his effectiveness. Since breathing heavily outpaced the capabilities of the mask's defensive chemicals, soldiers did little to jeopardize their lives by testing mask capability.[21(p282),22(p54)]

The last development in gas warfare came with the use of projectors which discharged enormous quantities of gas over small areas. The projector attack, introduced by the British in April 1917 and adopted by the Germans in December of the same year, was effective at ranges up to 2,000 yards. When a large number of mortars were fired simultaneously, by an electrical triggering device, the military could direct gas in high concentrations to specific localities almost without warning. These attacks produced high casualties. With the development of the Livens projector, the British took the lead in gas warfare.[15(p64),20(p6)]

TYPES OF GASES

Scientists and the military separated gases into several general categories or classifications.

Acute Lung Irritants. These included chlorine, phosgene, diphosgene, and chloropicrin. When breathed in sufficient concentration, they created acute pulmonary edema, with death occurring from interference with the respiratory function of the lungs because of the accumulation of fluid and injury to the pulmonary alveoli.

Simple Lachrymators. These included xylol bromide which created intense smarting and watering of the eyes and interfered with a soldier's efficiency.

Vesicants. These gases, notably mustard gas, caused intense conjunctivitis, chemical scorching and blistering of the skin, and severe injury to the mucous membrane of the trachea and bronchial tubes. When death occurred it was usually because of secondary bacterial infection of the damaged and necrotic mucous membrane of the air passages and the development of severe bronchopneumonia.

Secondary Irritants. These included the chlorarsines (diphenylchlorarsine, ethyldichlorarsine), which caused sneezing; intense burning and aching pain in the nose, mouth, throat, and chest; a smarting and watering of the eyes; nausea; and mental depression.[23(pp79-80)]

Those gases that were directly and immediately poisonous included hydrocyanic acid, arsenide of hydrogen, sulphide of hydrogen, and phosphide of hydrogen. Those that produced slow death by asphyxiation included oxychloride of carbide, chlorine, acroleine, bromacetone, chloropicrin, methyl chloride of chloroform, and menthyl chlorosulphate. Finally, those that incapacitated the victim without doing permanent harm included chloracetone, bromacetone, iodacetone, bromacetic ether, and iodacetic ether.[24(pp70-71)]

In all, the Germans used 18 different gases against the Allies. The three principal gases were known as green cross, blue cross, and yellow cross. Green cross contained phosgene, alone or combined with chlorpicrin and chlorarsines. Green cross "3" contained various arsenic compounds. Blue cross contained chlorarsines, cyanarsines, and N ethyl carbazol; while yellow cross contained dichlorethyl sulphide or mustard gas. At first, these gases were easily distinguishable from others because of the small bursting charge of the gas shell and the color of the gas. Later, artillery crews mixed the gases with high explosive shells, making it difficult to determine whether a bombardment involved the use of gas.[11(pp83-105)]

Gas warfare entered a final stage in 1917, during the third battle of Ypres, when the Germans introduced mustard gas. This gas was sent over in shells which burst with a small concussion, making a hole about one and a half feet in diameter and a few inches deep. During the daytime, soldiers on the receiving end of this gas easily recognized it by the burst of yellow cloud. At night, however, mustard gas was indistinguishable from other types of gas shells. In the first night of its use, the Allied front sustained nearly 5,000 casualties from the gas alone. Mustard gas caused considerable attrition from its tenacious ability to render land, equipment, and clothes unusable for extended periods of time. Until the end of the war, it remained a serious source of casualties, causing chemical burns, especially in the presence of moisture, and neutralizing large areas of the war theatre. The victims of mustard gassing created an enormous amount of extra work for the Medical Services. All patients had to be stripped and bathed in a soda solution and given fresh clothes; moreover, attendants had to take precautions to avoid being burnt themselves. In addition, armies were forced to take elaborate measures to restore areas neutralized by the mustard gas.[19(p240),25(pp775-776)]

EFFECTS OF GASSING

Men who had become indifferent to the usual dangers of the firing line, including shrapnel and machine guns, became completely demoralized on the approach of a gas cloud. Most who had received severe wounds would, once sedated, be relatively quiet and comfortable; but this was not the case of men who were gassed.

> When we got to the hospital we had no difficulty in finding out in which ward the men were, as the noise of the poor devils trying to get breath was sufficient to direct us. We were met by a doctor belonging to our division, who took us into the ward. There were about twenty of the worst cases in the ward, on mattresses, all more or less in a sitting position, propped up against the walls. Their faces, arms, hands were of a shiny gray-black color, with mouths open and lead-glazed eyes, all swaying slightly backwards and forwards trying to get breath. It was a most appalling sight, all these poor black faces, struggling for life, what with the groaning and noise of the effort for breath. There is practically nothing to be done for them except to give them salt and water to try to make them sick. The effect the gas has is to fill the lungs with a watery, frothy matter which gradually increases and rises till it fills up the whole lung and comes up to the mouth; then they die; it is suffocation, slow drowning, taking in some cases one or two days. . . . The nurses and doctors were all working their utmost against this terror; but one could see from the tension of their nerves that it was like fighting a hidden danger which was overtaking everyone.[5(p419)]

Men on the receiving end of the gas cloud gave a fairly consistent description of its effects as the greenish-yellow fumes enveloped them. There was choking, coughing, gasping for air, the inability to speak, smarting of the eyes, and spasms of the respiratory tract. These initial symptoms were quickly followed by headache, weakness of the lungs, with many falling to the ground where the greatest concentrations of gas were retained. The soldiers experienced a burning sensation in their throats, a dry mouth, dry retching followed by the vomiting of yellowish fluid, irritation of the trachea, and pain behind the sternum. In this condition, many became easy victims of machine-gun fire as, half stupefied, they stumbled across No-Man's-Land in search of shelter, or lapsed into semi-consciousness on the battlefield.[13(p235)]

At a meeting of the Academie de Medecine of Paris, Dr R. D. de la Riviere of the Institut Pasteur and Professor J. Leclercq of Lille reported on the effects of chlorine and bromine on 112 soldiers exposed to the gases at Langhemarck. Doctors found the victims prostrated with irritation of the respiratory mucous membrane, many having died vomiting blood, and others crawling to the rear, vomiting and expelling bloody urine. The eyelids of the soldiers were swollen, their cheeks and lips violaceous, and their speech painful and jerky. Most of the men suffered from dyspnea and cough, a frothy expectoration, and inflammation along the entire respiratory tract. In some, the pulmonary lesions took the form of bronchopneumonia or gangrene of the lungs.[5(p417),18(p879)]

In a typical gassing case, the soldier evidenced restlessness, with a slow but full pulse. Sanitary personnel at the casualty clearing stations placed the victim in the open air or, during poor weather, in a room with a high ceiling and open windows, giving maximum opportunity for fresh air. The face of the gas victim was typically cyanosed and the expression strained and anxious. In most of these instances, the patient sat propped up with his head thrown back and gasping for breath; sometimes he chose to lay on his side with his head over the edge of the stretcher as a means of aiding expectoration. Respiration remained jerky and hurried, with much coughing and frothy expectoration.[26(p165)]

From the second through the fifth day, the violent coughing and vomiting generally ceased, followed by deep asphyxia, congested face, distended arteries, and shallow breathing. Expectoration continued, beginning with a yellow, highly albuminous fluid followed by a viscous yellow sputum, streaked with blood. During this period, the patient preferred to lie in the bed rather than sit propped up. In addition, he experienced a frequent and shallow cough, a mild temperature, and dry skin. Many patients died during this period from emphysema and generalized edema.[13(p236)]

Early in the war, physicians triaged gas casualties into three distinct classes. The first consisted of what they described as the "grey cases." Some, especially the older men, were in a condition of complete collapse; their faces and hands were of a leaden hue, and their heads fallen forward on their chests. The majority of these cases did not rally. A second class consisted of those lightly affected by the gassing who generally recovered by themselves. The third class required extensive treatment and, if neglected, died or were incapacitated for weeks, months, or even years. These patients were fully conscious and fighting desperately for life. They were restless, cyanosed, with a constant strangling cough and a desire to sit up. The pulse was rapid and bounding, respiration rapid and deep, and the body covered with a profuse cold sweat, with frothy expectoration amounting in some cases to 10 to 20 ounces.[25(pp774-775),27(p33)]

The most common symptom was asphyxia due to the lack of oxygen and the resulting filling of the lungs and bronchial tubes with fluid. Victims drowned in their own secretions. Severity depended on the concentration of gas in the air, the duration of exposure, the efficiency of precautionary measures, and the ability to respond medically to the situations presented. In mild cases, surgeons faced asphyxia and spasm of the glottis; in moderately severe cases, the symptoms included asphyxia and fluid in the bronchial tubes; and in severe cases, there was acute bronchitis with slow asphyxiation and bronchopneumonia.[13(p238),28(p48)]

TREATMENT

Initially, physicians were at a loss in treating the gassed soldier. There were simply no known remedies. Instead, medical personnel often had to stand helplessly by as men slowly drowned in their own fluids, making futile efforts to expectorate. Exhausted, they passed from semi-delirium into stupor, collapse, and death. The vesicant gases were particularly insidious. Experiencing no immediate irritative properties, soldiers often worked or slept for hours before feeling its effects. Sanitary personnel removed their clothes, bathed them with soapy water, provided them with steam inhalations and morphine, only to find them dying a lingering death from bronchopneumonia days or weeks after the gassing. Although mortality rarely exceeded 5% of the men incapacitated by vesicant gases, incapacitation often lingered for weeks and months after an attack.[29(p126)]

Initially, physicians ordered victims to inhale the fumes of whisky, brandy, rum, or pure alcohol through a moistened cloth. They also recognized the need to rid the lungs of the gas, just as it was important to rid the lungs of water in near drowning. Depending on the degree to which the soldier had inhaled the gas, physicians prescribed dilute ammonia fumes, inhalation of a spray solution of hyposulphite of soda, oxygen by inhalation and subcutaneously, and atropine.[5(p422)]

Physicians also induced vomiting and performed venesection to prevent edema of the lungs. The cases in which physicians employed venesection were generally identified as "cyanotic," with the heart becoming increasingly weaker from the strain. With venesection, patients found immediate relief, enabling them to breathe and sleep more easily, and lessening acute headache. Physicians generally chose direct incision into the median cephalic or median basilic vein, with the amount of blood extracted depending on the patient. From plethoric and alcoholic types, surgeons extracted 20 to 25 ounces, and from elderly or less robust patients, 15 to 20 ounces.[30(p108)]

The New Haven Laboratories, originally under the Bureau of Mines and later taken over by the Chemical Warfare Service under the leadership of F. P. Underhill and Yandell Henderson, tested the effectiveness of venesection on animals following exposure to lethal gases of the chlorine-phosgene type. Additional tests were carried out at the British Experimental Field by Capt Samuel Goldschmidt, USA, and Capt D. W. Wilson, USA. The results in both laboratories suggested that the mortality of gassed animals subjected to bleeding dropped compared to untreated controls. The Medical Committee of the British Chemical Warfare Committee subsequently accepted these results as the basis of its own recommendations.[31(p130)]

Besides resorting to venesection to support a failing heart, physicians prescribed pituitary extract and brandy as well as hypodermic injections of camphor and caffeine. Another treatment involved subcutaneous injections of oxygen in the pectoral region, "the amount being sufficient to cause a lump in each side of the chest about the size of a small football."[26(p166),15(p228)] The technique involved using a cylinder of pure oxygen, rubber tubing, and a hypodermic or small aspirating needle. Physicians directed nurses to insert the needle into loose subcutaneous tissue; when a swelling occurred, they removed the needle and massaged the area to aid diffusion. They continued this treatment until approximately 1.5 liter (50 ounces) of oxygen were injected each day.[32] In milder cases, inhalations of steam mixed with compound tincture of benzoin in a closed tent gave relief. In cases of marked mental strain and restlessness, physicians administered tincture of opium, which gave relief and allowed the patients to sleep.[26(p166)]

Occasionally, soldiers requested linseed poultices on their backs in the belief that it would relieve congestion by drawing blood away from the lungs to tissues lying under the poultice. Using the same principle of counter-irritation, some applied hot water bottles to the feet and other body surfaces, particularly the abdomen.[33]

Postmortem examinations of gassed victims indicated that many died of acute congestion and edema of the lungs. For this reason, physicians encouraged patients to expel the excessive secretions from the lungs with the use of emetics and stimulating expectorants. A mixture of salt and water, administered in 10-ounce doses, followed by draughts of lukewarm water was given as an emetic. This induced vomiting and almost always brought relief, bringing up quantities of frothy yellowish fluid. Medical personnel often followed forced vomiting with artificial respiration using the Schafer method, and then administered ammonium carbonate every several hours as a stimulant and expectorant.[7(p119),26(pp165-167)]

Experiments with animals showed that the frothy fluid in the air tubes, which often drowned the victims, could be squeezed out of the lungs and trachea by rhythmic compression of the thorax. To achieve this in gas victims, doctors resorted to mouth-to-mouth respiration. Scientist L. Hill reported success in Flanders with an apparatus called the Vivator, which consisted of a foot-pump, face mask, and flexible tube. With each downstroke on the pump, the operator forced oxygen into the lungs and, by each upstroke, the operator drew fluid from the lungs by the recoil of the thorax and lungs.[7(p119)]

Surgeons warned against treating gas victims in dugouts or advanced dressing stations below ground or in a confined area, since the gas from the soldiers' clothes contaminated the area and incapacitated surgeons and medical personnel. Medical personnel preferred to treat gas cases in an open field, and many advanced dressing stations even posted an NCO at the entrance to ensure that no gas victims entered the dugout for treatment.[19(p241)]

In treating large numbers of gas casualties, medical administrators were challenged to meet the needs of the army with existing organizational structures. The American, British, and French created separate stations for treating gas casualties, but only the French retained the separate stations (called Z-system) through the end of the war. The British and Americans eventually abandoned the separate stations and used regular stations augmented with special medical personnel trained to treat gas victims.[6(p257)]

During the battle of the Somme, medical personnel introduced the gas-treatment center to provide support for the large numbers of soldiers gassed with mustard. Immediately on arrival of gas victims, personnel rid soldiers of all clothing and equipment contaminated with the gas. They then washed the victims with warm soapy water treated with bicarbonate of soda or lime water, and covered their burns with a dusting powder of boracic acid, chalk, starch, and zinc oxide, or calamine lotion. Because of the imminent danger of the gas to the eyes, personnel took special care to wash them with bicarbonate of soda or warm boric lotion. Doctors tried never to bandage the eyes, since it left soldiers with the impression of permanent eye injury. They considered it important to treat mustard gas poisoning as a "benign contagion" by adopting a strict and hopeful attitude; more guarded forms of treatment tended to create a severe depression among patients suffering from the effects of gassing.[15(pp231-232)]

In response to the need for early bathing of men exposed to mustard gas, the Medical Director of the Chemical Warfare Service ordered construction of the first mobile degassing station, which the Service built and demonstrated before representatives of the AEF, French, British, and Italian armies at Tours, France, in July 1918 (Fig 1). The AEF adopted the degassing stations as part of its field equipment and employed them along the western front under actual field conditions. A school of instruction was set up near Vouvray, France, where men from the AEF received special training in operating the stations.[34(p544)]

MASKS AND OTHER DEFENSIVE MEASURES

On April 23, 1915, the Royal Army Medical College received the first report, written at the scene of the attack, of the effects of the chlorine cloud that blew into the trench lines. It became obvious that front-line divisions had to be protected if they were to hold their positions. This meant the need for immediate assistance from the scientific community. In the emergency, Britain's War Office turned to the Army Medical Service to supply protective appliances for the troops. This meant the location, purchase, and assembly of materials and chemicals on a huge scale. At the government clothing factory, fleets of cars rushed cotton-waste wads to and from private laundries where the material was dipped and dried. Within 60 hours, the British had provided 98,000 pads of cotton waste packed in muslin containers and dipped in hyposulphite solution. The service sent another 300,000 in the next week and 2,000,000 within a month. The same hectic activity occurred with the French who, within days of the initial attack, had prepared 100,000 crude masks for their soldiers at the front.

By May 4, 1915, the British had provided their front-line soldiers with pad respirators made from five to six layers of flannel or cotton wool, wrapped in muslin, and soaked in a solution of sodium carbonate, hypo (sodium thiosulphate), and water. In addition, pads of cotton waste were kept in boxes scattered about the trenches and, during the gas attack, "eight or ten men would dive for the box, grab a pad of cotton waste, stuff it into their mouths and nostrils, and then fasten the pad over it."[17(pp3–4)]

Certain defects became immediately obvious in the use of the pads supplied or improvised at the front lines. Although they stopped a considerable amount of chlorine when properly cared for and adjusted around the nose and mouth, the problems outweighed the benefits. For example, soldiers needed to dip the pads in an alkali solution prior to wearing; if they did not wring out the excess fluid before attempting to breathe, the mask impaired breathing. By the time they had correctly replaced their masks, soldiers had often breathed toxic levels of the gas. Also, the early masks were placed in the trenches rather than carried by the soldiers. During the attack, several men might rush for the same mask.

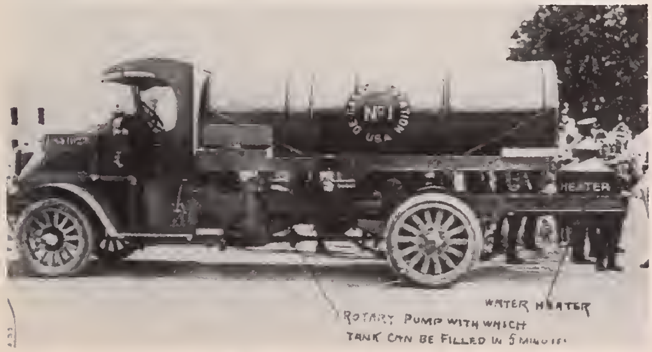

FIGURE 1. Mobile degassing station (reproduced from Gilchrist[34]).

The Black Veil Respirator, which replaced the improvised pad and pad respirator, consisted of a four-fold piece of black veiling a yard long and 8 inches wide, containing an 8-inch square of cotton wadding saturated with a solution of sodium carbonate, hypo, glycerine, and water. The black cotton netting was the same as that used for women's veils. First worn by the British during the German attack against Hill Sixty on May 10th, the respirator proved chemically effective but, because it did not fit snugly across the face, the British continued to suffer casualties.[11(p45)]

In order to enlarge the area of filtration, the Royal Army Medical College developed a third respirator known as the Pimlico helmet which provided 3.5 square feet for air entry during inspiration. This helmet, sometimes called the "Hypo" or "smoke" helmet, did not differ substantively from the helmet introduced by the Germans. The Pimlico consisted of a flannel bag worn over the head and tucked under the shirt or coat collar. As designed, however, the helmet had no outlet valve, which meant that air inside the mask soon became laden with carbon dioxide.[17(pp3–4)]

The demands for the khaki woolen textile used in these helmets exhausted available supplies and required extensive purchasing of smaller allotments. In initial tests on the permeability of the cloth, the Royal Army Medical College enlisted young men to run around the college yard while wearing the helmet; later the college used a manometric device for more rapid testing. There were also problems with the color of the khaki, since front-line soldiers wearing light-colored helmets made easy targets for enemy marksmen. To resolve this problem, scientists mixed and dipped a variety of dyes and tested their effect on the absorbent chemical solution required in the fabric to protect the wearer. Similar problems occurred with the selection of the eyepiece for the helmets. The single window pane or eyepiece made of mica proved too brittle and cracked easily. Moreover, the needles used to stitch the eyepieces were too large for the thread, leaving minute holes around the stitching which admitted unfiltered air into the helmet. Non-flammable cellulose acetate worked, but not enough could be supplied for manufacture. Efforts at using chromacized gelatin also proved disappointing. Finally, the military adopted glass disks fixed in tin rims, and later non-splintering triplex glass.[35(pp541–542)]

Shortly after the British introduced the Pimlico helmet, the Germans introduced a more deadly gas, phosgene (hydrocyanic acid). Nearly five months before its actual use, British intelligence discovered the German intent and learned as well the date of the intended attack and the sector of the front where it was to be tested, the Messines Ridge.[17(p4)] As a result of this advance notice, the British devised the P-helmet (sometimes called "tube helmet") which had the same general shape as the Pimlico helmet but had a longer skirt, a glass eyepiece, an outlet valve, and mouthpiece. This allowed the wearer to discharge carbon dioxide outside the helmet rather than through the fabric. The valve also permitted the soldier to increase the level of his exertion. Besides the hypo, sodium carbonate glycerine, and water solution, the British added sodium phenate and later added hexamethylene-tetramine at the suggestion of the Russians. With the addition of the hexamethylene-tetramine, the helmet became known as the PH helmet (the PH standing for phenol and hexamene), and gave its wearer an absorption of 100% against 0.1% of both carbonyl chloride and hydrocyanic acid gas for an hour.[17(p5),35(p548)] When, on December 19, 1915, the Germans launched their phosgene and chlorine attack against Messines, the defending troops were sufficiently protected. Nevertheless, casualties occurred from failure to place masks quickly enough, breathing too heavily and inhaling faster than the mask could filter the air, and removing the mask too early. Despite these errors in judgment, the PH helmet became a worthy protector of troops, with some 27,000,000 manufactured between July 1915 and February 1918, when the box respirator became the sole issue.[15(p68),17(p5)]

The constant rumors that the Germans intended to use more lethal gases created a high degree of paranoia within the scientific community. Advice from the industrial community on the difficulty of manufacturing these more exotic gases reduced some of the anxiety. More importantly, however, the scientific community decided on a single polyvalent helmet against all the potential gases. For the British, this meant development of a helmet that could protect a soldier from no less than 43 different gases.[35(p545)]

As scientists considered each new gas as a possible offensive weapon, the British Museum Library searched out all possible information, while chemists in the Imperial College of Science and Technology and the University of London Laboratories proceeded to make each gas in a quantity sufficient for testing. Then began the investigation of possible absorbents and the efficiency of the absorption material using the helmet and laboratory rats as test specimens. If the rat survived, scientists repeated the test on a pig; and if the pig survived, chemists then tested the gas on one of their fellow investigators. According to P. S. Lelean, professor of hygiene at the Royal Army Medical College, "everyone had at one time or another been incapacitated by an overdose of the simpler gases, but there was no time to do more than lay the victim on a bed that was kept ready in the laboratory; give oxygen and administer chloroform vapor, and leave an orderly to watch him until he recovered. There were no other means of treatment, for at that time no others had been found, or opportunity to seek them. But the victim was no sooner on his feet than he was back at his special work, shaken and ghastly, but resolute." Learning that a pig died suddenly a day or two after an experiment and knowing that a volunteer had donned the experimental helmet and completed an hour's test in the lethal chamber added a certain piquancy to the testing.[35(p546)]

By December 1915, the German army decided to use lachrymatory, or tear gas, sending it into the Allied lines in artillery shells. The Germans also began mixing their gases, forcing the Allied troops to use masks as well as goggles as protection. Unfortunately, soldiers found the PH helmet ineffective against tear gas, and this led to the addition of rubber-sponge goggles in each helmet. This innovation, called the PHG helmet, was soon discarded because of the length of time required to adjust it.[17(p6)]

In February 1916, the British developed the so-called tarbox respirator issued to troops in support areas, including artillerymen. However, the helmets were cumbersome and more difficult to breathe through than the PH helmet. In April 1916, the British introduced the small box respirator, or SBR, which, by October 1916, replaced the tarbox respirator as well as all previous forms of respirators (Fig 2). This respirator consisted of a canvas satchel divided into two compartments. At the lower end was a canister with a rubber valve which, when open, allowed the soldier to inhale air through chemicals consisting of soda lime, charcoal, potassium permanganate, sodium phenate, and hexamethylene. These chemicals either absorbed, neutralized, or oxidized the gases as they passed through. Connected to the canister was a flexible respiratory tube which attached to a mouthpiece that fit between the lips and the teeth. However, scientists soon discovered that gas tended to pass up the smooth interior against the side of the box. In order to correct this defect, they corrugated the sides of the box, leaving large air spaces above and below the absorbent layers to equalize the flow of air. The respirator system also included a nose-clip and a glass eyepiece. So constructed, the respirator was polyvalent against most gases and gave only slight resistance to respiration.[17(p6)]

The French mask, called the M2, consisted of 32 layers of muslin impregnated with absorbent chemicals and covered with a waterproof fabric. The soldier exhaled

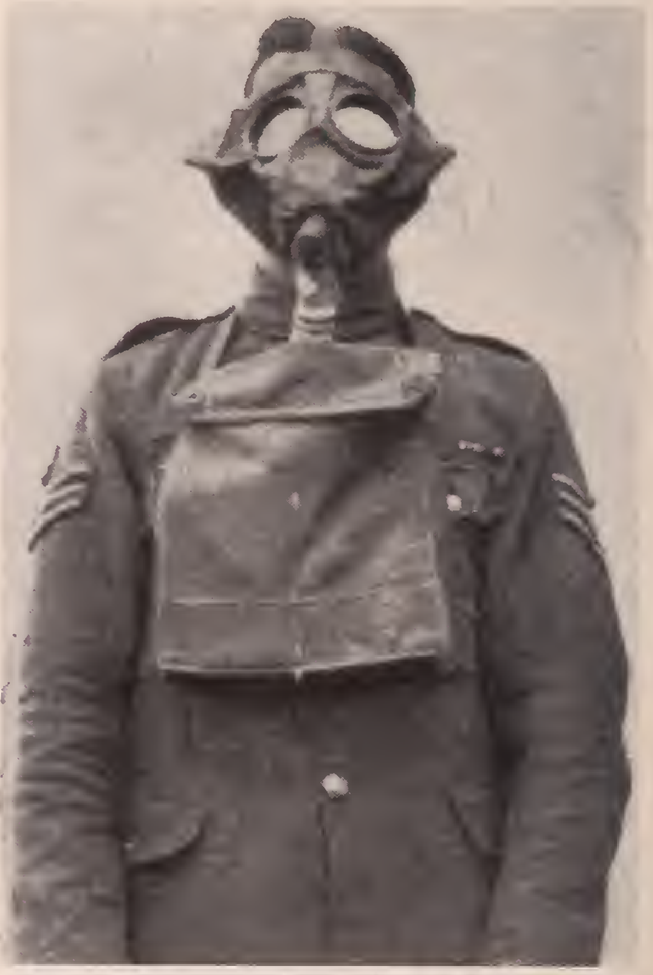

FIGURE 2. British small box respirator (reproduced from Macpherson et al[16]).

through the impregnated fabric and was also protected by a celluloid eyepiece. The mask proved very effective and many in the American Expeditionary Force carried the M2 mask as well as their own. Like the earlier British mask, however, it had no outlet valve, which meant that air drawn in had to be exhaled through the fabric. Later, the French adopted the so-called ARS mask which had an outlet valve. It resembled the German mask except that the French used a rubberized fabric facemask instead of the German leather mask (Fig 3). The Italians designed a mask similar to the French M2 mask but discarded it in 1918 for the British small box respirator. The Russians used a box canister filled with charcoal and a headpiece made of rubber.[11(pp76–77),17(pp8–9)]

The earliest German respirators were not much more than pads soaked in a sodium-thiosulphate-sodium carbonate solution. This they replaced with a mask which fit over the mouth and nose and worked on the same principle as the British and American respirators in that the soldier inhaled through a canister filled with chemicals. The Germans constructed their facemask of leather and treated it with oil to make it gas- and water-tight. The canister originally contained granules of baked earth soaked in a solution of potassium carbonate and covered with powdered charcoal, charcoal, and crushed pumice stone mixed with hexamethylene. In April 1918, the Germans replaced the baked earth saturated with potassium carbonate with a layer of charcoal zinc oxide. Unlike the British and American respirator, the German gas mask had no outlet or expiratory valve. Thus the soldier exhaled through the canister, which carried moisture into the chemicals, causing the canister to deteriorate more quickly[17(p7)] (Fig 4).

As for the Americans, in April 1917, a subcommittee of the National Research Council, under the chairmanship of the director of the Bureau of Mines, and consisting of representatives of the Army and Navy, civilian chemists, gas experts, and physiologists, initiated extensive research on gas warfare. Immediately following the United States' declaration of war on Germany, an American commission composed of George A. Hulett of Princeton University and Professor Amos of Johns Hopkins University traveled to France, where they joined with Major James R. Church to report on developments of gas warfare. This commission concluded that the British methods of gas defense were superior to those of the French and that the Ameri-

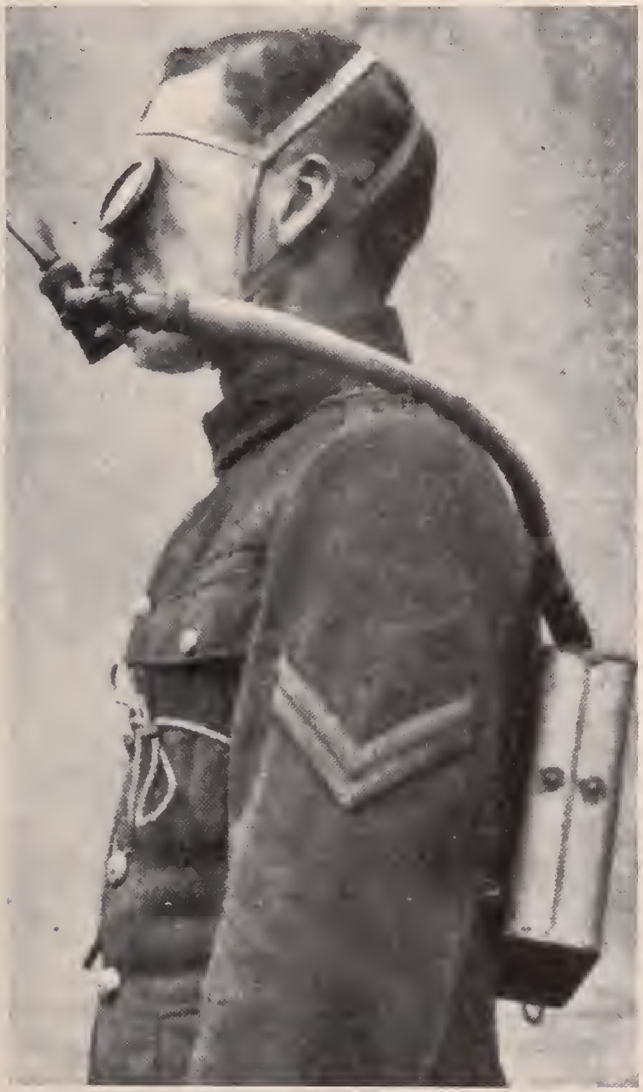

FIGURE 3. French respirator, Tissot pattern (reproduced from Macpherson et al[16]).

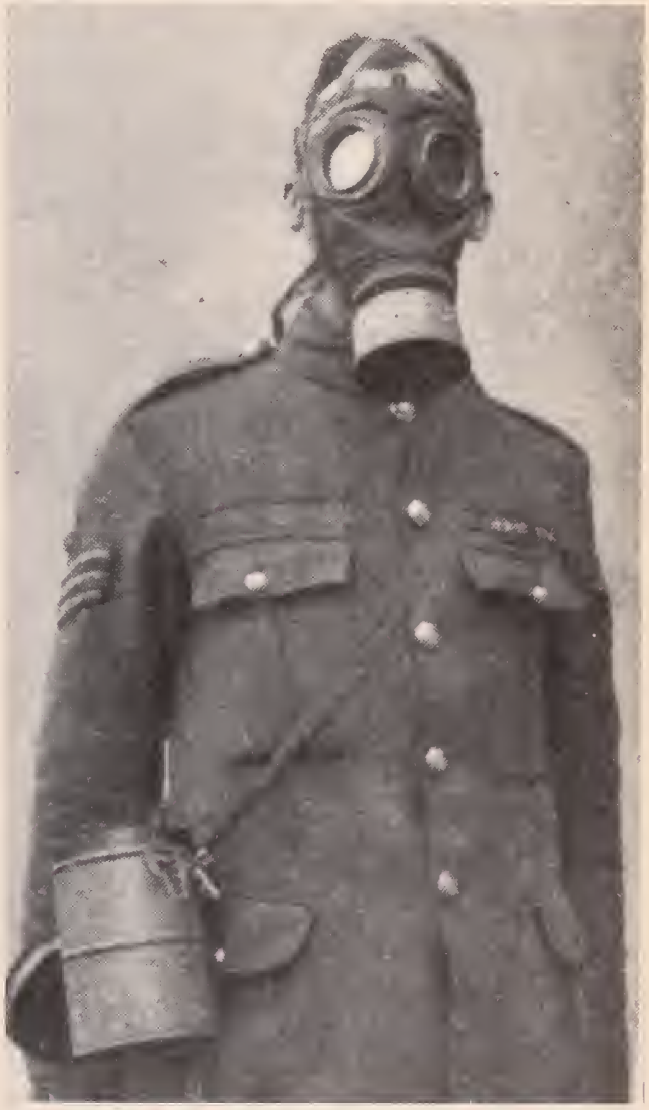

FIGURE 4. German respirator (reproduced from Macpherson et al[16]).

TABLE I. Types of Gas in Stages[23(p79),16(p271)]

Period	Method of Discharge	Gas Used	Troop Protection
April and May 1915	Cylinders	Chlorine	Cotton wastes; respirators in last attack
April 1915 to July 1916	Gas shell	Simple lacrymators	Cotton waste; respirators, goggles, fabric helmets (Hypo, P, PH, and PHG); a few large box respirators
December 1915 to August 1916	Cylinders	Chlorine and phosgene	Fabric helmets (P, PH, and PHG) and a few large box respirators
July 1916 to July 1917	Gas shell	Phosgene, diphosgene, chloropicrin, and lacrymators	PH or PHG and then the small box respirator
July 1917 to end of war	Gas shell	Mustard gas, phosgene, chloropicrin, and chlorarsines	Small box respirators
December 1917 to May 1918	Projectors	Chlorarsines, phosgene, diphosgene, chloropicrin	Small box respirators

can soldier should wear masks of the British small box respiratory type. Colonel Amos A. Fries, Chief of the Gas Service of the American Expeditionary Forces, ordered 100,000 of the small box respirators from the British Government, and these were distributed to the first troops arriving in France. The Gas Service awarded subsequent orders to companies in the United States. Eventually, a modified American Tissot mask, modeled from the French type, went into service in October 1918.[17(pp9-10)]

In the British and American armies, military authorities required soldiers to carry the respirator when traveling or posted within 12 miles of the front. Between 12 and 5 miles a man could remove the respirator box in order to sleep, but within five miles, even this was forbidden. Within two miles, military rules required soldiers to wear the mask in the "alert" position (slung and tied in front) and be able to make it fully operational within six seconds of a gas alarm. In addition, soldiers maintained a log noting the amount of time the apparatus was worn during cloud gas attacks or shell bombardments since the effective life of the container was approximately 40 hours.[15(p70),22(p57)]

TECHNIQUES TO REPEL GAS

The techniques suggested by inventors to repel or modify the character of the gas as it drifted across the trenches included fans of various types, airplane propellers set up at specific points and driven by electric motors, counter currents of ascending hot air produced by burning gasoline or other highly volatile materials, and projecting ammonia vapor or water towards the advancing cloud. But as one medical inspector noted, "the mechanical fans are as futile as the broom of Canute, and hot air currents and ammonia are quite likely to be wafted back on those who send them."[5(pp420-421)]

Sir Hiram Maxim paid particular interest to the degree to which gas diluted as it traveled. He developed a theory that by the time the gas moved 20 feet, the volume of chlorine was roughly one in five. By the time it reached the trenches of the opposing army, the dilution approximated 1 to 100. Given this ratio, Maxim designed and built incendiary bombs to create a rapidly spreading fire in the path of the advancing gas. As the gas wafted across the lines, Maxim intended the fires caused by the incendiary bombs to heat the air, causing an upward rush, driving the gas 20 to 40 feet above the ground and away from the trenches.[18(p881)]

As gas warfare became more sophisticated, armies learned how to deal with its various nuances. For example, when soldiers evacuated dugouts, pill-boxes, or aid-posts during a gas attack, they did not return until any remaining gas had been dissipated with fire. The military also devised a gas-proof curtain to prevent gases from entering dugouts. The curtain, treated with a solution of sodium thiosulphate and sodium carbonate in water, covered the entrance completely, with about 9 inches of additional cloth resting on the ground. Most dugouts had two curtains, one for the upper entrance to the dugout, and one closer to the bottom staircase. In cases of slight contamination, military personnel washed the dugout with a stiff brush and water, sprinkling the area with chloride of lime. However, if the dugout was heavily contaminated, the military usually evacuated and destroyed it.[15(p71)]

Mustard gas required special protections for gunners and stretcher-bearers who were required to handle equipment. In addition to respirators, they wore special leather gloves treated with linseed oil and often wore clothing treated with boiled linseed oil, castor oil, and paraffin wax. To decontaminate clothing, sanitary personnel treated it with steam or hot soapy water, chlorine gas, and exposure to fresh air. Motor ambulances needed thorough washing after carrying mustard gas victims.[15(p71)]

RETROSPECT

Gas warfare was believed to be more deadly and inhumane than any of the other instruments of killing or maiming human beings. In part, this attitude resulted from the Allied propaganda which rebuked Germany's use of chemical warfare as a reflection of the Hun's historical barbarism and lack of honor on the battlefield. According to Ludwig Haber in his book *The Poisonous Cloud* (1986), it became a "journalistic event" where accuracy took a backseat to the message of German indifference to human suffering and the rules of war.[11(p231)] Reports of men struggling in torment, gasping and choking, and dying in acute agony created the public impression that gas casualties were higher and more horrendous than those from more conventional weapons. To be sure, the early months of gas warfare were cruel, ghastly, and inhumane because there were no defensive mechanisms in place. For those medical personnel and soldiers who had to stand by helplessly as patients and comrades suffered, gas remained a barbarous instrument that violated the rules of combat and fair play. The public formed an opinion on gas in the shock of the chlorine gas cloud attack in April 1915 before any form of gas defense had been developed. But almost immediately, the Allies organized defensive tactics against the new weapon. Within months after the Allied armies retaliated with gas clouds of their own, discussion of gas warfare took on a more technical and defensive tone, replacing earlier attitudes of unfairness and violations of the rules of war.

Estimates on the total number of casualties from gas suggest that there is no reliable total. Prentiss estimated casualties as high as 1,297,000.[36(pp653-654)] Haber, on the other hand, suggested that casualties for Britain, France, Germany, and the United States was just under 500,000 which amounted to between 3% and 3.5% of the total killed, missing, or wounded on the western front.[11(pp239-242)]

For many who studied the use of asphyxiating gas in warfare, including Alfred T. Mahan, the American representative at the First Hague Conference in 1899, it was no more inhumane or unnecessarily cruel than other weapons in the armamentarium of nations. In fact, many believed that gas was actually a more humane weapon, if both sides were prepared for it. According to Lieutenant Colonel Albert P. Francine, writing in 1923, gas was 12 times a better risk than bullets and high explosives. Of the total battle casualties from all causes during the war among US troops, about a third were due to gas and, of that number, less than 2% died. This compared most favorably to injuries by gunfire and high explosives, of which 24% of the victims died. In other words, chemical warfare put more men out of action with less hurt than any other instrument of war. As Francine noted,

> So when I think of the hundreds of men littered in the great gas hospital every night or so, and the overflow [that] had to be cared for in adjacent hospitals, I do not picture men who were coughing, choking or gasping for breath, for they were not; but of men cold and exhausted from exposure and want of sleep, quiet, or occasionally laughing, fellows burned more or less seriously about the eyes and body. Casualties all right, but not suffering like torn men, and not to be permanently disabled or even scarred. Approximately 90% of the cases in the hospital were suffering from mustard gas, and 80% of these were not seriously gassed. In only 20% was the condition severe enough to confine the men to bed for more than 48 hours. The death rate in the hospital was 1.98%, and all deaths were from mustard gas. This percentage again goes to show that the mortality and effects of gas, where gas defense had become well-developed, have been, as was to be expected, greatly exaggerated in the minds of laymen, doctors, and soldiers themselves, who did not study it closely, and among large groups.[37(p368)]

Four-fifths of the deaths from gassing occurred within the first 24 hours; if the patient survived that initial period, he usually recovered. In all, gas caused 70,552 casualties among the Americans. Of that number, 1,221 died. In other words, approximately one of every three American soldiers in hospitals suffered from exposure to gas. However, less than two men out of each hundred (1.7%) died of gas, while more than nine out of each hundred died from other battle causes.[38(pp16-18)] Thus, supporters of chemical warfare were convinced that, rather than being more inhumane than other methods, gas had actually been "a refinement in warfare."[37(p370)]

On reflection, chemical warfare failed to achieve the military significance intended by its creators. The military, according to Haber, lacked commitment to this new mode of fighting and "pursued it haphazardly." Added to this, the planning and implementation of gas warfare was "amateurish" and, on the whole, defensive measures sufficient to contain the potential threat of this new weapon were developed.[11(p262)] Staff officers on both sides were reluctant players in gas warfare, hesitant to break with traditional weaponry for something new and unproven. Commanders and their staffs were unfamiliar with the concept of chemical warfare, however imperfectly defined and elaborated. For the duration of the war, it remained largely outside their frame of reference. "When one reads the analyses of cloud-gas attacks or the monographs of the chemical warfare specialists," observed Haber, "one is struck by the pedantry of the technical details and by the absence of all theory, or rather the inability to fit the operations into existing military thinking."[11(pp266-267)] Lacking a tactical purpose, gas warfare heralded no breakthrough for either side in the conflict. Other novel weapons—the tank, airplane, light machine-gun—fared better in terms of integration into battle tactics. Chemical warfare, once hailed as a solution to trench warfare, proved negligible. The need for close harmony of officer, soldier, civilian chemist, industrialist, and the weather never materialized at the level needed to influence matters of policy and strategy. In the end, chemical warfare became less a tactical weapon than a psychological one.

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Crack abuse and asthma: A fatal combination

A. NAVEEN RAO, MD; PETER G. POLOS, MD, PHD; FREDERICK A. WALTHER, MD

The National Institute on Drug Abuse refers to cocaine as "the drug of greatest national public health concern."[1] Cocaine is now being widely sold in a potent inhalable form known as crack. The simplicity and low cost of its preparation promotes crack's ready availability to increasing numbers of users.[2] During the past decade, because of the introduction of crack, there has been a greater incidence of cocaine-associated medical complications.[3] This report describes a patient with asthma who experienced fatal bronchospasm related to smoking of crack.

CASE REPORT

A 28-year-old man first entered the emergency department of the New York Infirmary-Beekman Downtown Hospital with acute shortness of breath. His medical history was significant for bronchial asthma since childhood. His medications included sustained action theophylline (Theo-Dur), 300 mg twice daily, albuterol (Proventil), 4 mg three times daily, and metaproterenol sulfate (Alupent) by inhaler as needed. He denied cigarette smoking.

Examination revealed an alert, anxious, young man in mild respiratory distress; he was using his accessory muscles of respiration. His blood pressure was 136/84 mm Hg without pulsus paradoxus; pulse rate, 100/min; respiratory rate, 26/min; and temperature, 97.8°F. A complete physical examination disclosed only bilaterally decreased air entry and diffuse expiratory wheezes in both lungs. Basic laboratory data, a chest radiograph, and an electrocardiogram were all within normal limits. Arterial blood gas determinations (breathing room air) were as follows: pH, 6.98; P_{CO_2}, 44 mm Hg; P_{O_2}, 87 mm Hg; and bicarbonate, 17 mosm/L.

Following treatment with subcutaneous epinephrine, 0.25 mg, intravenous aminophylline infusion at 40 mg/hr, and metaproterenol sulfate, 0.3 mL inhalation via high flow nebulizer, the patient's wheezing and respiratory distress resolved. He was discharged to home and advised to continue his usual medication, together with ampicillin, 250 mg every six hours.

Five hours later, while at home, the patient again experienced difficulty in breathing. The emergency medical service found the patient in cardiopulmonary arrest. Advanced cardiopulmonary life support techniques were applied, and intubation was performed. On the patient's arrival in the emergency department, life support techniques, dopamine, and isoproterenol were administered. Arterial blood gas determinations on 100% oxygen revealed pH, 6.88; P_{CO_2}, 72 mm Hg; P_{O_2}, 518 mm Hg; and bicarbonate, 13 mosm/L. On admission to the medical intensive care unit, physical examination revealed a comatose man with an endotracheal tube in place and respirations assisted by a mechanical ventilator. His blood pressure was 100/60 mm Hg; pulse rate, 120/min; respiratory rate, 20/min with assisted ventilation; and temperature, 98.0°F. The positive physical findings included fixed and dilated pupils, bilateral decreased air entry, and diffuse expiratory wheezes in both lungs. There was absence of deep tendon reflexes and no response to deep painful stimuli.

The electrocardiogram indicated sinus tachycardia at 120/min, with ST depressions in the inferior and lateral leads. The chest film showed no infiltrates and confirmed the correct position of the endotracheal tube. The serum theophylline level was 15.2 mg/dL. A toxicologic screen of urine was positive for cocaine and cocaine metabolites. The patient's family, when informed of this finding, confirmed the patient's history of crack abuse. Apparently four months earlier, the patient's elder brother, a crack addict, had died of an acute exacerbation of bronchial asthma.

On admission to the medical intensive care unit, the patient was treated with intravenous aminophylline, methylprednisolone, metaproterenol by nebulizer, ampicillin, and ceftriaxone. Dopamine was required to maintain adequate blood pressure. A bedside electroencephalogram disclosed a "flat line" electrical response consistent with electrocerebral silence. A positive apnea test confirmed clinical brain death. All life support measures were discontinued, and the patient was pronounced dead.

Autopsy at the office of the Chief Medical Examiner revealed anoxic encephalopathy secondary to irreversible bronchospasm. A toxicologic screen by the medical examiner also revealed cocaine and cocaine metabolites in both blood and bile.

DISCUSSION

Unlike the production of freebase, which is a dangerous process, crack is easily made by mixing cocaine hydrochloride with baking soda and water. The name crack was given to this form of the drug because of the sound it makes when heated for smoking. Crack smoking produces an immediate euphoric effect, which strongly reinforces the compulsion to repeat the experience. Given unrestricted access to cocaine, animals will self-administer the drug with a greater than 90% mortality.[4] The South American coca leaf chewer consumes 200 mg of cocaine per day; the modern suburban crack smoker uses 1,500 mg per day.[5]

The clinical course of this asthmatic patient suggests the ability of crack abuse to trigger fatal, irreversible bronchospasm. Functional disorders of the lungs, gas exchange abnormalities, and reactive airway disease occur with increased frequency among freebase cocaine abusers.[6,7] Pulmonary infiltrates, fever, and bronchospasm (crack lung) can be caused by inhalation of cocaine.[8] The medical literature offers little information on the association of asthma and crack abuse.[9]

The toxic effects of cocaine are due to stimulation and subsequent depression of the cardiovascular, respiratory, and central nervous systems. Central respiratory depression with reactive bronchospasm can be fatal in asthmatic patients. The underlying mechanisms for intensified bronchospasm in asthmatic crack smokers may be complex: bronchial hyper-reactivity similar to that caused by other nonspecific offenders (cold air, certain chemicals,

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stress, exercise); immunologic reactions with specific IgE formation; direct liberation of bronchoconstrictor substances; possible drug interactions between active cocaine metabolites and asthma medications.[8,9]

Many of the signs and symptoms of cocaine toxicity rapidly resolve as the drug is metabolized and require no treatment. Since no specific antidote for cocaine poisoning exists, serious side effects are treated based on their symptoms. Bedside pulmonary function tests, especially the performance of forced vital capacity maneuvers, are useful to measure the degree of obstruction present and to follow response to treatment. The therapeutic strategy is to support ventilation and circulation.[10]

The present case suggests the possibility that certain patients with pre-existing asthma may suffer fatal irreversible bronchospasm with crack smoking. The increasing abuse of crack intensifies the need for physicians to be aware of crack's adverse and potentially fatal effect on the asthmatic patient.

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Eosinophilia-myalgia syndrome and L-tryptophan

RICHARD L. GOLDEN, MD

A cluster of three cases comprising eosinophilia and severe myalgia in patients taking oral L-tryptophan preparations was first noted in New Mexico in October 1989.[1] Since that initial report, the disease has assumed epidemic proportions, with 1,269 cases reported nationwide as of February 1990. Thirteen deaths have been reported among patients with eosinophilia-myalgia syndrome. A statistical analysis of the first 429 cases showed the dose of L-tryptophan to have been from 0.026 g to 15.0 g per day, with a median of 1.5 g per day; the patients ranged in age from 11 to 84 years, with a median age of 48 years. This new syndrome, still in the process of delineation, has been characterized by severe, incapacitating myalgias, eosinophilia, weakness, arthralgias, fever, pulmonary signs or symptoms, rash, and edema.[1-4] In addition, an ascending polyneuropathy, vasculitis, heart failure, arrhythmias, thromboembolic phenomena, and hepatomegaly have been noted in some patients.[5]

CASE REPORT

A 60-year-old man presented with a three-week history of generalized muscle aching, which was at times severe, dryness of the mouth, episodic sense of flushing, a low grade fever, and a productive cough. He had been in good health following triple coronary artery bypass graft surgery nine months earlier. His medications included aspirin, 0.3 g/day, and multivitamins. In addition, he had been taking L-tryptophan for insomnia, averaging 0.75 g/day for the previous four months. Each capsule was formulated with 0.5 g L-tryptophan (of Japanese manufacture) and 10 mg of pyridoxine. Physical examination was significant for mild conjunctival injection, mild tremor, a trace of pedal edema, and an absent vibration sense.

Laboratory studies disclosed the following pertinent values: white blood cell count, 1.2×10^9/L, with 38% polymorphonuclear forms, 11% lymphocytes, 5% monocytes, and 46% eosinophils; eosinophil count, $4 \times 10^3/\mu$L; lactic dehydrogenase, 329 IU/L (normal, 100-250 IU/L); alanine aminotransferase, 56 IU/L (normal, 0-50 IU/L); total protein, 5.5 g/dL; albumin, 3.3 g/dL; aldolase, 10.6 U/L (normal, 1.2-7.6 U/L). Creatinine phosphokinase, rheumatoid arthritis latex screen, antinuclear antibodies, erythrocyte sedimentation rate, and a serologic test for *Trichinella* antigen were negative or within normal limits. The chest roentgenogram showed increased bronchovascular markings at the right base and was otherwise normal. An electrocardiogram showed an old anteroseptal infarction.

The patient was advised to stop taking L-tryptophan at the time of the initial examination. Over the next month he reported mild improvement in his muscle pain and cough. The eosinophil count decreased to 3.2×10^3/mL; lactic dehydrogenase to 287 IU/L; and aldolase to 8.2 IU/L.

DISCUSSION

Eosinophilia-myalgia is a new disease entity for which diagnostic criteria have not been fully elaborated. The Centers for Disease Control has recommended that the diagnosis be based on the fulfillment of all of the following: an eosinophil count of at least 1,000 cells/mm^3, generalized myalgia of sufficient severity to interfere with normal activities at some time during the illness, and absence of any infection or neoplasm that could account for the aforementioned criteria.[2] An earlier criterion no longer considered necessary was the exclusion of trichinosis by serologic testing or muscle biopsy.[1]

L-tryptophan is an essential amino acid that has had a growing popularity for the treatment of insomnia, premenstrual syndrome, and depression.[6] While the association of L-tryptophan-containing products with this syndrome is firmly established, the possible role of contaminants is under current investigation.[2]

Eosinophilia-myalgia syndrome shares some clinical similarities with the toxic-oil syndrome, epidemic in Spain in 1981. This syndrome, which may have been caused by the ingestion of adulterated rapeseed oil, was also associated with marked eosinophilia and myalgia, as well as other clinical and laboratory findings that are not manifest in the current epidemic.[7,8] Other recent case reports bear some similarity to eosino-

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philia-myalgia syndrome, but have been neither epidemic nor associated with L-tryptophan.[9-11]

The differential diagnosis should also include the spectrum of eosinophilic muscle diseases (eosinophilic polymyositis, eosinophilic perimyositis, eosinophilic fasciitis, and focal eosinophilic myositis), as well as polyarteritis nodosa and trichinosis. The finding of xerostomia and conjunctivitis in this patient initially suggested the inclusion of Sjögren's syndrome in the differential diagnosis; however, the absence of a true sicca syndrome and arthritis definitively ruled out this consideration. Recommended treatment is the withdrawal of the product containing L-tryptophan. Some patients are reported to have benefited from corticosteroid therapy. Nonsteroidal anti-inflammatory drugs and narcotic analgesics are indicated for the control of severe muscle pain.[5]

The patient reported here, about whom some preliminary data have been published,[12] ingested approximately 90 g of L-tryptophan over a four-month period, with clinical manifestations appearing at the beginning of the last month. During this time, he continued his normal activities for the most part, although he was at times severely affected as a result of myalgias. The elevation of the serum aldolase and a normal creatinine phosphokinase level reflected similar disparities in other reported patients with eosinophilia-myalgia syndrome.[4,13] Withdrawal of L-tryptophan has not resulted in a rapid amelioration of symptoms. A modest improvement in abnormal laboratory studies was seen.

Patients may not volunteer the fact that they have taken L-tryptophan as they frequently consider all health food products as safe and quite separate from their other medications. With the large number of cases being reported, the pathogenesis and clinical manifestations of this syndrome will no doubt become clearer. In the meanwhile, physicians should be on the alert for patients who have taken L-tryptophan-containing products, and the possible manifestations of eosinophilia-myalgia syndrome.

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Neurocutaneous manifestations of neurofibromatosis-2

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In contrast with the more common form of neurofibromatosis known as von Recklinghausen's neurofibromatosis, or neurofibromatosis-1 (NF-1), bilateral acoustic neurofibromatosis (NF-2) tends to appear late in life.[1] The neurologic manifestations of NF-2 may be devastating, while the dermatologic signs are often discrete.[2,3] Early diagnosis of NF-2 affords the best chances for preservation of function. An increased awareness of the dermatologic signs of NF-2 may help in the detection of patients and their relatives at risk. We present a patient with the clinical manifestations of this disease and review the recommendations for management.

CASE REPORT

A 29-year-old woman was referred for evaluation of her previously diagnosed neurofibromatosis. She had inherited it from her father, who had undergone surgical removal of bilateral acoustic neuromas at another hospital. She had several siblings and no children. No other family members were known to have neurofibromatosis or any signs of it. However, all her relatives lived in distant states, and it was therefore impossible to perform a medical survey of the relatives at risk to detect possible affected members of the family. Letters were sent to those relatives who could be contacted with the advice to seek appropriate evaluation for NF-2. The letters led to the discovery of previously unsuspected bilateral acoustic neuromas in one sister. (Unfortunately, we have been informed that this sister died as a result of asystole caused by the injection of contrast material during angiography performed to investigate the tumors.)

The patient had been in good health until three years prior to evaluation, when she started complaining of low back pain radiating to the posterior aspect of both thighs and calves, accompanied by a sensation of numbness in her feet. As a teenager, she had become aware of a few scattered skin lesions, but they were dismissed as "birthmarks." She subsequently noted a progressively enlarging mass on her left wrist. In the last few years she had noticed a tingling and numbness in the index, middle, and ring fingers of her left hand, more so at night. These symptoms were relieved by shaking and rubbing the hand.

One year prior to evaluation, her low back pain became more severe and she was admitted to a hospital in Texas. Electromyogra-

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phy and myelography led to the diagnosis of a tumor involving the cauda equina. She underwent surgical resection of the tumor, which was a large neurofibroma. After surgery, she remained with residual loss of bowel and bladder control. She must perform self-catheterization to empty her bladder. She also has permanent loss of sensation of her gluteal and perianal areas, as well as distal weakness in both lower extremities necessitating the use of braces to assist ambulation. At the time of hospitalization she was also found to have a mild right facial weakness of the lower motor neuron type, and bilateral hearing loss. A computed tomographic (CT) scan of the head revealed the presence of bilateral acoustic neuromas, and the diagnosis of NF-2 was made. She underwent a resection of the right acoustic neuroma resulting in improvement of the right facial weakness, but sustained complete right-sided deafness.

The physical examination one year after the resection of the acoustic tumor was remarkable for bilateral deafness (complete on the right and partial on the left) and a mild right-sided facial weakness. Additional findings included the following: flaccid paraparesis with severe weakness and atrophy in the muscles supplied by the fourth and fifth lumbar roots and in all muscles supplied by the sacral roots; areflexia in both lower extremities and marked sensory loss to all forms of sensation with a saddle distribution affecting the perianal area and the posterior aspect of both lower extremities. The anal reflex was absent.

The examination of her skin revealed two large café-au-lait lesions on the right thigh and the right shoulder and a small neurofibroma on the right occipital scalp. She exhibited none of the discrete signs of NF-1 such as Lisch nodules, axillary freckling, pectus excavatum, or dysplasias in any other area of her body. An ophthalmologic examination disclosed bilateral posterior subcapsular lenticular opacities. A bulging subcutaneous mass was palpable over her left wrist. Percussion of this mass elicited a Tinel's sign. Holding the left wrist in extreme flexion for a minute or two caused painful paresthesias (Phalen's sign). No atrophy or weakness was noted in the intrinsic hand muscles, and no clear-cut hypoesthesia was found in the distribution of the median nerve. A CT scan of the wrists (Fig 1) demonstrated the presence of a tumor within the left carpal tunnel.

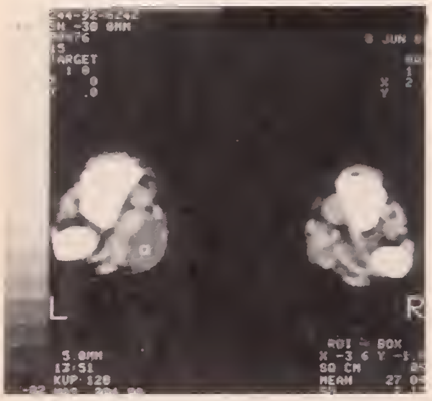

FIGURE 1. CT scan of both wrists showing a tumor in the left carpal tunnel.

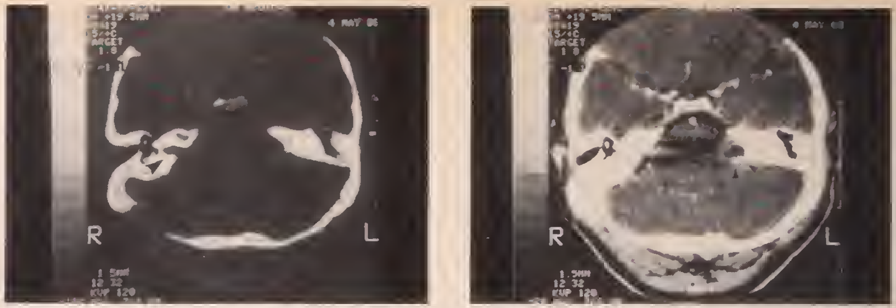

FIGURE 2. **Left,** CT scan of the head (bone window) showing an enlarged right internal auditory canal (large arrow). **Right,** CT scan shows a tumor in the posterior fossa occupying the left cerebellopontine angle (small arrows).

A contrast CT study of the head (Fig 2) showed enlarged internal auditory canals. A small enhancing tumor was present on the left cerebellopontine angle. The previously diagnosed right acoustic neuroma appeared to have been removed completely.

Brain stem auditory evoked responses (BAER) demonstrated absence of all waves after wave I on stimulation of the right ear and absence of all waves after wave II on stimulation of the left ear (Fig 3).

Electromyography of intrinsic hand muscles was normal bilaterally. The median motor distal latency was not prolonged in either side, and there was no reduction of the action potential of the thenar muscles. However, the median sensory distal latency to the left ring finger (the digit suffering the most paresthesias) was prolonged (Fig 4). Nerve conduction velocities of the ulnar and radial nerves were normal, showing that the abnormality was confined to the median nerve and was not part of a generalized polyneuropathy. The diagnosis of left carpal tunnel syndrome with preferential involvement of the sensory fibers from the fourth digit was suggested.

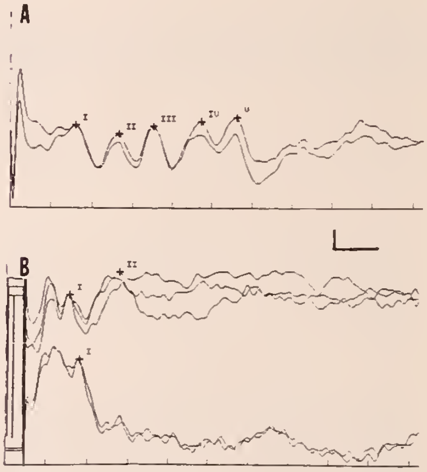

FIGURE 3. **A,** Brain stem auditory evoked responses (BAER) of a normal subject show a sequence of five waves generated by the acoustic nerve and brain stem structures. **B,** BAER of this patient shows the presence of wave I and II to stimulation of right ear (top tracings) and presence of wave I to stimulation of left ear (bottom tracings), with absence of all subsequent waves. Recordings done between vertex and mastoid on the side of stimulation. Positivity at the vertex is plotted upward. Each tracing is an average of 2,000 responses to rarefaction stimuli. Calibration: vertical, 0.15 UV; horizontal, 1 msec.

Since the patient had diminished but useful hearing in the left ear, surgical intervention was not considered appropriate in the management of the left acoustic neuroma. Sequential CT scans are done at regular intervals to monitor a possible increase in the tumor size. The left carpal tunnel syndrome was also managed conservatively with wrist splint, analgesics, and observation of the tumor size and the potential complications.

DISCUSSION

Neurofibromatosis is a heterogeneous disease.[4,5] Riccardi and Eichner have described up to seven different categories in the spectrum of disorders referred to as neurofibromatosis.[2,6] Cutaneous manifestations are a major part of the neurofibromatosis syndrome, and their early recognition is important for the diagnosis and classification of the disease in the different categories.

The better known categories of this disease are referred to as neurofibroma-

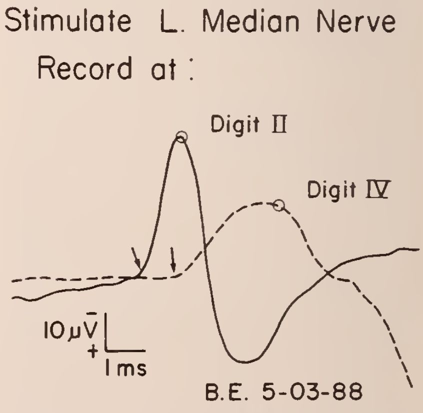

FIGURE 4. Antidromic median sensory nerve stimulation at 14 cm with recordings over digits II and IV. Note the delayed onset of the sensory nerve action potential in digit IV as compared with digit II.

tosis-1 (NF-1) and neurofibromatosis-2 (NF-2), corresponding to the old terms von Recklinghausen neurofibromatosis, and bilateral acoustic neurofibromatosis or central neurofibromatosis, respectively.[1] It is important to distinguish NF-2 from NF-1, as the clinical expression, genetic basis, and natural history of these two diseases are very different. NF-2, a genetic defect associated with multiple tumors of neural crest origin, features the presence of bilateral acoustic neuromas as its hallmark.[7] Both diseases have an autosomal dominant form of inheritance, but the genes responsible for NF-1 and NF-2 are located in different chromosomes. Genetic linkage studies have located the NF-1 gene on chromosome 17.[8] Specific loss of alleles from chromosome 22 has been detected with polymorphic DNA markers in cases of acoustic neuromas and other tumors associated with NF-2.[9,10]

NF-1, a disorder affecting one in 4,000 individuals, is more frequently seen than NF-2, which only occurs in approximately one in 50,000 individuals. The mutation rate is high in NF-1 and is unknown in NF-2. Some studies have suggested that in NF-2, a growth factor may be implicated in tumor formation.[7] Antigenic but not functional activity of nerve growth factor is increased, and high levels of a glial growth-factor-like activity have been found in NF-2 tumors.[11]

The National Institutes of Health Consensus Development Conference[12] concluded that the criteria for NF-2 are met by an individual who has bilateral eighth nerve masses seen with appropriate imaging techniques (eg, CT or MRI), or a first degree relative with NF-2 and either a unilateral eighth nerve mass or two of the following: a neurofibroma, meningioma, glioma, Schwannoma, or a juvenile posterior subcapsular lenticular opacity.

Cutaneous lesions, although less common in NF-2 than in NF-1, are also important markers of the disease.[1] The significance of café-au-lait spots in NF-2 is not established, but their presence in someone at risk should heighten suspicion. In the patient described here, a thorough examination of the skin disclosed a café-au-lait spot, 4 × 2 cm in size, on the left shoulder, and another 8 × 5-cm lesion on the right thigh. Since the patient's skin was darkly pigmented, these lesions were barely visible. The rule of Crowe et al,[13] that six or more café-au-lait spots larger than 1.5 cm in diameter must be present for establishing the diagnosis of neurofibromatosis, does not apply in the NF-2 form of the disease. The freckling that frequently involves the axilla as well as other intertriginous regions in NF-1 is distinctly uncommon in NF-2.[1] Neurofibromas of the skin tend to be much less frequent than in NF-1, but when present are virtually diagnostic. They tend to be small and only minimally raised, and therefore can be easily overlooked. Neurofibromas may compromise the spinal cord or the cauda equina, as seen in this patient, or compress the spinal nerve roots. The neurofibroma in the flexor aspect of this patient's left wrist illustrates the possibility of NF-2 having both central and peripheral nervous system manifestations, and consequently, the previously used term "central neurofibromatosis" should be abandoned.

In addition to acoustic neuromas, other tumors of the central nervous system, sometimes multiple, are commonly seen in NF-2.[14,15] Meningiomas in the intracranial cavity or in the spinal canal are frequent. Schwannomas similar to the acoustic neuromas may develop in other cranial nerves or spinal roots. Gliomas, especially low grade astrocytomas, are also common in this disease. Other associated features in NF-1, such as skeletal abnormalities, intellectual handicaps, and epilepsy, are not encountered in NF-2.

In addition to a careful personal and family history, the physical examination should give particular attention to skin manifestations such as café-au-lait macules and neurofibromas. The accessibility of skin to firsthand visual and anatomo-pathologic analysis emphasizes the role of the dermatologist in the detection of NF-2. Although there are no normal control data, thorough skin examination with Wood's light is advised. The neurologic examination should include a detailed evaluation of hearing, balance, and coordination, as well as the function of the cranial nerves.

The National Institute of Health Consensus Development Conference concluded that NF-2 testing must include an audiogram and brain stem auditory evoked responses (BAER).[12] Magnetic resonance imaging (MRI) was recommended for patients with evidence of hearing impairments or abnormal BAER. The entire neuroaxis can be screened with magnetic resonance with no danger of radiation.

If the differential diagnosis between NF-1 and NF-2 is not clearly established, a thorough ophthalmologic examination, with emphasis on the lens and iris, may be helpful. Lisch nodules (pigmented hamartomas of the iris) are present in up to 94% of patients with NF-1, but are rare in NF-2.[16] On the other hand, the presence of juvenile posterior subcapsular and nuclear cataracts has been recently recognized in NF-2, affecting about half of the patients.[17] This finding has important implications for patients and for the detection of relatives at risk. Slit lamp biomicroscopic examination has been urged as part of the work-up of these patients and their families.

Genetic counseling should be offered to patients and families.[1] In view of the autosomal dominant form of inheritance with low mutation rate, all family members, particularly first degree relatives, should be evaluated periodically to determine whether they have NF-2.

Other complementary investigations should be used as appropriate. In the patient presented here, paresthesias in the left hand prompted an electromyographic study, which demonstrated involvement of the median nerve at the carpal tunnel. The compression of this nerve appeared to affect the sensory fibers of a single digit. Involvement of the fibers of a single digit in cases of carpal tunnel syndrome has been reported in the medical literature.[18] Most cases of carpal tunnel syndrome are caused by an idiopathic hypertrophy of the carpal ligament.[19] However, there are multiple cases described in the literature of median nerve compression in the carpal tunnel by other space occupying processes, both benign and malignant, which may compress the tunnel's contents. Carpal tunnel syndrome may be caused by bony callus after a fracture,[20] by tophi,[21] by ganglia,[22] and by many other expansive processes. To our knowledge, the compression of the median nerve at the carpal tunnel caused by a neurofibroma in a case of NF-2 has not previously been reported in the literature.

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THE DOCTOR'S RESPONSIBILITY TO HIS COMMUNITY

. . . Medicine has always been and is now motivated by a desire to be of service to the public. To achieve this ideal, the profession must be free. In these times in which we live, we are presented with advances in the service of medicine to mankind which are conditioned by coincident, far-reaching economic and social changes. It will require wise and forward-looking men to guide these changes by evolution rather than revolution. Socialized medicine, compulsory health insurance, or whatever name you wish to use to designate the efforts on the part of government to direct and control the practice of medicine, is not a dead issue in this country. There is no political appeal quite so intriguing as "something for nothing. . . ."

The doctor, each in his own community, has a prestige which is tremendous. You should be aware of this asset and assert this influence which is yours on your patients, lay leaders, and above all others, your legislators. . . .

Again, by teamwork at the local level, I believe much will be accomplished by the medical profession in maintaining its independence from political control. As physicians, you are already members of a medical team which has carried us to where we are—the healthiest nation in the world. You are conscious of the value of teamwork; it is part of your life as a practicing physician whether you are in private practice, in the hospital, or taking part in the activities of your county medical society. I urge all of you, your wives and friends, to extend this type of teamwork in your own communities. Make yourselves important cogs in the local philanthropic activities in your area which deal with cancer, tuberculosis, heart disease, cerebral palsy, mental hygiene, and many others. You must get away from the strict confines of your offices and the limits of the sickroom.

Become increasingly more civic-minded in these days of corruption and moral deterioration in high places. We must all do our full share to restore civic virtue.

Therefore let us participate in programs for sound government as we understand it; let us uphold the freedom of the American people, aid our universities and medical colleges, promote culture and tolerance and understanding in our home towns. We have the education, the training, and the ideals to contribute to these programs which help to make our own community a better place in which to live for ourselves, our families, and our children.

By this kind of community team effort we know more intimately the problems, hopes, and desires of our neighbors. In turn these neighbors will appreciate their doctors and realize that our desires and struggles for the freedom of medicine are closely tied in not only with their health but with the happiness and liberty of their own lives.

J. STANLEY KENNEY, MD
(*NY State J Med* 1952; 52:1922–1923)

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Defining brain death

TO THE EDITOR: Dr Irving Bunkin's special article, "He had a change of heart," is interesting and moving.[1] Dr Bunkin has written a thought-provoking piece about the complex interface between technology and humanism in modern medicine.

I was dismayed, however, at one comment in the article, which I think betrays a dubious perception of when death occurs. Quoting himself in a discussion with one of the patient's surgeons, Dr Bunkin asks, "How does it feel to open the chest of a man who is still alive, but brain-dead . . . ?"

What's going on here? It is my understanding that a person who is brain-dead is *dead.* To suggest that a brain-dead organ donor "is still alive" not only misconstrues the meaning of brain death, but also places an almost insurmountable obstacle in the path of physicians, ethicists, attorneys, and others who are struggling to educate the public on exactly this point.

Can we not settle it once and for all? To be brain-dead is to be dead, notwithstanding the fact that the heart and other organs may be viable for purposes of transplantation.

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[1] Bunkin IA: He had a change of heart. *NY State J Med* 1990; 90:253-255.

In reply. It is not surprising that Dr Rogatz was "dismayed" by my remark quoted above. Agreed, in 1990, "brain dead" means dead. However, I shall not get into too long a physiological-philosophical-semantic argument with Dr Rogatz. When a family offers the body of a "brain-dead person" for "harvesting" of organs, the operating room becomes a macabre scene at 2:00 AM. A variety of "harvesters" (strangers to one another) arrive. Eyes are removed to supply corneas somewhere; the kidneys are extirpated for those awaiting renal transplant; then the liver and the pancreas, or maybe the lungs, are removed. The heart harvesters, for obvious reasons, are always last. There remains a beating (live?) heart. Remove it from connections in the donor's chest and it stops beating. During transport in hyperosmolar saline, it is quiescent. Once connected to the large vessels in the recipient's chest, it begins to beat spontaneously 50% of the time when blood flows through it again. Often it has to be electrically shocked into regular contractility. The transplanted heart now produces an electrocardiogram which exhibits two P waves.

In my essay, the intent was to convey the fact that chemical and electrophysical integrity *persisted* (at least for several hours) in the heart.

In Plato's *Phaedo,* Phaedo says that the soul is "deathless." Saint Augustine might have added that the beating heart in a "brain-dead" person is evidence of a living soul. Maybe it is the living soul that the organ harvesters carry with them in the bag of ice. Restated, the heart has the potential of "life after life."

To physicians, ethicists, attorneys, and others a "brain-dead" person is dead. But, as a romantic, I was merely trying to dramatize the fact that the sequence of events during a heart transplant looked like a resurrection, circa 1990, a miracle of modern medicine. Nevertheless, Dr Rogatz does make a clear and cogent point.

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Trade and generic names of drugs

TO THE EDITOR: I read with interest the case report on fetal heart rate changes during general anesthesia.[1] However, I take exception to the interchange between trade and generic names of the drugs used. Versed is the trade name for midazolam, fentanyl citrate is the generic name for Sublimaze, and Surital is the trade name for thiamylal sodium.

Residents taking their specialty boards must be familiar with the generic name of every drug. Would it not be more rational to consistently use the generic name with the trade name following in parentheses?

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[1] Faustin D, Gutierrez L, Chaudhry MR, et al: Fetal heart rate changes during general anesthesia. *NY State J Med* 1990; 90:272.

In reply. I want to thank Dr Marx for bringing the above matter to our attention. It is the *Journal's* policy to use generic names and this is outlined in our Guidelines to Authors.

The inconsistencies pointed out by Dr Marx occurred through oversight in the copyediting process. We will try to do better in the future.

PASCAL JAMES IMPERATO, MD
Editor

A neglected resource

TO THE EDITOR: The Medical Society of the State of New York is experiencing a curious and puzzling phenomenon. It is the relatively small number of spouses who are members of our auxiliary. In the entire state of New York only 2,780 spouses are auxilians. If even 20% of the spouses of our 26,000 physician members were auxilians, this would increase the number of auxilians to 5,200.

This deficit becomes even more striking when one realizes that it comes at a time when we need all the support we can get if we are to succeed. Historically, spouses have been staunch defenders of their families. Today, as never before, we need their help and we simply cannot afford to ignore this superb resource. We must encourage our spouses to join the auxiliary.

We are fortunate that we have had the faithful few to stand by us, but they need more members if they are to carry out the vital programs that they are planning. If we believe that the practice of medicine is worth protecting from the doctor bashers, the efforts of our spouses are sorely needed. They can bring home critical information from their meetings with legislators and others. They are effective lobbyists and collectively they can mobilize communities on our behalf.

Such activities need not be at a sacrifice to the existing interests of our spouses. By joining the auxiliary and giving only a few hours of their time the reward would be very worthwhile. Our spouses would have a clearer and more sensitive understanding of what we are going through. They would also acquire skills that could be applied elsewhere.

It is exceedingly clear that we cannot fight the battles of health care alone. We must remember, as we seek to find allies, our very talented spouses. This neglected resource is such an important part of the fabric of our lives and our hopes.

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Reversible hypoxic cerebral edema in severe hemolytic crisis

TO THE EDITOR: The causes of cerebral edema are varied and multifactorial; they can include trauma, infection, tumors, surgical manipulation, metabolic disturbances, and cerebral ischemia.[1-3] Severe hemolytic anemia can produce cerebral ischemia and mental changes but rarely, if ever, leads to demonstrable brain edema. Recently, we encountered a patient in hemolytic crisis who presented with seizures and computed tomographic (CT) evidence of diffuse brain swelling.[5,6] The clinical and CT findings resolved completely with appropriate therapy.

Case Report. A 33-year-old woman with a history of sickle thalassemia and multiple blood transfusions was seen in the emergency room complaining of lethargy and exhibiting bizarre behavior. She had been discharged from the hospital just three days prior to this admission after having undergone a termination of pregnancy. During that hospital stay, the patient had received two units of packed red blood cells to achieve a hematocrit of 16%.

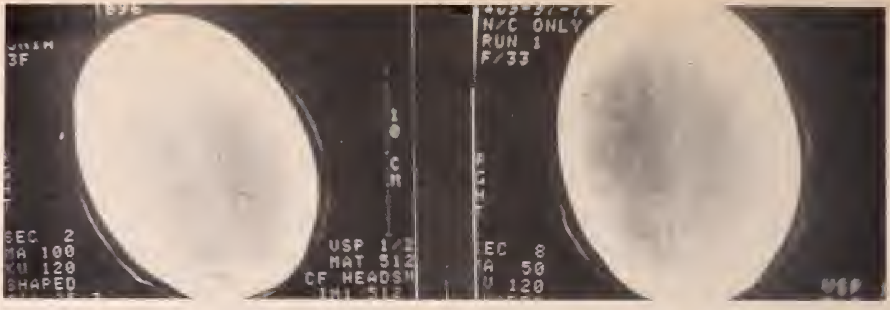

FIGURE 1. Left, computed tomographic scan showing cerebral edema with absence of folds of sulci. Right, computed tomographic scan after treatment of cerebral edema showing normal pattern of sulci and gyri and resolution of edema.

Physical examination revealed an afebrile woman who was lethargic, disoriented, and confused. There was evidence of bilateral papilledema, bilateral abducens palsies, and a left hemiparesis. She was also noted to have moderate vaginal bleeding. While in the emergency room, the patient had a grand mal seizure as witnessed by a physician on duty.

Pertinent laboratory results revealed a hemoglobin of 2.5 g/100 mL; hematocrit, 8.5%; white blood cell count, 30,000/mm^3; reticulocyte count, 18%; haptoglobin, >1; platelet count, 170,000/mm^3; Po_2, 56 mm Hg; and normal serum electrolyte levels. All cultures were reported as negative. The patient underwent a CT scan of the head which demonstrated diffuse cerebral edema (Fig 1). Coombs' direct (anti-IgG and C3) and indirect tests were strongly positive, and blood could not be crossmatched because of serum antibodies anti-C, anti-Fya, anti-Jka, and anti-N. Intubation was performed, and the patient received a transfusion of three units of packed red blood cells of the most compatible blood available. She received 12 mg of dexamethasone by intravenous push and subsequent bolus injections of 8 mg every six hours, as well as folic acid, 5 mg by intravenous push, and mannitol, 20% solution in 100 mL (20 g) by intravenous piggyback. After receiving the first unit of packed cells, the patient had a dramatic response, with improved mentation and a complete reversal of her neurologic deficits.

Extubation was done in less than 48 hours, and a repeat CT scan of the head (Fig 1) showed resolution of the cerebral edema. The patient was discharged on a regimen of folic acid, 1 mg twice a day, and prednisone, 10 mg three times a day. On follow-up in our office, her hematocrit was 30%.

Discussion. To our knowledge this is the first reported case of reversible cerebral edema secondary to hemolytic crisis causing severe brain hypoxia and ischemia. In patients with acute mountain sickness,[4] hypoxia associated with high altitude can produce a reversible type of brain edema that responds to diuretic and oxygen therapy and a return to lower altitudes.

In the patient described here, the cause of hypoxia was hemolytic anemia, resulting from sickle cell thalassemia, isoimmune antibodies, and autoimmune antibodies. We documented the rapid reversibility of cerebral edema as seen by CT scanning (Fig 1) within 48 hours following transfusion to correct the hypoxia, as well as administration of mannitol and dexamethasone.

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—ERRATUM—

The article by Yankauskas in the June 1990 issue, "Primary female syndromes: An update" (90:295–302), contained an error in Table IV. Verapamil should have been categorized as a calcium channel blocker, not as an angiotensin-converting enzyme inhibitor.

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The community health worker program

The New York State Prenatal Care Act of 1987, which mandated public education and outreach and referral services for low income pregnant women, led to the establishment of the Community Health Worker Program, a community-focused outreach program that directs women to early and continuous prenatal care services.

Pregnant women do not obtain prenatal care and related services because of personal, institutional and financial barriers to those services.[1–3] Through Community Health Worker Program contracts, the Department of Health hires and trains indigenous residents of high risk areas to be community health workers (CHW).

The CHW works with pregnant women and their families to assist the women to overcome the barriers to health care and support services. The CHW's background is similar in language, culture, and socioeconomic levels to the majority of the affected families so the worker is better able to understand and help bridge the gap between services and people in the community. The CHW is in a unique position to understand the problems confronting the population because of her or his own life experiences. She or he has overcome the obstacles or barriers to utilization of community services and now can assist others do the same.

The CHW develops a highly individualized approach to each family, and maintains a relationship with the family through repeated home visits. In this way, the worker is better able to influence the family members to take the necessary steps to improve and/or maintain the health status of the family. The role of the CHW with the family includes: case finding, basic preventive health education, health risk assessment, translation and cultural interpretation with service providers, referrals to needed health, social and community services, follow-up and continuing support of individuals and families with a focus on prenatal and parenting family. The CHW provides information, support, advocacy, encouragement, and assistance to families to identify, prioritize, and resolve problems that adversely affect the family's health status, even though the problems might not be defined as health related.

To assist the CHWs with the family, an extensive educational program was developed and includes: communication and interviewing skills, community assessment, community resource and referral network development, the effect of culture on health behavior, and basic public health education and information. The content focuses on: prenatal care, parenting, child growth and development, nutrition, family planning, AIDS prevention, and loss and bereavement. The CHW is also trained to assess and describe family high risk situations such as child abuse, domestic violence, substance abuse, environmental, and housing problems. As part of the community resource assessment, the CHW visits providers to observe and gain first hand knowledge of the services available and the background information necessary to complete applications to obtain agency services, and to observe procedures that the client will face. With this information, CHWs can help their clients prepare for and, therefore, maximize the first appointment to an agency.

The CHW program has been developed in 14 locations across New York State in both urban and rural settings. The department developed contracts with community-based organizations and local health units to establish the programs. To strengthen the CHWs' resources knowledge, staff of local agencies trained the CHWs whenever possible.

The target areas for the Community Health Worker Programs were identified using the following criteria:

- Share of births weighing under 2,500 grams was greater than 1986 statewide average of 7.3% and/or upstate (New York State exclusive of New York City) average of 5.9%.
- Infant mortality rate was greater than 1986 statewide rate of 10.6 per 1,000 live births and/or upstate rate of 9.2 per 1,000 live births.
- Share of births with early prenatal care was less than 1986 statewide average of 67.8% and/or upstate average of 82.7%.
- Share of births with late or no prenatal care was greater than 1986 statewide average of 11.2% and/or upstate average of 3.3%.
- A high percentage of PCAP eligible patients unserved as of July 1987.
- The ability of existing PCAP providers to take new patients in an acceptable time frame.

The department piloted the program in both urban, suburban, and rural areas to determine the feasibility and appropriateness of this program design and implementation strategy in a variety of settings and using a variety of contractors (both local health units and community-based organizations). Based on this analysis CHW programs were implemented in Albany, Buffalo, Rochester, and Syracuse, as well as in Oneida and Herkimer counties and St Lawrence, Jefferson, and Lewis counties. Metropolitan New York programs are located in Bronx, Brooklyn, Far Rockaway, Hempstead, Wyandanch and Yonkers. CHWs also work with families living in homeless shelters throughout New York City.

Some CHW clients are referred from Department of Social Service programs, health care providers, neighbors and other family members. CHWs also canvas neighborhood residents, after informing community members and leaders of the program. Major problem areas and needs have been identified, including financial assistance, clothing, bedding and supplies, health care, transportation, nutrition and food related needs, day care, vocation/education, housing and safety problems, and immigration concerns.

Much of the first year of the statewide Community Health Worker Program was spent in program development, curriculum development and implementation, selection of training consultants for certain curriculum content areas, and development and refinement of data gathering instruments for use in program evaluation. At the local levels, the programs selected suitable office sites, developed program implementation strategies, recruited, hired and trained the CHWs, selected resource materials, implemented ongoing inservice training, assessed the community and its resources, and developed and promoted program referral linkages.

Evaluation of the program's effectiveness examines both process and outcome measures—the number and types of referrals that the CHWs made and that are completed, the number of women who actually entered care and/or received services as a result of recruitment and referral, the pregnancy outcomes for prenatal clients relative to birth weight, gestational age and health status of the infant and other problems that are resolved as a result of CHW intervention.

CHWs are prepared to work with pregnant and parenting families, as well as those families with a specific problem such as substance abuse. By changing the focus of some components of the educational program, CHWs can work with other populations in need of their services. The department is developing Community Health Worker Programs to work with schoolchildren and their families, migrant and seasonal farmworkers and other population groups. The department continues to explore ways of working together with people experiencing difficulty in using the complex health and social service systems.

Additional information regarding the Community Health Worker Program is available from Patricia Casaw, Center for Community Health, at (518) 473-4223.

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Skin cancer prevention

People need to be aware of the danger of overexposure to the sun. Skin cancer is the most common form of cancer, with more than 500,000 new cases of skin cancer reported annually in the United States. Fortunately it is one of the most curable. Better still, it can be prevented.

Malignant melanoma, one form of skin cancer, spreads quickly and rapidly invades other organs of the body. Between 1950 and 1986, the incidence rate for this form of skin cancer increased fivefold in men and more than tripled in women. More than 1,300 new cases and 435 deaths from malignant melanoma occur each year in New York State. Early diagnosis and treatment can prevent most malignant melanoma deaths.

Other major forms of skin cancer are basal cell and squamous cell carcinomas. These are less aggressive than melanomas but occur much more frequently. Non-melanoma skin cancers are not required to be reported to the New York State Cancer Registry, so an accurate count is not available. It is estimated, however, that as many as 20–30 thousand non-melanoma skin cancers occur in New York State each year.

Skin cancer more frequently occurs among people who have worked or spent a great deal of recreational time in the sun—sunbathing, gardening, sailing—especially if they have been severely burned in their early years of life. Ninety percent of skin cancers occur on parts of the body that are not usually covered by clothing, such as the face, tips of the ears, hands and forearms. In the last 50 years, however, the shoulders, backs and chests of men, and lower legs of women have become common areas for skin cancer because more people are sunbathing, deliberately exposing themselves to ultraviolet radiation.

People who should be especially careful are those with fair skin that burns and freckles easily, and those with light eyes and red or blond hair. People who have many moles or have family members with malignant melanoma are more likely to develop skin cancer.

There are several important warning signs for early recognition of malignant melanoma and other forms of skin cancer.

- Be alert to changes in the number, size, shape, or color of moles or other pigmented areas or skin lesions.
- Look for changes in the surface of a mole such as scaliness, oozing, bleeding or the appearance of a bump, redness or a new swelling beyond the mole's edge.
- Note changes in sensation like itchiness, tenderness or pain.

The American Academy of Dermatology promotes a simple "ABCD" rule to help in remembering important characteristics of moles that suggest malignant melanoma:

A. Asymmetry—one half does not match the other.
B. Border Irregularity—the edges are ragged, notched or blurred.
C. Color—the pigmentation is not uniform. Shades of brown, tan and black are present. Red, white or blue may add to a mottled appearance.
D. Diameter—if it is larger than six millimeters in diameter (the size of a pencil eraser). Any sudden or continuous increase in size should be of special concern.

Even more desirable than early detection is prevention through avoidance of overexposure to ultraviolet radiation, either from the sun or artificial sources. Repeated sunburns are particularly risky. People can follow several simple protective measures: Cover up with a widebrimmed hat and wear long sleeved shirts and pants whenever possible.

Use sunscreen with a sun protection factor (SPF) of at least 15 to reduce sunburn, risk of skin cancer and premature aging of the skin. Apply sunscreen at least one hour before going into the sun and again after swimming or perspiring.

Avoid the direct sun between 11 AM and 2 PM when the sun's rays are strongest. Sunburn can occur even on cloudy days.

A skin cancer fact sheet is available from the Bureau of Cancer Epidemiology, NYSDOH, Rm 565, Corning Tower, Albany, NY 12237-0683.

Cervical cancer in New York State

Cervical cancer remains an important problem in New York State, despite success in controlling cervical cancer in comparison to other forms of cancer. Approximately 1,100 invasive cervical cancer cases and 325 deaths from cervical cancer are reported among New York State women each year. Cervical cancers account for over 5,200 hospital admissions, 44,000 hospital days and 3,000 years of potential life lost each year. Cervical cancer incidence rates in New York State are generally comparable to those reported by the National Cancer Institute for the country as a whole. Mortality rates have been consistently lower in New York State when compared with national averages.

The major risk factors for cervical cancer are listed in Figure 1. Cervical cancer risk is related to a number of sociodemographic characteristics, including place of residence, race and socioeconomic status. Risk tends to be higher in the nonwhite and lower socioeconomic segments of the population. Residents in highly urbanized and extremely rural areas have higher incidence and mortality rates than suburban women. The relationship of cervical cancer risk with sexual activity is a long-standing observation. In recent years, an understanding has begun to emerge of how sexually transmitted infections, possibly in the presence of cofactors (eg smoking, oral contraceptives), might influence the development of cervical cancer.

The incidence rate for invasive cervical cancer has declined by 67% from 1950 to 1986. This decline in invasive incidence has occurred, over the same time period, in conjunction with an increase in in-situ cervical cancers. The in-situ lesion is the earliest and least serious form of cervical cancer, and is potentially 100% curable. In-situ cervical cancers are most often detected by the Pap smear and represent cancer screening successes. The increase in the in-situ cervical cancer rate and the decline in the rate of invasive cervical cancer rate are reflected in a substantial decrease in the cervical cancer mortality rate, which has declined 79% between 1950 and 1986 (Fig 2).

Modern cervical cytology has become an established part of medical practice in the United States. Despite the lack of clinical trials linking the Pap smear with decreasing cervical cancer mortality, a great deal of supporting evidence has been derived from population-based epidemiologic studies. An association of the widespread use of Pap screening and reductions in cervical cancer risk is supported by differences in cervical cancer mortality rates among screened and unscreened women from the same population, correlations between cervical cancer mortality rates and screening rates in different populations, and time trend analyses showing declines in cervical cancer mortality following the introduction of Pap screening.

Physicians are strongly encouraged to provide Pap smears and pelvic examinations to appropriate hospital inpatients and to more effectively use other patient encounters (eg, private office visits, non-emergency and outpatient care) as opportunities for reaching previously unscreened women.

One important aspect of cervical cytology is the clear communication of the findings from the pathologist to the referring physician. This has been hindered by currently used terminology, which is often ambiguous and confusing. The National Cancer Institute convened a workshop of distinguished cytopathologists and representatives

FACTOR	HIGH RISK	LOW RISK	MAGNITUDE OF RISK
Place of Residence	Urban and Rural	Suburban	1-2 fold increased risk
Race	Black and Hispanic	White	
Socioeconomic Status	Lower	Upper	
Age at First Coitus	<17	17+	2-3 fold increased risk
Number of Sexual Partners	Multiple	Single	
Cigarette Smoking	Yes	No	
Herpes Virus Type 2 (HSV2)	Previous Infection	No Previous Infection	3-4 fold increased risk
Human Papillomavirus (HPV)	Previous Infection	No Previous Infection	3-9 fold increased risk

FIGURE 1. Risk factors for cervical cancer.

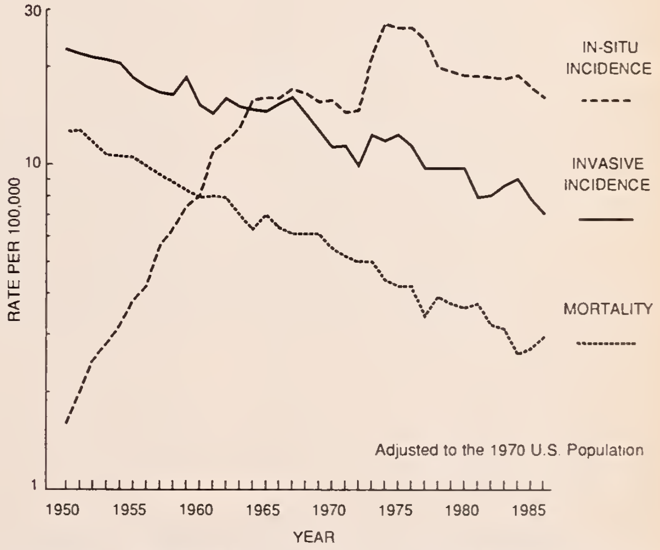

FIGURE 2. Age-adjusted cervical cancer incidence and mortality rates in New York State (exclusive of New York City), 1950–1986.

from national medical organizations to propose a new set of guidelines called the Bethesda System.[1] They hope adoption of this standard system of terminology will improve the clarity and the utility of cervical cytopathology.

Promotion of cervical cancer screening for currently underserved and high risk populations remains the best hope for reducing mortality from cervical cancer in New York State. The Bureau of Cancer Epidemiology is developing cancer data profiles for New York State counties to assist local health officials in cancer control planning. Each profile will contain a section specific to cervical cancer screening. For more information, contact Dr Mark Baptiste, Bureau of Cancer Epidemiology, (518) 474-2354.

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Note: The information in this article was condensed from the New York State Department of Health report Cancer in New York State. *For a copy of this report, contact the Bureau of Cancer Epidemiology, NYS Department of Health, Rm 565, Corning Tower, Albany, NY 12237-0683.*

FROM THE LIBRARY

THE TREATMENT OF CARCINOMA OF THE CERVIX

. . . The present therapy for cervical carcinoma is only a phase of constant development and progress, requiring time for evaluation.

The Wertheim type of operation was favored during the early part of the century. This procedure removed the uterus, adnexa, parametria, and upper portion of the vagina. After World War I, radiation with x-ray and radium proved more effective and was generally accepted. During World War II, slow progress in radiation technics and marked progress in surgical technics resulted in renewed interest in surgical treatment. More extensive surgery than the Wertheim operation seemed possible.

The most significant operation developed is best called a radical panhysterectomy with bilateral lymphadenectomy, called radical hysterectomy for brevity. This procedure removes the uterus, adnexa, parametria, upper half of the vagina, and the lymphatic fascia of the lateral pelvis. In early selected cases operated by specially trained teams, it provides results comparable to radiation. It has not been proved superior to radiation in unselected series, and it is not technically feasible in more advanced cases.

Total pelvic exenteration has been developed during recent years. This procedure, which requires colostomy and urinary diversion, removes all of the pelvic viscera and fascias. It has proved reasonably safe in specialized centers but is best considered experimental since results do not justify its universal adoption.

The "nucleonic" age has brought unexpected progress in radiation therapy. Advances are slow in application, however, because they involve basic physical knowledge and expensive equipment beyond the grasp of the average operator. The medical profession must keep pace if carcinoma is to be treated with the best possible technics. Knowledge of variations in radiosusceptibility is growing. Future methods for predicting radiosensitivity may prove successful in guiding us in electing surgery, radiation, or both in individual cases.

. . . Satisfactory treatment for advanced cervical carcinoma has not been found. The best curative and palliative treatment is still radiation with x-ray and radium. Radical hysterectomy is technically too difficult, and the best of surgeons have not attempted this procedure in these stages. Exenteration is appealing on the theory that it could excise all of the carcinoma. Practice has not proved this to be true, and the pain and annoyance caused by the surgery is often added to that of the persistent carcinoma.

Brunschwig reported extensive surgery on 100 advanced cases of genital carcinoma. Twenty percent died within thirty days, another 49 percent died within eight months, and 10 percent are listed as "well" one year and six months after surgery. Some patients may deserve referral to centers capable of such surgery, but it is not yet the treatment for most cases. . . .

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ALFRED S. BERNE, MD, CHESTER E. CLARK, MD
(*NY State J Med* 1955; 55:3480-3484)

CLINICAL IMPLICATIONS OF ABNORMAL DIGESTIVE TRACT RADIOGRAPHS

By David A. Morowitz, MD. 235 pp, illustrated. Philadelphia, Lea & Febiger, 1990. $39.50 (hardcover)

This book consists of a series of single page descriptions of a panoply of gastrointestinal (GI) disorders with accompanying radiographs. Its purpose is to convey in succinct form a summary statement about these entities while describing their salient radiologic features. It is written from the point of view of a clinical gastroenterologist. The intended audience for this book is not clear to this reviewer, also a gastroenterologist. The book is not sophisticated or complex enough for the specialist, yet it describes a number of entities with which the nonspecialist will probably never be concerned. While it is written with enthusiasm, its place in the medical literature is unclear.

In the text, diseases are followed down the gastrointestinal tract. Included are a clinical description, some comments on differential diagnosis, methods of evaluation, x-ray features, and treatment. This is a tall order for one page and leads to some oversimplified comments. While it is admirable to emphasize that too much endoscopy is being performed and probably not enough radiology, there is a general bias against endoscopy that is excessive. The upper GI series is called the gold standard for the diagnosis of duodenal ulcer, despite all the H-2 blockade studies showing the superior accuracy of endoscopy. In the section on colonic polyps, it is stated that most polypoid cancers were never benign polyps and that "colonoscopy seems an unavoidable adjunct to the barium examination." While the author is forthright in stating his personal opinion, such statements are quite debatable. Similarly, there is not enough emphasis on false negative barium enemas and the importance of flexible sigmoidoscopy in the diagnosis of rectal carcinoma.

Since half of the pages of the text are devoted to x-rays, a comment on their quality is relevant. A number of the x-rays depict conditions that are rarely demonstrated, such as Meckel's diverticulum, gallstone ileus, esophageal leiomyoma, and Hirschsprung's disease. The reproductions, however, are somewhat uneven, with a tendency to be too light, obscuring some of the findings described in the corresponding text.

The array of clinical entities and x-ray photographs is impressive; clearly this book is written by someone with extensive clinical experience. Certainly in this era of over-utilized endoscopy, ultrasonography, CT scans, and other expensive imaging techniques, it is timely to emphasize the importance of the relatively inexpensive, safe radiological examination. Unfortunately, this book may have a problem finding its proper audience because of its broad scope and narrow discourse.

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A DAY HOSPITAL GROUP TREATMENT PROGRAM FOR ANOREXIA NERVOSA AND BULIMIA NERVOSA

Edited by Niva Piran, PhD, and Allen S. Laplan, MD. 168 pp, illustrated. New York, Brunner/Mazel Publishers, 1990. $25.00 (hardcover)

This is a brief specialized text describing a day treatment model for patients with moderate to severe anorexia nervosa and bulimia nervosa. Its audience is therefore limited to individuals who are interested in setting up a similar program, or who wish to incorporate this strategy in less intensive outpatient treatment or more intensive inpatient treatment. The limitations of the book in no way reflect upon what would appear to be an innovative, useful treatment program.

The authors are from Toronto General Hospital, which has a history of excellence in the treatment of eating disorders. The program which they describe in the 168 pages of this text is for patients who are refractory to the usual forms of outpatient treatment. The program treats ten to 12 patients at a time, five days a week, eight hours a day, with an average duration of treatment of two to four months. It uses a predominantly group model, whereby each patient is seen in five to six groups a day, and is staffed by a multidisciplinary team including a psychologist, a psychiatrist, an occupational therapist, a social worker, two nurses and a nutritionist. Although actively suicidal and severely medically ill patients are excluded, most of the people accepted have been ill for a long period of time and have severe symptomatology.

Two meals per day plus snacks are provided at the site. A cognitive behavioral approach is stressed, with all meals being carefully supervised and with meal plans, cooking supervision, psycho-education around nutrition, and time management being active parts of the program.

The role of the psychiatrist, nutritionist, family therapist, occupational therapist and nurse are all described in separate chapters. The rationale behind the day treatment program is that it costs less, can serve more patients, and fosters less regression than an inpatient program. However, no actual cost data were included in the text.

I have a few minor questions about the way the program is set up. For example, it specifically precludes any ongoing psychotherapy outside of the day treatment program. It was not clear to me why this is the case and there is also very little discussion about follow-up treatment once the person completes the two-to-four month day program. The psychiatrist also acts as the medical physician, and does an initial physical examination which seems to include a breast exam and rectal exam. For a variety of reasons, most psychiatrists would be more comfortable with a consulting internist doing the initial physical examination so that the psychiatrist can continue as a group leader and pharmacologist without needing to deal with issues that may arise from this rather invasive initial examination.

Prior to being admitted to the program, all patients agree to eat all their meals. Therefore, the program deals with a somewhat skewed sample who have indicated a significant motivation for change. Nonetheless, these quibbles are somewhat minor and do not take away from the overall quality and creativity of the program.

In the final chapter, the authors evaluate the first 53 patients who were admitted over a 20-month period. Nine of them (17%) dropped out within the first two weeks. Of the anorectics, 73% gained at least ten pounds (N=19) by the end of the program. Of the bulimics, 54% ceased binging and purging by the end of the program, 88% had at least a 75% reduction in binging, and 100% had at least a 50% reduction in binging (N=35). These gains seems to be holding on three to six months' follow-up.

Although this program seems worthwhile, the general psychiatrist would probably not want to invest the $25 for this specialized volume. It is of most interest to eating disorder specialists who wish to set up a day treatment program. Eating disorders are often refractory to traditional treatment and this innovative approach is a welcome addition to the treatment options available to us.

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PERSPECTIVES ON ANIMAL RESEARCH

Volume 1. Edited by Stephen R. Kaufman, MD, and Betsy Todd, RN. 134 pp. New York, Medical Research Modernization Committee, 1989. $15.00 (hardcover)

Perspectives on Animal Research was published by the Medical Research Modernization Committee, a strong proponent of animal rights, which objects to being called an "anti-vivisection society" because this term "does not enjoy a positive connotation" (page 121). The book is a hodgepodge of censure and criticism of contemporary animal research in the behavioral and medical sciences. Although the foreword states that "*Perspectives on Animal Research* will be a forum for thoughtful essays on issues related to animal research" and that "essays which support animal research are welcomed," the entire book is devoted to condemnations of medical experimentation using animals.

The first part of the book includes seven critiques of animal models of human diseases. Two critiques attack specific research grant applications. The second part of the book is devoted to toxicity testing in animals, with a rehashing of the animal rights groups' objections to the Draize test. Part 3 consists of essays on mathematical models vs animal models and the use of nonpatient animals in veterinary physiology and pharmacology courses. In the latter essay, the author reports on a survey of "52 students representing 20 of 27 American veterinary schools," hardly a representative sample.

Part 4 of the book contains four book reviews. All four books were published, supported, and endorsed by animal rights groups such as the American Anti-Vivisection Society and the Physicians' Committee for Responsible Medicine. All four books were reviewed by animal rights supporters. The final section of the book presents two brief commentaries by the vice chairman of the Medical Research Modernization Committee which published the book.

This book might have had some redeeming features had it presented point and counterpoint with essays that document the views of both those who support and those who oppose the use of animals for medical research. Unfortunately the book in its entirety is a denunciation and condemnation of most medical research that uses animals. A biased, one-sided presentation is not helpful to a neutral reader. This book is therefore not recommended.

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PRACTICE MADE PERFECT: THE PHYSICIAN'S GUIDE TO COMMUNICATION AND MARKETING

By Edna Kaplan, MS. 261 pp. Boston, Barrington Press, 1990. $39.95 (hardcover)

This book is an interesting hybrid, partially a "how to" book and partially a tour through the collective wisdom of a variety of medical practice analysts. Those range from Arnold Relman, MD, Editor of the *New England Journal of Medicine*, to a bevy of experts on marketing, business promotion, and advertising, the latter often viewing medicine from a client-consultant perspective.

The author possesses a Master of Science degree in communications and is a public relations practitioner. At one time she managed a busy group-practice office and has also held other medically related jobs. She is the wife of a physician.

When Kaplan discusses doctors and their problems, she is clearly familiar with the issues that trouble them. Her stance is strongly pro fee-for-service private practice. Her principal message is that doctors should make every effort to be more effective communicators. She stresses that this is essential for physicians in adapting to what has become a hostile environment, long on public suspicion and short on the traditional respect that most doctors probably feel they deserve. On the last page, the author states her belief that "above all, gratifying doctor-patient relationships, always based on communication skills, are at the heart of a rewarding lifetime career."

A logical progression of chapters first discuss conservative communication strategies. These involve assessing one's practice and finding new ways to satisfy patients, developing referral sources and participating in both individual patient and public health education. More aggressive techniques are presented that are a response to the current competitive environment. These include involvement in marketing, public relations and perhaps securing media attention. On the one hand, the reader will learn how to prepare for a radio or TV interview (the advice is good), and on the other, how to use marketing to reshape an unrewarding career.

Paid promotion and advertising are aggressive strategies. Kaplan stresses that local norms make advertising acceptable only in parts of Florida, California, and Nevada. Nevertheless, her chapter on advertising is one of the best in the book. It stands in contrast to a discussion of billing and collection techniques which is somewhat superficial.

The subtleties of the differences between marketing, public relations, and advertising are explored. The confusion that these engender when blended with traditional medical values is epitomized by a statement on page 101: "An image of community participation and social responsibility may be just the shot in the arm the medical profession needs at a time when the future of medical practice is being publicly weighed."

Though the author takes pains to point out that "all the publicity in the world won't generate a thriving practice for a physician perceived by patients to be uncaring or incompetent," the question of whether one is talking about the candy bar or the wrapper never did seem to be satisfactorily resolved for this reader from the old school.

Practice Made Perfect covers familiar ground in a clear, well-organized way. It will likely be thought provoking for physicians who have significant doubts about their practices. The book's principal weakness lies in its hybrid nature. It is neither definitive in the "how-to" sense in too many areas nor sufficiently academic (despite 19 pages of references) to qualify as a scholarly contribution. The author is also too modest, often qualifying statements as the opinion of other consultants and authorities when they are also clearly what she herself wants to say.

The book is not sufficiently different

from others of its type to justify the promise of its catchy title. But its author does emerge as a likable and decent individual, traits that are very disarming to a physician who has serious doubts about the commercialization of medical practice.

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THE ENCYCLOPEDIA OF PHOBIAS, FEARS, AND ANXIETIES

By Ronald M. Doctor, PHD, and Ada P. Kahn, MPH. 487 pp. New York, Facts on File, Inc, 1989. $40.00 (hardcover)

This is one of a series of encyclopedias on subjects ranging from suicide, drug abuse, and child abuse to an upcoming title on women's sexuality. The last named is edited by Ada P. Kahn, MPH, a graduate of the Northwestern University School of Medicine. She is also the co-editor of the volume being reviewed here, along with Ronald P. Doctor, PHD, Professor of Psychology at California State University, Northridge. A member of the Phobia Society of America, he must have provided the technical skeleton on which Ms Kahn has applied a literary fleshing. The book has more than 450 double-column pages of text with alphabetized entries. The boldfaced headings stand out for easy assimilation of information. The emphasis in the book seems to be listing every kind of phobia and "fear of" under the sun. My uncharitable thought at one point was whether the authors themselves were not suffering from a fear of not including *everything.* It is true that in the preface, inclusion over restriction is stressed, but scotomaphobia (fear of blind areas in the visual field), thalassophobia (fear of sitting down) and fear of doorknobs seem rather farfetched in a professional book.

This is not to say that the book does not have some excellent encapsulated accounts of more common phobias. It does. There are phobias mentioned to gladden the hearts of many a fiction writer: hylephobia (fear of empty rooms), lacanophobia (fear of vegetables), dromophobia (fear of crossing streets). Imagine what Mr Alfred Hitchcock could have done with that one, and many others. The two detailed sections, on agoraphobia and the history of phobias, stand out as among the best entries in the book.

There is a fairly detailed account of anxiety followed by a self inventory of anxiety symptoms. Obsessive compulsive disorder and depression are covered, albeit skimpily. There are entries on a variety of related topics, including anorexia nervosa, panic, and post-traumatic stress disorder.

There are good sections on some of the therapeutic approaches. Behavior therapy, psychotherapy, cognitive therapy, biofeedback, and others are covered. The areas that I found weak were the sections on medications. Too many outdated pieces of information ("lithium's side effects may rule it out as an antidepressant") and lack of cohesiveness in the presentation make these entries lose some appeal.

In general, the book is an attractive compendium of information on phobia and anxiety. There are bonuses in the form of short biographical sketches of Sigmund and Anna Freud, Janet, Wolpe, Pavlov, and others, and references at the end of important entries. The extensive bibliography at the end is certainly a plus. There is no current book with exactly the same format and its appeal to lay readers will be considerable. There is a tremendous amount of material presented in an easy to understand manner. Students of medicine, psychology and even first year residents in psychiatry will find it useful for quick reference and interesting bits of information. This certainly means that the authors have abundantly fulfilled what they set out to accomplish.

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LETTING BLOOD

By Richard Platt, MD, and Orah Platt, MD. 176 pp, illustrated. New York, St Martin's Press, 1988. $14.95 (clothcover)

Devotees of the murder and mayhem literary genre know that female sleuths are rare, Jane Marple, Jessica Fletcher, and Nancy Drew notwithstanding. When they put on their gumshoes, they are usually in the company or employ of some male whose superior intelligence has to be thwarted before the womenfolk are acclaimed for the clever little vixens they really are. This slender but fast-paced book gives us a new twist, the shamuses being an egalitarian husband-and-wife doctor/doctor team, whose intelligence is honed to a lethal edge by their training, respectively, in epidemiology and infectious disease, and hematology. Do doctors make a good team of private eyeballs? You bet they do! Selleck, you've finally been outclassed by a pair of Ivy League Yuppie docs who never wash their teacups, produce kids named Abe, Isaac, and Jake, and maintain a dog named Whimsy.

The crime is murky. A bigshot has suddenly gone to meet his maker in the teaching hospital we all know and love. It takes our epidemiologist half the book to come to the conclusion that the death was bizarre enough to warrant his wife's diagnosis of murder by AIDS. The pursuit of the facts, and the identification of the perpetrator constitute the text into which all the hideous facets of modern hospital life are woven. *House of God*, move over and lie down with Selleck. This is what you would have looked like had Sam Shem written his immortal tribute to "back-stage at the ICU" in this, the computer era.

The Platts' eye for detail is extraordinary, and we are bombarded with it, to the point that we feel we have been in every room, and known every person mentioned in the book. Strangely, this intimacy seems to detract from the suspense of the action itself, as we are more intrigued by the clever descriptions than with the events unfolding. And the book is nothing if not clever. A character named Ru Low could only have been named by a hematologist, and who but two physicians could have dreamed up a happy hour at a place called the Vermillion Border. The sheer number of puns, double entendres, and sly little digs are so overwhelming to those "in the know" as to make them wonder how anyone but a doctor could read this book without missing half the fun.

I suppose one of the perks of being a doctor is to be able to read a "detective" novel that breaks many of the rules of competence and still find it fun because it both mocks and extols the world we know. Seeing all that we have witnessed exposed with articulate cynicism is a great balm to our troubled spirits. I carefully refrain from saying any more about the theme and development of this whodunnit except to note that it is commendable vacation reading, and would make a great movie. With a little more practice, Richard and Orah Platt could well join Michael Crichton, Robin Cook, and others of their ilk for another round of ratiocination, CPC style.

NAOMI R. BLUESTONE, MD, MPH
Consulting Editor

Trends in medical school enrollment

The results of a yearly evaluation of US medical schools revealed a slight increase in the number of applicants for the school year 1989–1990 (*JAMA* 1990; 264:809). The class entering in 1989 demonstrated a greater percentage of women and minorities than in the previous year.

The study also showed a small decline both in the percentage of students with "A" grade point averages (3.5–4.0) and in four of six Medical College Admissions Test (MCAT) subtest scores.

Vacancy rates for full-time faculty exceeded 5% in the following departments: genetics, pathology, dermatology, family medicine, neurology, obstetrics-gynecology, orthopedics, otolaryngology, pediatrics, and surgery.

The number of applicants rose by 194 from 26,721 in 1988–1989. The applicant acceptance ratio was 1.6, as it was in 1988–1989. This compares with a ratio of 2.1 in the first half of the 1980s.

New "sharps" disposal system reduces waste

"Sharps," or medical utensils that have a sharp edge or point, such as syringes, scalpels, and razor blades, pose a potential hazard to health care workers. Nearly half of all "needle-stick" injuries occur after the needle has been in contact with blood and may be contaminated with such infectious agents as human immunodeficiency virus (HIV) and hepatitis B. Packaging, transporting, and storing sharps at disposal sites increases the risk to waste handlers and the environment.

A new Sharps Disposal System (Disposal Sciences, Inc, Engelwood, Colo), based on thermal decontamination, features a reusable collection unit and a central processing unit. Sharps made of metal, glass, plastic, or other material are placed in a collection unit immediately after use. There is no further contact with the material and no recapping of needles—a major cause of needle-stick accidents. Attachment of a full collection unit to the central processing unit is followed by automatic transfer of the material, grinding, and melting, to produce a compact, pathogen-free, disposable disk that can be treated like ordinary trash.

Federal funds allocated for clinical care of AIDS patients

On August 18, 1990, President Bush signed into law the Ryan White Comprehensive AIDS Resources Emergency Act, which provides for federal funding for the clinical care of patients with AIDS.

Effective for the fiscal year beginning in October 1990, the bill would earmark up to $875 million in federal assistance for four new programs aimed at improving the diagnosis and treatment of patients with AIDS (*The Morning Call*, August 2, 1990, pA15). This money would not come out of the $1.7 billion proposed by the Bush administration to combat AIDS in the upcoming year; these funds would continue to support research and educational efforts.

One of the programs targeted by this new legislation would provide as much as $275 million to assist cities that have reported the greatest numbers of AIDS cases. Another $275 million would be distributed to states in the form of grants according to the number of AIDS cases reported during the previous two years. Early intervention efforts, including HIV screening, patient counseling, and availability of drugs to treat AIDS, would get a boost from $305 million in federal grants.

NIH consensus paper on surgery for epilepsy

With growing frequency, brain surgery has become an accepted alternate treatment for patients with epilepsy that is refractory to antiepileptic drug therapy. The National Institutes of Health (NIH) has prepared a summary of its recent Consensus Development Conference on surgery for epilepsy.

The NIH reports that 10–20% of the 150,000 people in whom epilepsy develops each year have seizures that do not respond to drug therapy. Whereas currently approximately 500 patients undergo surgery each year for epilepsy, surgery may be a viable alternative for 2,000–5,000 new patients.

Surgery may ameliorate three types of seizures: "partial seizures of temporal or extratemporal origin; secondarily generalized seizures (attacks that begin locally and spread to both sides of the brain); or unilateral, multifocal epilepsy associated with infantile hemiplegia (paralysis of one side of the body)."

Prior to even considering surgery for the prevention of seizures, the NIH recommends a neurologic examination that confirms a diagnosis of epilepsy, determines the type and syndrome of the seizures, and identifies the metabolic or physiologic cause of the seizures, as well as an acceptable trial of antiepileptic medication.

Surgical options include removal of epileptogenic tissue, which is associated with a 30–70% success rate depending on the origin of the seizures, section of the corpus collosum for patients with generalized seizures, and hemispherectomy for patients with seizures that result in infantile hemiplegia.

Hydergine shown ineffective in Alzheimer's disease

The only medication approved by the US Food and Drug Administration to treat Alzheimer's disease is Hydergine, a combination of ergoloid mesylates (*N Engl J Med* 1990; 323:445–448). However, the authors concluded that "the administration of Hydergine-LC for up to 24 weeks, although safe and well tolerated, had no notable beneficial effects on our patients with probable Alzheimer's disease." The authors performed a double-blind, placebo-controlled trial with 80 patients with a clinical diagnosis of Alzheimer's disease. They evaluated the safety and efficacy of Hydergine-LC, a newer preparation of ergoloid mesylates that may offer greater bioavailability.

In general, the results of cognitive and behavioral tests demonstrated that the condition of both the Hydergine-LC and placebo groups deteriorated during the 24-week

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NEWS BRIEFS are compiled and written by Vicki Glaser.

treatment period, and there were no noteworthy significant differences between the two groups in the test scores. The authors noted that the recommended dosage of 3 mg/day in divided doses may not be sufficient to evoke a beneficial response.

Study reveals relationship between placental size, birth weight, and hypertension in adult life

A sample of 449 men and women born as singleton infants in Britain between 1935 and 1943, for whom there were complete and detailed birth records, served as the study population in this evaluation of intrauterine growth and hypertension (*Br Med J* 1990; 301:259–262).

The authors found such a strong correlation between systolic and diastolic blood pressures and placental weight in their initial study of 259 subjects that they repeated the evaluation with another group of 190 subjects. The results were the same. "For each birth weight systolic and diastolic pressures rose with placental weight, and for each placental weight both pressures fell with increasing birth weight . . . .The highest pressures were among people who had been small babies with large placentas and the lowest were among people who had been large babies with small placentas."

Based on these findings, the authors suggest that two routine measurements taken at birth—birth weight and placental weight— may be useful in predicting the risk of hypertension later in life.

Simulated effects of the planned Medicare Fee Schedule

The Medicare Fee Schedule (MFS), mandated by the Omnibus Budget Reconciliation Act of 1989, "will dramatically alter how Medicare pays physicians," and "could profoundly alter the economics of medicine," according to Levy et al of the Health Care Financing Administration (Baltimore, Md) and the Prospective Payment Assessment Commission (Washington, DC) (*JAMA* 1990; 264:717–722). The authors based these observations on their simulation models of the effects of the Medicare legislation.

The MFS, which will go into effect in 1992, will differ in several ways from the current Customary, Prevailing, and Reasonable (CPR) payment system. The complex formula used to determine MFS payments incorporates seven variables: the procedure's relative value for physician work; the procedure's relative value for overhead costs; the procedure's relative value for malpractice costs, an index that adjusts for geographic differences in the value of physician work; an index that adjusts for geographic differences in physician overhead; an index that adjusts for geographic differences in physician malpractice; and a conversion factor that converts these relative values into payment amounts.

The simulations demonstrated that under the MFS, redistributions by specialty would result in substantially higher total payments for specialties such as family practice and internal medicine, and smaller payments for ophthalmology, radiology, pathology, and thoracic surgery. The redistribution effects on individual providers will vary; for example, "overall Medicare payments to orthopedic surgeons will change only 2%, but half of orthopedic surgeons will experience a gain or loss of at least 10%.

Most surgical and imaging procedures would be allowed reduced charges under the new legislation, whereas payments would increase for visits and consultations.

Two important variables that could greatly influence the outcome of the MFS could not be incorporated in these simulations: whether physicians will adjust the degree of services they provide or their coding practices in response to the MFS; and whether payers in addition to Medicare will adopt the new Medicare Fee Schedule.

Study refutes symptom-provocation method for determining food allergies

The symptom-provocation procedure is a controversial method of diagnosing food allergies and other sensitivities. It involves challenging a person with intradermal injections of extracts of suspected allergens and monitoring for associated symptoms. Neutralization of the sensitivity reaction is achieved by injecting a different dose of the specific allergen.

Duplicating the symptom-provocation procedure commonly used in clinical practice, Jewett and colleagues carried out a double-blind study with 18 patients. Active injections contained the suspected allergen, while control injections contained diluent composed of normal saline.

Of the active injections (60), the subjects judged 27% (16) to contain an allergen; whereas, for the control injections, 44 of 180 or 24% of these injections were also judged to contain the active substance.

"We had expected the rate of reaction to the diluent to be much lower," stated the authors. They concluded that the symptoms reported were "placebo responses" and were in no way related to the contents of the injections. "A system of treatment based on the relief of symptoms alone may come to be dominated by placebo reactions," warned the authors. Questioning the accuracy and effectiveness of the symptom-provocation method, Jewett et al emphasized the need for double-blind testing in the clinical setting as well, in order to evaluate and to avoid the effects of suggestion and anxiety.

Isabel Beck, MD, Albany. Died January 16, 1990; age 92. Dr Beck was a 1923 graduate of the Columbia University College of Physicians and Surgeons, New York. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Ivan L. Bennett, MD, New York. Died July 21, 1990; age 68. Dr Bennett was a 1946 graduate of the Emory University School of Medicine, Atlanta, Georgia. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Philip Sandler Bergman, MD, New York. Died July 23, 1990; age 69. Dr Bergman was a 1944 graduate of the Tulane University School of Medicine, New Orleans, Louisiana. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Neurological Association, the New York County Medical Society, and the Medical Society of the State of New York.

Ulric Irving Carrington, MD, Derry, New Hampshire. Died August 29, 1989; age 82. Dr Carrington was a 1935 graduate of the Howard University College of Medicine, Washington, DC. His memberships included the American Gastro-Enterological Association, the New York County Medical Society, and the Medical Society of the State of New York.

Irving I. Crouse, MD, Buffalo. Died May 26, 1990; age 90. Dr Crouse was a 1925 graduate of the State University of New York Upstate College of Medicine, Syracuse. His memberships included the American Academy of Family Practice, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Benedict J. De Respinis, MD, Staten Island. Died July 20, 1990; age 83. Dr De Respinis was a 1934 graduate of the Facolta di Medicina e Chirurgia dell'Universita di Napoli, Naples, Italy. He was a member of the Richmond County Medical Society and the Medical Society of the State of New York.

Louis S. Del Bello, MD, Webster, Massachusetts. Died July 8, 1990; age 84. Dr Del Bello was a 1935 graduate of the State University of New York at Buffalo School of Medicine, Buffalo. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Leo Duschnitz, MD, New York. Died October 5, 1989; age 93. Dr Duschnitz was a 1921 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Alfred John Ephraim, MD, Miami Beach, Florida. Died May 28, 1990; age 92. Dr Ephraim was a 1924 graduate of the Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Germany. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Raymond E. Frisk, MD, Elmira. Died July 4, 1990; age 66. Dr Frisk was a 1952 graduate of the Georgetown University School of Medicine, Washington, DC. He was a Diplomate of the American Board of Family Practice. He was a Fellow of the American College of Emergency Physicians and a Fellow of the American Academy of Family Practice. He was a member of the Medical Society of the County of Chemung and the Medical Society of the State of New York.

John Henry Hamel, MD, Jamesville. Died July 17, 1990; age 76. Dr Hamel was a 1940 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Diplomate of the American Board of Urology. His memberships included the New York Academy of Medicine, the American Urological Association, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Kenneth G. Jennings, MD, Brooklyn. Died June 30, 1990; age 93. Dr Jennings was a 1922 graduate of the New York University School of Medicine. He was a Diplomate of the American Board of Pediatrics. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Burton Stanley Koplon, MD, New York. Died April 24, 1990; age 52. Dr Koplon was a 1963 graduate of the Birmingham Medical College, Birmingham, Alabama. He was a Diplomate of the American Board of Dermatology, Inc. His memberships included the New York State Dermatological Society, the New York Academy of Medicine, the American Academy of Dermatology, the New York County Medical Society, and the Medical Society of the State of New York.

Yuan Kuang Ku, MD, Brooklyn. Died February 16, 1990; age 57. Dr Ku was a 1955 graduate of the Second Shanghai Medical College, Shanghai, China. He was a Diplomate of the American Board of Internal Medicine. He was a Fellow of the American College of Cardiology. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Edward J. Matthews, MD, Staten Island. Died July 5, 1990; age 77. Dr Matthews was a 1939 graduate of the Middlesex University School of Medicine, Waltham, Massachusetts. His memberships included the American Academy of Family Practice, the Richmond County Medical Society, and the Medical Society of the State of New York.

Helen Lovena Miller, MD, Kinnelon, New Jersey. Died June 18, 1990; age 94. Dr Miller was a 1926 graduate of the University of Oregon Medical School, Portland, Oregon. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Francisco Negrete, MD, Flushing. Died July 10, 1990; age 65. Dr Negrete was a 1953 graduate of the Facultad de Medicina de la Universidad de La Habana, La Habana, Cuba. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a Fellow of the American College of Obstetricians and Gynecologists. His memberships included the American Fertility Society, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Nicholas R. Occhino, MD, Johnson City. Died July 8, 1990; age 84. Dr Occhino was a 1932 graduate of the State University of New York Upstate College of Medicine, Syracuse. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. His memberships included the American Society of Clinical Hypnosis, the Broome County Medical Society, and the Medical Society of the State of New York.

Ben Sobel, MD, Willow Grove, Pennsylvania. Died June 18, 1990; age 73. Dr Sobel was a 1932 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a member of the Broome County Medical Society and the Medical Society of the State of New York.

Samuel Carlyle Trattler, MD, New York. Died June 24, 1990; age 92. Dr Trattler was a 1923 graduate of the New York Medical College. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a Fellow of the American College of Obstetricians and Gynecologists and a Qualified Fellow of the International College of Surgeons. His memberships included the New York Academy of Medicine, the Pan American Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Michael F. Volpe, MD, Brooklyn. Died December 19, 1989; age 86. Dr Volpe was a 1926 graduate of the New York University School of Medicine. He was a Fellow of the American College of Surgeons. His memberships included the American Society of Abdominal Surgeons, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Fred Morris Weiss, MD, New Bedford, Massachusetts. Died November 4, 1989; age 87. Dr Weiss was a 1928 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Myron Wright, MD, New York. Died March 29, 1990; age 75. Dr Wright was a 1940 graduate of the New York University School of Medicine, New York. His memberships included the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1988; 88:456–462). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al*. Names of journals should be abbreviated according to *Index Medicus* and underlined.

Sample references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine*, PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

ACQUIRED IMMUNODEFICIENCY SYNDROME

Current Issues and Scientific Studies

Edited by Pascal James Imperato, MD

With a foreword by
George J. Lawrence, Jr., MD, Chairman
AIDS Task Force
Medical Society of the State of New York

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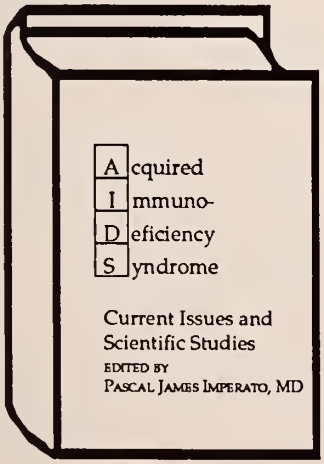

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(*continued from p 9A*)

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Nov 10. **Fiberoptic Endoscopy Workshop for the Management of the Difficult Airway.** 8 Cat I Credits. Alumni Hall, NYU Medical Center. *Nov 30.* **New Directions for Platinum Compounds in the Nineties.** 8 Cat I Credits. Grand Hyatt Hotel. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Avenue, New York, NY 10016. Tel: (212) 340-5295.

Nov 18. **Perinatal AIDS: Medical Perspectives on Transmission, Diagnosis, Treatment.** New York Hospital-Cornell Medical Center. Contact: Judith E. Steinhart, Director of Professional Education, Program Development Department, Greater New York March of Dimes, 233 Park Ave South, New York, NY 10003. Tel: (212) 353-8353.

Nov 30. **Management of Cerebral Gliomas in Adults.** 7 Cat I Credits. Holiday Inn Crowne Plaza at La Guardia Airport. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

Nov 30–Dec 1. **Recent Advances in Understanding the Addictive States.** Penta Hotel. Contact: Ivan Bodis-Wollner, MD, Secretary-Treasurer, Association for Research in Nervous and Mental Disease, Box 1052, Mount Sinai Hospital, One Gustave L. Levy Place, New York, NY 10029. Tel: (212) 348-8133.

NEW HYDE PARK

Nov 17. **4th Annual Conference on Common Orthopaedic Problems in Children.** 8 Cat I Credits. Teaching Center Auditorium, Long Island Jewish Medical Center. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Medical Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

SYRACUSE

Nov 30–Dec 1. **41st Annual Post-graduate Review Course in Ophthalmology.** 14 Cat I Credits. Hotel Syracuse. Contact: Office of Continuing Education. SUNY Health Science Center at Syracuse, 750 East Adams St, Syracuse, NY 13210. Tel: (315) 464-4607 or (800) 283-4606.

AROUND THE NATION

ARIZONA

Nov 15–18. **Psychiatric Medicine in the '90s. The Emergence of Consultation/Liaison Psychiatry.** The Pointe at Squaw Peak, Phoenix. Contact: Executive Director, Academy of Psychosomatic Medicine, 5824 North Magnolia, Chicago, IL 60660. Tel: (312) 784-2025.

CALIFORNIA

Nov 2–3. **Update on Thyroid Disease 1990.** 11½ Cat I Credits. Hyatt Regency Embarcadero. *Nov 8–10.* **The UCSF Otolaryngology Update: 1990.** 18 Cat I Credits. Union Square Holiday Inn, San Francisco. *Nov 9–11.* **The 36th Annual Group Therapy Symposium.** Sir

(*continued on p 17A*)

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Nov 9–11. **Advanced Seminars in Diagnostic Imaging.** 20 Cat I Credits. Ritz-Carlton Resort Hotel, Laguna Niguel. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

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Nov 19–21. **IV Curso en Espanol de la Academia Americana de Neurologia.** 20 Cat I Credits. Holiday Inn, Orlando. Contact: Office of Continuing Education, SUNY Health Science Center at Syracuse, 750 East Adams St, Syracuse, NY 13210. Tel: (315) 464-4606.

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Nov 12–14. **NIH Consensus Development Conference: Clinical Uses of Botulinum Toxin.** Masur Auditorium, NIH Clinical Center. Contact: William H. Hall, Director of Communications, Office of Medical Applications of Research, National Institutes of Health, Bldg 1, Rm 259, Bethesda, MD 20892. Tel: (301) 496-1143.

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Nov 7–9. **Lasers in Gynecology.** 21 Cat I Credits. Worcester. Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024. Tel: (202) 638-5577.

Nov 16–20. **43rd Annual Scientific Meeting of The Gerontological Society of America.** Boston Marriott Copley Place. Contact: Elizabeth Borgen, Director of Promotion, The Gerontological Society of America, 1275 K Street, NW, Suite 250, Washington, DC 20005. Tel: (202) 842-1275.

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Nov 8–10. **Fall Update in Geriatrics.** 18 Cat I Credits. Omni Hotel at Charleston Place, Charleston. Contact: Gerri LaVia, Department of Family Medicine, Medical University of South Carolina, Charleston, SC 19425. Tel: (803) 792-2426. □

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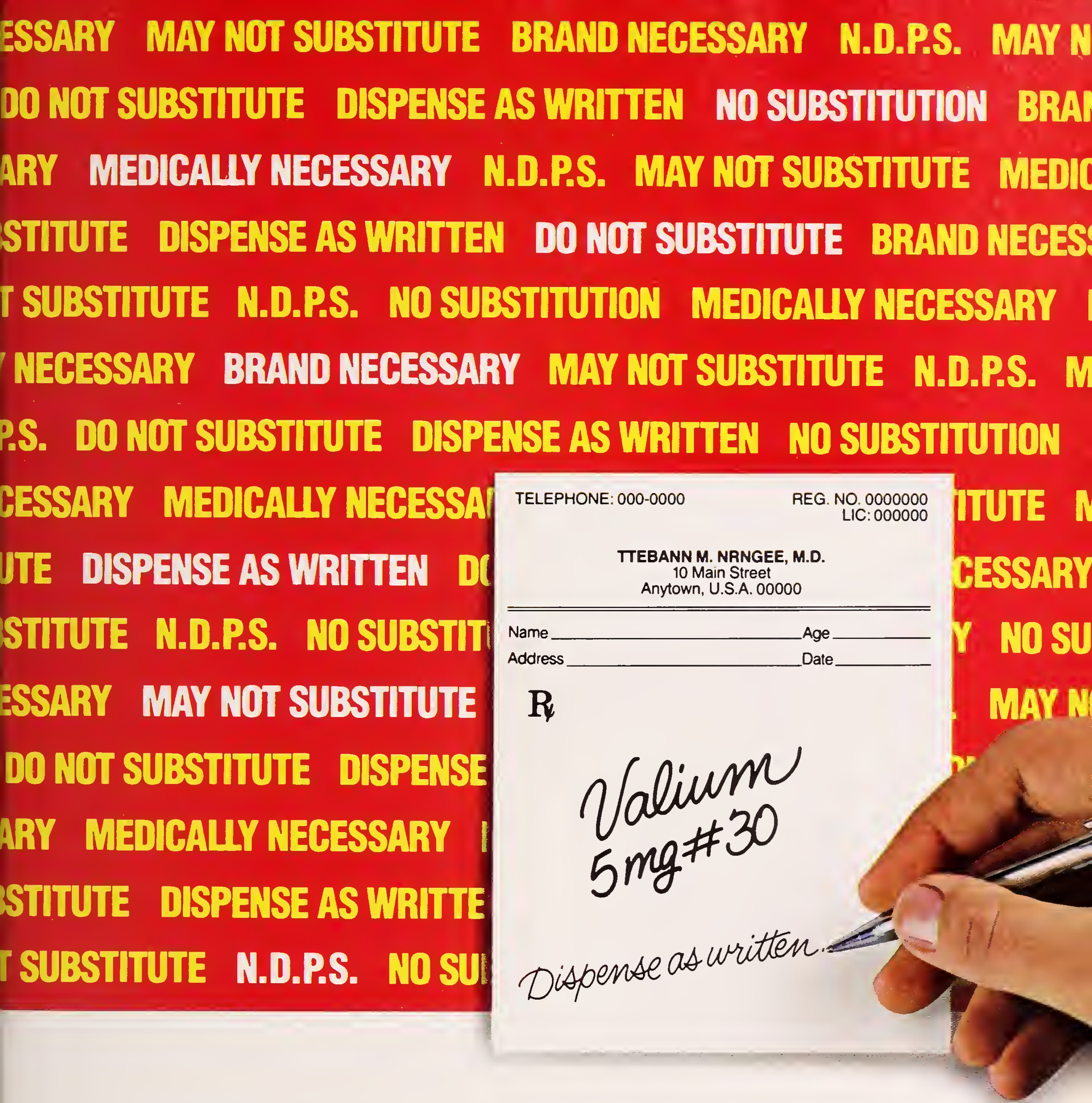
Cahners Publications 3A, 15A
Cutter Biological 5A
Excu-Flow Systems 13A
Kern & Augstine 12A
Eli Lilly and Company 7A
Marine Midland Bank .... 2nd Cover
Metropolitan Life 8A
Palisades Pharmaceuticals 9A
Roche Laboratories .... 3rd & 4th Cover
Searle Pharmaceuticals 1A
U.S. Air Force 17A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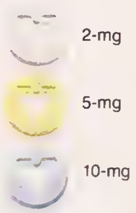

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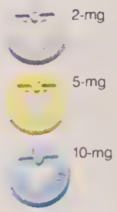

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VOL 90
NO 11
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1990

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MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

DECEMBER 1990

AROUND THE STATE

BRONX

Dec 5. **Forensic Techniques of the Crime Scene Unit.** Lehman College campus. Contact: Mark L. Taff, MD, President, New York Society of Forensic Sciences, 511 Hempstead Ave, Suite 2, West Hempstead, NY 11552. Tel: (516) 292-2300.

MANHATTAN

Dec 1–2. **Therapeutic Alternatives in Urology.** 15½ Cat I Credits. NYU Medical Center. *Dec 3–6.* **Neurosurgery NYC/1990.** 24 Cat I Credits. Grand Hyatt. *Dec 7–9.* **Review of Genito-Urinary Pathology.** 16 Cat I Credits. NYU Medical Center. *Dec 8.* **The Evaluation and Management of Epilepsy.** 7 Cat I Credits. UN Plaza Hotel. *Dec 10–12.* **Managing Clinical Problems in the Elderly.** 18½ Cat I Credits. NYU Medical Center. *Dec 17–22.* **9th Annual CT/MRI Head To Toe.** 39½ Cat I Credits. Grand Hyatt. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 340-5295.

Dec 5. **Prognosis After MI.** 2 Cat I Credits. New York Academy of Medicine Building. Contact: The New York Cardiological Society, Inc, 681 Lexington Ave, Suite 310, New York, NY 10022. Tel: (212) 888-5595.

Dec 5–7. **Controversies, Problems, and Techniques in Surgery.** 21 Cat I Credits. Grand Hyatt Hotel. Contact: Office of Continuing Medical Education, Albert Einstein College of Medicine/Montefiore Medical Center, 3301 Bainbridge Ave, Bronx, NY 10467. Tel: (212) 920-6674.

Dec 6–7. **Total Knee Reconstruction: Current State of the Art.** 12 Cat I Cred-

(continued on p 15A)

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Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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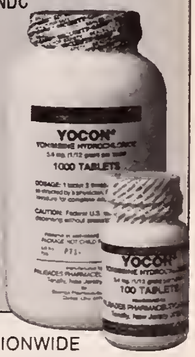


New York State Journal of Medicine

NOVEMBER 1990 / Volume 90, Number 11

Contents

COMMENTARIES

531 **A reevaluation of physician recredentialing**
PETER B. FARNSWORTH, MD

533 **Rural obstetric care and the family physician**
DOUGLAS R. SMUCKER, MD

535 **Adenocarcinoma of the cervix**
MICHAEL P. HOPKINS

RESEARCH PAPERS

537 **Primary care obstetrics in rural Western New York: A multi-center case review**
THOMAS C. ROSENTHAL, MD; DAVID M. HOLDEN, MD; WILLOW WOODWARD, BS

541 **The prevalence of subclinical hypothyroidism in adults with low-normal blood thyroxine levels**
MICHAEL JOSEPH MILLER, MD; CHAO PAN, MA; URIEL S. BARZEL, MD

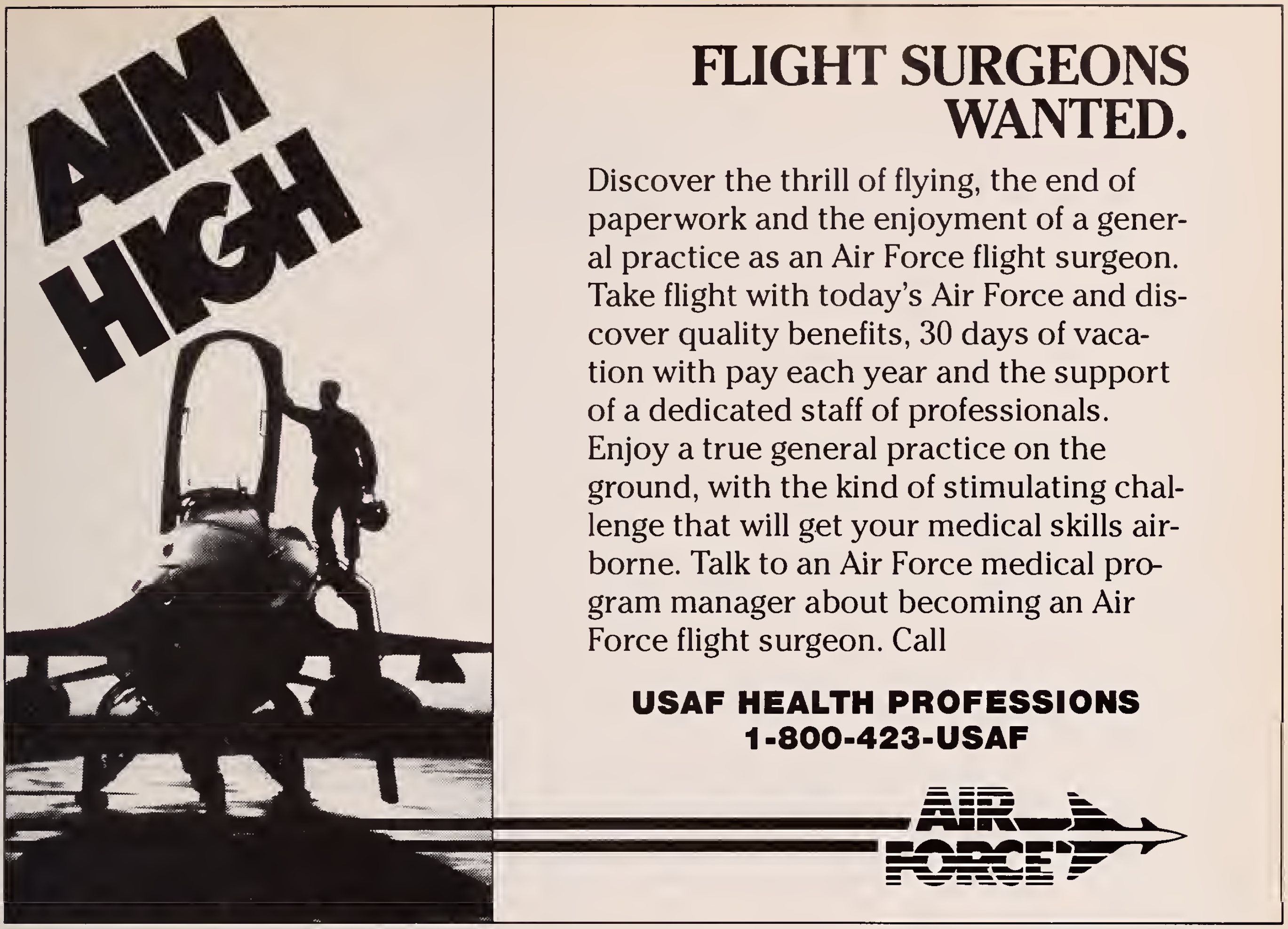
SPECIAL ARTICLE

545 **An overview of the activities of the New York State Board for Medicine**
HENRY A. FERNANDEZ, JD, THOMAS J. MONAHAN, MA

COMMITTEE REPORT

552 **The allocation of scarce medical resources**
FRED ROSNER, MD; PETER P. SORDILLO, MD; PHILIP H. SECHZER, MD; HERMAN M. RISEMBERG, MD; FERNANDO V. ONA, MD; PATRICIA J. NUMANN, MD; LAURENCE LOEB, MD; PETER B. FARNSWORTH, MD; ALLEN J. BENNETT, MD; JONATHAN R. WOLPAW, MD; PETER ROGATZ, MD; ALLISON B. LANDOLT, MD; PIETER R. KARK, MD; VIRGINIA KANICK, MD; JOHN B. HENRY, MD; ABRAHAM L. HALPERN, MD; ERIC J. CASSELL, MD

(*continued on page 6A*)



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FULL TIME TRAVEL REQUIRED (one year minimum). Health research organization needs Physicians for National Health and Nutrition Examination Survey sponsored by the U.S. Public Health Service. Individuals will be part of a large medical team conducting health examinations in government examination centers traveling to 68 areas of the U.S. through 1993. Physicians must be licensed in one state. Competitive salary, paid malpractice, per diem, car, four weeks paid vacation per year, holidays, and health, life, dental, disability insurance offered. Call Beverly Geline at 800-937-8281, ext. 8248. Westat, Inc., Rockville, Maryland. EOE/M/F/V/H.

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New York State Journal of Medicine

CONTENTS, CONT'D

CASE REPORTS

559 **Primary signet ring cell carcinoma of the uterine cervix**
UTE M. MOLL, MD; JOHN C. CHUMAS, MD; WILLIAM J. MANN, MD; BRUCE PATSNER, MD

561 **Intestinal obstruction secondary to bezoars**
RAYMOND D. LARAJA, MD; AMEDEO USAI, MD; EDITH PRYCE, MD; MICHAEL CIOROIU, MD

562 **Metastatic cancer of the spinal intradural extramedullary space**
RAMACHANDRA C. REDDY, MD; REZA KHATIB, MD; GEORGE S. AZER, MD; RAJA SALEM, MD

LETTERS TO THE EDITOR

565 **Health and health care challenges for New York City in the 1990s**
ADRIAN W. ZORGNIOTTI, MD

565 **The New York State Department of Health's Part 405 regulations**
ARNOLD B. RITTERBAND, MD

566 **Gender and sex**
PAUL DER KOLISCH, MD

566 **Cross-dressing successfully treated with fluoxetine**
VIRGINIA T. JORGENSEN, MD

ESSAY REVIEW

568 **Unnatural causes: America's three leading killer diseases**
ALLEN D. SPIEGEL, PHD

572 LEADS FROM EPIDEMIOLOGY NOTES

576 BOOK REVIEWS

581 NEWS BRIEFS

583 OBITUARIES

3A MEDICAL MEETINGS AND LECTURES

CONTROL
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See adjacent page for Brief Summary
of Product Information

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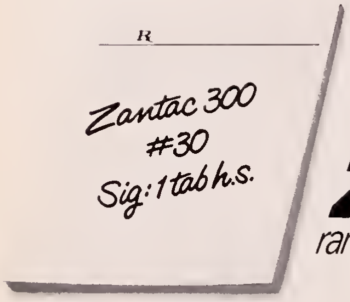

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Zantac® 300 Tablets
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BRIEF SUMMARY

The following is a brief summary only. Before prescribing, see complete prescribing information in Zantac® product labeling.

INDICATIONS AND USAGE: Zantac® is indicated in:
1. Short-term treatment of **active duodenal ulcer.** Most patients heal within four weeks.
2. **Maintenance therapy** for duodenal ulcer patients at reduced dosage after healing of acute ulcers.
3. The treatment of **pathological hypersecretory conditions** (eg, Zollinger-Ellison syndrome and systemic mastocytosis).
4. Short-term treatment of **active, benign gastric ulcer.** Most patients heal within six weeks and the usefulness of further treatment has not been demonstrated.
5. Treatment of **gastroesophageal reflux disease (GERD).** Symptomatic relief commonly occurs within one or two weeks after starting therapy. Therapy for longer than six weeks has not been studied.

In active duodenal ulcer; active, benign gastric ulcer; hypersecretory states; and GERD, concomitant antacids should be given as needed for relief of pain.

CONTRAINDICATIONS: Zantac® is contraindicated for patients known to have hypersensitivity to the drug.

PRECAUTIONS:
General: 1. Symptomatic response to Zantac® therapy does not preclude the presence of gastric malignancy.
2. Since Zantac is excreted primarily by the kidney, dosage should be adjusted in patients with impaired renal function (see DOSAGE AND ADMINISTRATION). Caution should be observed in patients with hepatic dysfunction since Zantac is metabolized in the liver.

Laboratory Tests: False-positive tests for urine protein with Multistix® may occur during Zantac therapy, and therefore testing with sulfosalicylic acid is recommended.

Drug Interactions: Although Zantac has been reported to bind weakly to cytochrome P-450 *in vitro*, recommended doses of the drug do not inhibit the action of the cytochrome P-450-linked oxygenase enzymes in the liver. However, there have been isolated reports of drug interactions that suggest that Zantac may affect the bioavailability of certain drugs by some mechanism as yet unidentified (eg, a pH-dependent effect on absorption or a change in volume of distribution).

Carcinogenesis, Mutagenesis, Impairment of Fertility: There was no indication of tumorigenic or carcinogenic effects in lifespan studies in mice and rats at doses up to 2,000 mg/kg/d.

Ranitidine was not mutagenic in standard bacterial tests (*Salmonella, Escherichia coli*) for mutagenicity at concentrations up to the maximum recommended for these assays.

In a dominant lethal assay, a single oral dose of 1,000 mg/kg to male rats was without effect on the outcome of two matings per week for the next nine weeks.

Pregnancy: ***Teratogenic Effects: Pregnancy Category B:*** Reproduction studies have been performed in rats and rabbits at doses up to 160 times the human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Zantac. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: Zantac is secreted in human milk. Caution should be exercised when Zantac is administered to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

Use in Elderly Patients: Ulcer healing rates in elderly patients (65 to 82 years of age) were no different from those in younger age groups. The incidence rates for adverse events and laboratory abnormalities were also not different from those seen in other age groups.

ADVERSE REACTIONS: The following have been reported as events in clinical trials or in the routine management of patients treated with Zantac®. The relationship to Zantac therapy has been unclear in many cases. Headache, sometimes severe, seems to be related to Zantac administration.

Central Nervous System: Rarely, malaise, dizziness, somnolence, insomnia, and vertigo. Rare cases of reversible mental confusion, agitation, depression, and hallucinations have been reported, predominantly in severely ill elderly patients. Rare cases of reversible blurred vision suggestive of a change in accommodation have been reported.

Cardiovascular: As with other H_2-blockers, rare reports of arrhythmias such as tachycardia, bradycardia, atrioventricular block, and premature ventricular beats.

Gastrointestinal: Constipation, diarrhea, nausea/vomiting, abdominal discomfort/pain, and rare reports of pancreatitis.

Hepatic: In normal volunteers, SGPT values were increased to at least twice the pretreatment levels in 6 of 12 subjects receiving 100 mg qid intravenously for seven days, and in 4 of 24 subjects receiving 50 mg qid intravenously for five days. There have been occasional reports of hepatitis, hepatocellular or hepatocanalicular or mixed, with or without jaundice. In such circumstances, ranitidine should be immediately discontinued. These events are usually reversible, but in exceedingly rare circumstances death has occurred.

Zantac® 150 and 300 (ranitidine hydrochloride) Tablets
Zantac® (ranitidine hydrochloride) Syrup

Musculoskeletal: Rare reports of arthralgias.

Hematologic: Blood count changes (leukopenia, granulocytopenia, thrombocytopenia) have occurred in a few patients. These were usually reversible. Rare cases of agranulocytosis, pancytopenia, sometimes with marrow hypoplasia, and aplastic anemia have been reported.

Endocrine: Controlled studies in animals and man have shown no stimulation of any pituitary hormone by Zantac and no antiandrogenic activity, and cimetidine-induced gynecomastia and impotence in hypersecretory patients have resolved when Zantac has been substituted. However, occasional cases of gynecomastia, impotence, and loss of libido have been reported in male patients receiving Zantac, but the incidence did not differ from that in the general population.

Integumentary: Rash, including rare cases suggestive of mild erythema multiforme, and, rarely, alopecia.

Other: Rare cases of hypersensitivity reactions (eg, bronchospasm, fever, rash, eosinophilia), anaphylaxis, angioneurotic edema, and small increases in serum creatinine.

OVERDOSAGE: Information concerning possible overdosage and its treatment appears in the full prescribing information.

DOSAGE AND ADMINISTRATION: (See complete prescribing information in Zantac® product labeling).

Active Duodenal Ulcer: The current recommended adult oral dosage is 150 mg or 10 ml (2 teaspoonfuls equivalent to 150 mg of ranitidine) twice daily. An alternate dosage of 300 mg or 20 ml (4 teaspoonfuls equivalent to 300 mg of ranitidine) once daily at bedtime can be used for patients in whom dosing convenience is important. The advantages of one treatment regimen compared to the other in a particular patient population have yet to be demonstrated.

Maintenance Therapy: The current recommended adult oral dosage is 150 mg or 10 ml (2 teaspoonfuls equivalent to 150 mg of ranitidine) at bedtime.

Pathological Hypersecretory Conditions (such as Zollinger-Ellison syndrome): The current recommended adult oral dosage is 150 mg or 10 ml (2 teaspoonfuls equivalent to 150 mg of ranitidine) twice a day. In some patients it may be necessary to administer Zantac® 150-mg doses more frequently. Doses should be adjusted to individual patient needs, and should continue as long as clinically indicated. Doses up to 6 g/d have been employed in patients with severe disease.

Benign Gastric Ulcer: The current recommended adult oral dosage is 150 mg or 10 ml (2 teaspoonfuls equivalent to 150 mg of ranitidine) twice a day.

GERD: The current recommended adult oral dosage is 150 mg or 10 ml (2 teaspoonfuls equivalent to 150 mg of ranitidine) twice a day.

Dosage Adjustment for Patients with Impaired Renal Function: On the basis of experience with a group of subjects with severely impaired renal function treated with Zantac, the recommended dosage in patients with a creatinine clearance less than 50 ml/min is 150 mg or 10 ml (2 teaspoonfuls equivalent to 150 mg of ranitidine) every 24 hours. Should the patient's condition require, the frequency of dosing may be increased to every 12 hours or even further with caution. Hemodialysis reduces the level of circulating ranitidine. Ideally, the dosage schedule should be adjusted so that the timing of a scheduled dose coincides with the end of hemodialysis.

HOW SUPPLIED: Zantac® 300 Tablets (ranitidine hydrochloride equivalent to 300 mg of ranitidine) are yellow, capsule-shaped tablets embossed with "ZANTAC 300" on one side and "Glaxo" on the other. They are available in bottles of 30 (NDC 0173-0393-40) tablets and unit dose packs of 100 (NDC 0173-0393-47) tablets.

Zantac® 150 Tablets (ranitidine hydrochloride equivalent to 150 mg of ranitidine) are white tablets embossed with "ZANTAC 150" on one side and "Glaxo" on the other. They are available in bottles of 60 (NDC 0173-0344-42) and 100 (NDC 0173-0344-09) tablets and unit dose packs of 100 (NDC 0173-0344-47) tablets.

Store between 15° and 30° C (59° and 86° F) in a dry place. Protect from light. Replace cap securely after each opening.

Zantac® Syrup, a clear, peppermint-flavored liquid, contains 16.8 mg of ranitidine hydrochloride equivalent to 15 mg of ranitidine per 1 ml in bottles of 16 fluid ounces (one pint) (NDC 0173-0383-54).

Store between 4° and 25° C (39° and 77° F). Dispense in tight, light-resistant containers as defined in the USP/NF.

July 1990

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New York State
Journal of Medicine

COMMENTARIES

A reevaluation of physician recredentialing

In 1986, at the Annual Meeting of the Board of the Office of Professional Medical Conduct, the Chancellor of the Board of Regents of the State of New York claimed that there were many bad doctors in practice. The Commissioner of Health of the State of New York immediately agreed and announced that he would correct this situation by relicensing physicians. After a moment of reflection, he realized that licensure is the purview of the Department of Education. He then considered the term "recertification," but realized that this is a function of the specialty boards. He eventually settled for the term "recredential."

There is a problem with the term recredential. There is only one definition of recredential in *Webster's Third New International Dictionary*,[1] which defines recredential as follows: "a letter of appreciation given to a diplomatic envoy on his permanently leaving a post by the head of the state to which he has been accredited."

Nevertheless, notwithstanding this definition, Governor Mario Cuomo appointed the New York State Advisory Committee on Physician Recredentialing, which published its first report entitled *Phase 1: General Principles, Proposed Process, Recommendations* in January 1988.[2] This report proposed a nine-year recredentialing cycle with various methods for three classes of physicians—the physician with hospital privileges, the physician with an office-based practice, and physicians not in active practice. This report was certainly not enthusiastically embraced by the medical profession, and organizations representing hospitals and even the Department of Education had serious concerns with it.

In response to the report of the New York State Advisory Committee on Physician Recredentialing, the Medical Society of the State of New York (MSSNY) adopted a position paper on the issue.[3] In this position paper, among many of MSSNY's concerns, the following is perhaps most important.

> The MSSNY agrees with the statement, made on several occasions in the report, that the recredentialing process broadly outlined in the report "is not designed to measure medical competence . . . ."
>
> We agree with the report that there does not exist a single methodology for measuring competency and agree that employment of a combination of methodologies to measure competency would be logistically and economically unrealistic. The evaluation of competence in the health professions has not yet reached maturity. Measurement of changes in practice as a consequence of additional education, assessment of the validity of examinations and the determination of goals for competence are all necessary parts of the ongoing development of competence evaluation.[3]

It is generally agreed that competency is a combination of an individual's cognitive knowledge, clinical skills, practice patterns, and those personal factors such as judgment, lack of impairment, experience, interpersonal relationships, etc. Most agree that at present there is no methodology for collectively evaluating these components of competency. Described below are two mechanisms which should satisfy all concerned. However, before proceeding, I would suggest that the most basic index of a physician's competency is patient outcome or quality of care which must be the foundation of any evaluation process.

It is now four years later and it is my view that the notion of recredentialing should be reevaluated. It has been two years since the committee released its Phase 1 report. However, the enormous complexity of the project and the cost seem to have blunted the committee's zeal. More hopefully, the committee is shifting its focus to methods of identifying the incompetent and impaired physician rather than attempting to recredential all physicians, of which the vast majority are probably competent, with a very favorable patient outcome and quality assurance record.

To date there are little or no data to support the contention that there are many clinically incompetent physicians. For example, those physicians determined to be guilty of clinical misconduct (negligence and incompetence) by the Office of Professional Medical Conduct (OPMC) represent a very small fraction of their referrals: 35 of 3,934 completed cases.[4]

When one considers the many systems in place to evaluate the competency of a hospital-based physician, it is unlikely that incompetent ones would go undetected. The list of such mechanisms is impressive and includes physician credentialing regulations and mandatory reporting of adverse events, implementing the Medical Malpractice

Comprehensive Reform Act[5]; Health Care Financing Administration (HCFA) Peer Review Organizations (PROs)[6]; the New York State Department of Health comprehensive survey[7]; the New York State Insurance Department Physicians and Surgeons' Professional Liability Merit Rating Plan[8]; and the OPMC.[9] That physicians practice excellent medicine in hospitals is supported by the recently released Harvard study which determined that only 0.3% of patients had an adverse outcome of extended duration.[10] Do these physicians need recredentialing? I think not.

There is a need to develop a mechanism to identify the incompetent and impaired office-based physician; more importantly, to identify the office-based physician, who has no significant peer contact. There are two mechanisms for consideration.

The first is a proposal set forth by the Federation of State Medical Boards. "The Federation of State Medical Boards of the United States believes it is essential that the population of licensed physicians be systematically assessed at appropriate intervals to identify those whose qualifications and/or fitness for continued licensure are open to reasonable question."[11] The federation established an ad hoc committee to study physician reevaluation for registration; its report was adopted by the federation in 1983. This is an important and carefully thought out document, which is divided into two parts; the first part is the five principles of assessment which are fundamental to any system of reevaluation for registration.

Based on its principles of assessment, the second part of the federation proposal was adopted in its 1985 edition of *A Guide to the Essentials of a Modern Medical Practice Act*.[12] This mechanism would require a physician at the time of reregistration to complete and/or report on a number of informational items which would serve as indicators of competence (or incompetence). The information items required include among others any action taken against the licensee by credentialing or regulatory agencies. It considers adverse judgments or settlements, voluntary surrender of license, etc. The system also would require that the physician sign the registration and would be subject to disciplinary action should the report not be correct.

An essential requirement for this mechanism is that an effective system for reviewing reregistration forms be developed. This proposal would be strengthened by information obtained from the National Practitioner Data Bank mandated under the Health Care Quality Improvement Act of 1986.[13] This method of reevaluating a physician at the time of reregistration could be readily adopted in New York State. It would not be expensive and would give a much better profile of the physician's activity than other proposals which would only identify an individual component of competency.

The second mechanism is in place, well-tested, and effective. The Health Disciplines Act of Ontario, Canada, sets out a framework within which the College of Physicians and Surgeons of Ontario licenses physicians and regulates the practice of medicine in the province. Since 1981 the College has had a peer assessment program, which in my opinion is the most fair and comprehensive program to date.[14] Under its peer assessment program, all licensed physicians are randomly selected by computer for peer assessment. Based on the college's early experience, the randomization is now skewed to older physicians and those without peer contact. This program involves an in-office review of the physician's practice, including chart review and patient outcome. In most instances, the physician is given an opportunity to correct identified deficiencies. If the physician cannot change his practice to conform with the peer assessment findings, appropriate disciplinary action is instituted. This program has been well received by physicians in Ontario and aside from initial start-up costs, it is not expensive. As a result of this peer assessment program, a program called "Physician Enhancement Program" for Ontario has been developed. This project has been instituted jointly by the Ontario Medical Association, the Council of Ontario Faculties of Medicine, and the College of Physicians and Surgeons of Ontario, to implement focused/enhanced or remedial medical education programs.

The two mechanisms described above should be sufficient to identify the incompetent physician. The former would satisfy the demands of reevaluation at time of reregistration; the latter would be an ongoing assessment program under which all physicians would be liable.

In conclusion, I would suggest that the New York State Department of Health's ill-conceived notion of recredentialing be abandoned in favor of a mechanism for reevaluation for registration as proposed by the Federation of State Medical Boards and that a peer assessment program similar to that in Ontario be developed to assess physician competency in a fair and comprehensive manner.

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Rural obstetric care and the family physician

Any discussion of the quality of medical care in the United States invariably includes mention of perinatal or infant mortality rates. The poor showing of the United States compared to other developed countries in these crude indicators of perinatal health belies the number of dollars per capita spent on health care in our nation.[1] This paradox has sparked much discussion, research, and controversy regarding the system (or lack of system) of obstetric and neonatal care in the United States.

One must look beyond the overall perinatal mortality rate to adequately assess the quality of perinatal care in a given system. Sweden and other nations with low perinatal and infant mortality rates tend to have healthier childbearing populations when compared to the United States. This results in fewer low birth weight infants and lower overall mortality. A more accurate view of the efficiency of health care after infants are delivered lies in the measurement of birth-weight-specific perinatal and infant mortality.[2] In these areas the technology of the United States' well-equipped neonatal intensive care units, and our ability to sustain low birth weight infants, places us in a more favorable light compared to other health care systems.

While acknowledging the importance of efficient regionalized perinatal care systems supported by centers with effective neonatal intensive care, it is clear that further improvement in salvaging low birth weight and other infants at risk is a high cost option with relatively little added benefit in most regions. Improving maternal health and decreasing the number of low birth weight infants through improved access to prenatal care, health promotion and education efforts, accurate risk assessment, and efficient regionalized systems of care hold much more promise for improved perinatal health in our country. Targeting these preventive efforts to high risk segments of the population, particularly in poor rural and inner city areas where health care availability and access are often deficient, is the first step toward improved perinatal care.[3]

To ensure improved overall perinatal morbidity and mortality rates, however, we must also maintain excellent care for relatively healthy, low risk populations. In regions such as the one in western New York described in this issue of the *Journal*, a healthy childbearing population served by well-staffed regionalized care systems results in fewer low birth weight infants, and neonatal mortality rates below national averages.[4] The article by Rosenthal et al[4] in this issue is a retrospective, descriptive case series of 297 pregnant women who sought initial prenatal care from one of 12 family physicians in western New York. The overall health of the maternal population in this series is evidenced by the fact that there were only seven low birth weight infants out of 292 live births (2.4%), well below the national average in 1986 of 6.8%.[3] All seven of these infants were identified during prenatal care to be at risk for low birth weight and six of the mothers were appropriately transferred to the regional perinatal center prior to delivery. The three infants who were born weighing less than 1,000 grams all survived.

This case series enriches the literature of successful regionalized perinatal care systems that serve predominantly rural, relatively healthy, low risk populations.[5–7] While these small descriptive studies do not prove "safety" in comparison to other care systems, they do describe family physicians who work in concert with obstetrician specialists and regional perinatal centers to identify mothers and fetuses at risk, intervene appropriately, and show excellent outcomes. Larger studies of obstetric care in small hospitals have suggested that for low risk populations, birth-weight-specific mortality for infants over 2,500 grams may actually be lower in small hospitals compared to larger urban centers.[8]

During the next decade, while public health officials target underserved populations in inner city and certain rural areas to improve high perinatal mortality rates, what changes might be anticipated in regions such as western New York State? Some rural areas may improve care further by organizing more efficient regionalized systems and protocols. In many regions where such networks are already in place, however, the number of family physicians and obstetrician specialists willing to offer obstetric care is rapidly decreasing. The scenario has been played out in hundreds of small towns in recent years: The local group of family physicians decides that they no longer want to provide prenatal and intrapartum care. Consequently, the local small rural hospital no longer has an obstetrics unit. The closest practitioner is an obstetrician located in another town 30–50 miles away. The majority of women who would have enjoyed continuing care from their physician a few miles down the road during pregnancy now must drive a significant distance for each prenatal visit. Problems of access to care become obvious. One need only envision the same long drive on a snow-covered rural highway while a woman is in active labor to imagine a few of the new risks that might be added.

It is no secret that significant numbers of both family physicians and obstetricians are giving up obstetric care. This is especially true in rural and inner city areas. A committee from the Institute of Medicine (IOM) recently reviewed 21 state surveys and five national studies which focused on the effect of increasing liability insurance costs and litigation rates on physicians' decisions to discontinue obstetric care.[9] The committee estimated that the number of obstetric providers in nonmetropolitan areas in the United States has decreased by 20% in the past five years. This trend has been especially prominent among family physicians. In Pennsylvania, fewer than 9% of family doctors now deliver babies.[10] A study of Ohio physicians showed that while 54% delivered babies in 1975, only 16% planned to continue obstetric care into 1989.[11] Most surveys have shown that increasing liability insurance costs and fear of litigation have been major determinants in this trend. However, life style issues and the increasing complexity of a generalist's practice also play major roles in the decision to discontinue obstetric practice.[12]

Well-trained family physicians must continue to provide accessible obstetric care if we are to maintain the high level of perinatal health that exists today in many rural areas. Unfortunately this important team member is on the verge of extinction in many sections of the country. Only with changes in the current medical liability environment and new directions in family practice education and research will family physicians continue to play a significant role in responding to the present and future obstetric care needs in our country.

First, but not necessarily most important, the burden of liability risk and the costs of liability insurance for obstetric providers must be decreased. Obstetrics is an area of practice in which, under the current tort system, liability for adverse outcomes is at least as prominent as that incurred by clearly negligent practice. The dramatic rise in the number and severity of claims against obstetrical providers in recent years has not always been a linear increase. During the past two decades there have been at least two cycles, each lasting two to three years, when an explosive increase in the number of medical malpractice claims has been followed by a shorter "leveling off" of litigious activity.[13] Figures show that we may be currently experiencing a slowing or leveling of malpractice claims. At least one report from Connecticut attributes, in part, a recent increase in the number of family physicians practicing obstetrics in that state to a seemingly stabilized liability environment.[14] The fact remains that the costs of medical liability insurance for obstetric providers continues to rise astronomically and a new crisis may be just around the corner.

Many options to help decrease the burden of liability risk and the cost of malpractice insurance are currently being debated in the literature. The most comprehensive report on this subject to date from the Institute of Medicine focuses on alternative methods to resolving medical malpractice claims instead of reforms of the current tort system.[9] Alternatives include the American Medical Association's fault-based administrative system, a variety of no-fault schemes with designated compensable events, and legislation authorizing the use of private contracts between providers and patients to govern professional liability. The IOM report also calls for the federal government to support demonstration projects and finance pilot programs testing various solutions. In addition to these long term possibilities, immediate measures to help correct current deficiencies in obstetric care of the poor in inner city and impoverished rural areas by offering immunities to obstetric providers in government financed communities and migrant health centers have been suggested.

There must also be a restructuring within the family practice educational system if obstetrics is to be maintained as part of family practice. As family physicians drop out of obstetrics there are fewer role models who demonstrate tenable options for family practice obstetrics to residents in training. Every year fewer graduating family practice residents choose to include obstetrics in their practice.[15,16] Medical students who have an interest in obstetrics and see no concerted efforts to reverse this trend are drawn away from family practice training. Without a national effort to strengthen and reorganize residency education in this area, our descent down this slippery slope will simply become faster.

Zervanos[10] and others have proposed that residents in family practice training be given the option of attaining one of three levels of competency in obstetrics. Every resident would be expected to attain level I skills which includes a basic understanding of prenatal care and labor and delivery. Residents who decide to practice obstetrics after graduation would need to attain at least level II competencies requiring up to six months of training during a three-year residency. For those wishing to practice high risk obstetrics and include procedures such as cesarean sections, an additional six months to one year's specific training in obstetrics would be necessary for such level III competencies.

All family practice residencies are currently required to provide level I training as described in this education scheme. The availability and quality of level II training varies greatly among residency programs. Residents often find it necessary to supplement their experience by travelling to a large county hospital with a busy obstetrics service, usually run by obstetrician specialists, where a "high risk clinic" atmosphere is the rule. Viable alternatives for educating residents must be developed if family physicians are to continue to provide the kind of "family-centered" obstetric care that is desirable to a majority of the population. A concerted effort from national family practice organizations would be needed to organize and develop regional training centers, staffed primarily by family physicians, that would provide level II and III training for family practice residents. Residents interested in practicing obstetrics would come from residencies throughout the region to receive training from family physicians who demonstrate a workable paradigm of including obstetrics in family practice, working in concert with obstetrician specialists, midwives, and other practitioners. These centers would also promote the concepts of family practice and family centered obstetrics to medical students by developing elective rotations and summer preceptorships.

A regionalized approach to level II and III education would enhance training for residents who want to include obstetrics in their practice by gathering the best educators in techniques of family centered obstetric care and providing strong family physician role models for residents and students. A number of studies have suggested that these role models are important if residents are to choose to include obstetrics in their practice.[17,18] These training centers would best allow specific training in order to respond to obstetric care needs for a particular area of the country. For example, a regional center in South Carolina training residents to practice in poor rural areas of greatest need would have a different educational emphasis than a center in inner city Chicago.

A regionalized family centered educational system would lend itself well to new directions in primary care obstetrics research. Past attention in family practice research has often focused on comparing the quality of obstetric care by family physicians with that offered by obstetricians. These attempts to ensure that specialty training is not a risk factor for poor outcomes in pregnancy has at times implied that family doctors wish to validate themselves as "mini-obstetricians." Instead, as suggested by Rosenblatt,[19] we must continue to redefine the family physician's role as a primary care obstetric provider in response to the changing needs of the population and increasing technical capabilities. A research agenda focused on the family physician as primary obstetric provider might concentrate on improving prenatal risk assessment tools, the appropriate application of new technology to low risk populations, patient education and health promotion efforts during prenatal care, and techniques of family centered care.

A recent discussion of obstetric care in a rural Utah county illustrates the potential impact of family physicians on rural obstetric care.[20] Grand County, Utah, is an isolated area in the southeastern corner of Utah which suffered a neonatal mortality rate twice that of the rest of the state from 1971 through 1980. Primarily through the efforts of a residency trained family physician who began practice in the area in 1980 and initiated community oriented efforts to improve obstetric care, neonatal mortality in Grand County was below the state average in 1984 and 1985. Well-trained family physicians can be valuable team members in providing obstetric care in rural areas, whether it be in Grand County, Utah, or in rural New York State as described by Rosenthal[4] in this issue of the *Journal*. Access to obstetric care is diminishing in many rural areas as more and more family physicians discontinue the practice of obstetrics. With decreasing access to care, decreased quality of care and more adverse outcomes are sure to follow.

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Adenocarcinoma of the cervix

In this issue of the *Journal*, Moll et al[1] present the first case of a primary signet ring adenocarcinoma of the cervix. The signet ring cell pattern, as the authors discuss in their manuscript, is highly suggestive of metastatic lesion from the gastrointestinal (GI) tract, classically from the stomach. In their reported patient, there was no other primary source for this malignancy, supporting the cervix as the primary site. Unfortunately, the patient's family refused autopsy, highlighting a continuing problem. More and more families are refusing autopsy when their loved ones die. While this is understandable, it compromises a case study such as this where a negative autopsy examination of the GI tract would be most convincing. Notwithstanding, the authors did make a diligent search preoperatively, intraoperatively, and postoperatively for a GI source. One must conclude that the GI tract would probably have been negative had an autopsy been performed.

Adenocarcinoma of the cervix is a less frequent cancer of the cervix, classically accounting for approximately 5% of all cervical cancers.[2] In recent years there has been an apparent increase in the percentage distribution of adenocarcinoma of the cervix, with this type representing anywhere from 10% to 20% of the total.[2] It is unclear at this point whether this represents an absolute increase in the number of adenocarcinomas or a decline in the number of invasive squamous cell carcinomas of the cervix due to earlier diagnosis in the premalignant state. Nevertheless, it is the impression of many gynecologic oncologists that they are, in fact, seeing more adenocarcinoma of the cervix.

A number of subtypes of adenocarcinoma have been described. The most common subtype is endocervical or endometrioid. Probably the most publicized type has been the clear cell adenocarcinoma, which can occur either in the cervix or in the vagina. Other types include papillary, adenosquamous, and mucoid. While these cell types are interesting from a pathologic perspective they do not influence the prognosis.

The case reported by Moll et al[1] highlights three pertinent problems in patients with adenocarcinoma of the cervix. The first problem with adenocarcinomas is the difficulty in diagnosis. An adenocarcinoma usually arises high in the endocervical canal, thus escaping detection by routine cytologic screening. These patients may have a negative pap smear rate as high as 40%.[3] Many of these patients will present with bleeding, a normal appearing cervix and a normal pap smear. Not surprisingly a diagnosis of dysfunctional bleeding is often made. The examining physician should bear in mind that any woman with unexplained bleeding may have a high endocervical lesion that can escape detection. Additionally, these lesions can escape detection if a dilation and curettage (D&C) is performed without a fractional curettage. With the new International Federation of Gynecology and Obstetrics (FIGO) surgical staging for endometrial cancer, the fractional D&C is no longer required for staging.[4] The clinician should remember, however, that the occasional adenocarcinoma of the cervix will be diagnosed by endocervical curettage, and a routine endometrial sampling may miss the endocervical lesion.

The second problem is the status of the ovaries at the time of radical hysterectomy. In the patient reported by Moll et al,[1] there was metastatic disease to the ovary. Additionally, there was gross intra-abdominal disease. Some authors have suggested that all patients undergoing radical hysterectomy for adenocarcinoma of the cervix have their ovaries removed due to the chance of metastatic disease. While case reports have been published demonstrating metastatic disease to the ovaries, this does appear to be a rare occurrence.[5] In a large series from the University of Michigan, patients who underwent radical hysterectomy with ovarian preservation actually did better than those whose ovaries were removed.[6] In reviewing the literature, the incidence of ovarian metastases appears to be very low. Many of the patients reported with metastatic disease to the ovaries had other evidence for metastatic spread. The chance of spread to the ovaries does, however, appear to be greater than that with squamous cell disease where the chance is remote. Given the low possibility of metastatic spread to the ovary, I currently recommend that the ovaries in young women undergoing radical hysterectomy for adenocarcinoma of the cervix be preserved when there is no evidence of metastatic spread elsewhere. Obviously, these ovaries need careful inspection and should be removed if gross abnormalities are present.

The third problem highlighted by the patient described by Moll et al,[1] is the significance of the spread of metastatic disease to lymph nodes. In both squamous cell cancer of the cervix and adenocarcinoma of the cervix the involvement of regional lymph nodes significantly alters prognosis. In Stage I squamous cell disease, when regional nodes are involved, survival will drop from approximately 90% to 60%. The adenocarcinomas appear to behave differently, however, when regional nodes are involved as survival drops to the 20% range.[6] Unfortunately, there is no adjuvant therapy which can improve survival once metastatic disease is present. Until such adjuvant therapy is reported, the involvement of lymph nodes will carry an ominous prognosis.

In summary, adenocarcinomas of the cervix are being seen with an increasing frequency and portend a poorer prognosis than squamous cell cancer. The signet ring cell type of adenocarcinoma is a new subtype and appears to occur in the cervix as a primary neoplasm.

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Primary care obstetrics in rural Western New York

A multi-center case review

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ABSTRACT. Obstetric care in rural communities is in crisis due to the shortage of practitioners and the pressures of regulatory agencies on small obstetric units. We reviewed the records of 297 pregnant women presenting to the offices of 12 family physicians in rural areas of Western New York State. Local deliveries were done in rural hospitals that reported fewer than 500 deliveries per year. Family physicians obtained consultation in 36% of the pregnancies. Of these, care responsibility was transferred to an obstetrician 50% of the time (18% of all pregnancies). Two percent of the cases were transferred to a regional referral center. There was one perinatal death and one intrauterine fetal death in this series. Of the 222 deliveries by family physicians, oxytocin was used in 23%, narcotic analgesia was used in 15.3%, and outlet assist in 8.1%. Labors were somewhat longer than standard labor curves, and the overall cesarean section rate was 13.5%. We conclude that obstetric care by rural family physicians utilizing small rural Western New York hospitals is consistent with standards of care elsewhere when practiced in the context of an organized referral network.

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In 1980, 56% of family physicians had hospital privileges to practice obstetrics.[1] By 1988, only 28% of family physicians were doing deliveries.[2] The family physician's tradition of watchful and restrained interference may result in less operative intervention, longer labors, but safe outcomes in low risk pregnancies.[3–5] Despite these reputable outcomes, attrition from obstetrics continues.[6] Concerns about litigation, malpractice premiums, government regulations, the perception of incomplete training, and the time commitment for obstetrics are thought to be responsible.[7,8]

Nowhere is the impact of this attrition more likely to be felt than in rural communities, where as many as 60% of family physicians have obstetric privileges and may provide much of the obstetric care. Regionalization of obstetric services has improved care of the high risk patient, but has done little for the low risk pregnancy in spite of increased costs.[9,10] Studies have suggested that small rural units (100–500 deliveries per year) have equivalent or lower birth weight-specific perinatal mortality, a lower operative rate, and lower cost in all but the lowest birth weight categories when compared with referral hospitals.[11,12] Obstetric care is the most common discharge diagnosis in New York State and is obviously important to the financial status of rural hospitals.[13] Also, local obstetric care enhances accessibility, which may be the most important factor in reducing infant mortality.

It was our hypothesis that obstetric care in Western New York's regionalized perinatal care network was safe for patients who entered the system through the offices of rural family physicians using standard risk factor assessments, consultations, and referrals. The system components are tertiary referral centers for high risk cases, local obstetricians for moderate risk cases, and a local rural hospital for low risk deliveries. This information is essential for family practice training programs, health care regulators, and patients residing in rural communities.

METHODS

The records of all obstetric patients with expected dates of confinement from March 1, 1987, to March 1, 1988, that presented to the offices of 12 family physicians in rural Western New York State were retrospectively reviewed. The physicians were board certified in family practice, and all but one had residency training. These physicians were selected because they were within a 90-minute drive of the university and had been practicing for more than three years. Physician ages ranged from 31 to 66 years. The practice sites and the hospitals were located 40 to 60 miles from a secondary or tertiary referral center. The family physicians used three different rural hospitals with 30, 46, and 97 total beds and 208, 284, and 481 deliveries annually. The larger hospital had three board eligible obstetricians on staff residing in the community. The two smaller hospitals utilized obstetric consultants who lived in other communities 20 to 30 minutes away, with whom they had a defined coverage relationship. All three hospitals were the only hospitals in their communities. All these physicians used the American College of Obstetrics and Gynecology prenatal forms in original or revised format, and all physicians referred high risk patients for consultation. The decision to deliver locally or in a referral center was made with the consultant on an individual basis.

Every pregnancy that presented to these practices was reviewed from the first visit to conclusion. Two of the practices kept a log of all pregnancies in their practices from point of entry. Point of entry may have been a positive pregnancy test or first obstetric visit, whichever was earliest. A chart audit was necessary to obtain these cases in the third practice, and this may have resulted in underreporting of early pregnancy events. Utilizing a chart review instrument devised for this study, data were collected on every case, whether it terminated in abortion, referral and delivery, or local delivery.

The data were manually analyzed. Data in three referred cases were incomplete due to inability to obtain complete records from

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the referral hospital. One case was lost to follow-up and was not included in the data.

RESULTS

A total of 297 pregnancies were followed to conclusion. All but two women were white. One practice cared for an Amish community. Spontaneous abortions before 16 weeks of gestation accounted for eight (2.7%) pregnancies. One of these was a twin pregnancy documented by ultrasonography that aborted at 16 weeks. Five of these patients underwent curettage for persistent bleeding or incomplete abortion. No complications were reported in this group.

A referral (transfer of care responsibility) to a local obstetrician was made in 16% (48 of 297) of the pregnancies, and a consultation without transfer of care was obtained in another 18% (54 of 297 cases). The patients' care was transferred to a regional perinatal center in 2% (6 of 297) of cases for an overall consult/transfer rate of 36%. Another 13 (4.4%) were home deliveries in the Amish community by Amish midwives. A total of 222 (80%) pregnancies (excluding miscarriages and home deliveries) were delivered by the family physician. Primiparous patients accounted for 38% (113) of the pregnancies and 62% (184) were multiparous.

The average number of prenatal visits was 12.9 (standard deviation (SD) = ±4.5) excluding Amish home delivered pregnancies. Review of the records of high visit cases indicated that some portion of the extra visits included upper respiratory complaints, gastroenteritis, or acute injuries. Generally, a pregnancy evaluation was completed at each visit for nonpregnancy complaints. Payor source was as follows: private insurance, 57%; public assistance, 21%; and self-pay, 14%. There was no difference in the average number of prenatal visits according to payor type.

Oxytocin Use: Non-Referred Patients. The 222 vaginal deliveries by the family physicians were performed at the respective local rural hospital. Labor was spontaneous in 75% (165), induced in 7% (17), and augmented in 18% (40). Therefore, oxytocin was used in 57 (25%) labors. Obstetric consultation was obtained prior to the use of oxytocin in all cases, though often this was by telephone. Inductions were performed for post-date (53%, N = 9), premature rupture of membranes (29%, N = 5), and pre-eclampsia/hypertension (18%, N = 3). Augmentation was performed for prolonged labor or inadequate contractions. There were no Apgar scores less than 7 in the group receiving oxytocin.

Labor Characteristics: Non-Referred Patients. Average length of labor was 13 hours, 19 minutes (SD = ± 417 minutes) for primiparous women and 10 hr, 38 min (SD = ± 375 min) for multiparous women. The second stage of labor averaged 79 min (SD = ± 154 min) and 23 min (SD = ± 26 min) for primiparas and multiparas, respectively. Labor was defined as the time when contractions were reported to occur every five minutes until delivery. A fetal monitor was used in 99% of the family physicians' patients, although in many instances it was used for only specific intervals during labor. The frequency of abnormal monitor patterns was 9%. Abnormal monitor patterns were associated with a one-minute Apgar score less than 7 in two cases, but there were no five-minute Apgar scores less than 7. Abnormal monitor patterns accounted for 6% (3) of the local referrals.

Narcotic analgesia was used during 15.3% (34) of the labors. Low-outlet forceps were used in 4.5% (10), and vacuum assist in 3.6% (8) of deliveries by family physicians (total = 8.1%). No significant complications were recorded for either procedure. Conservative use of outlet assist may have contributed to somewhat longer second stages of labor in the pregnancies in this study. Episiotomies were performed in 59% (131) of the deliveries. Third degree extension of the episiotomy occurred in 9% (12) of these, and there were two fourth degree extensions. Of the patients without an episiotomy, 45% had at least a second degree laceration. There was one third degree and no fourth degree lacerations in the group delivered without episiotomy.

Outcomes: Non-Referred Patients. There was one intrauterine fetal death at 31 weeks' gestation. Labor was induced for this patient because she failed to go into spontaneous labor after death was confirmed. The macerated fetus weighed 299 grams.

One perinatal death occurred. The mother was a 30-year-old woman who had had 17 perinatal visits and had no identified risk factors. She was primiparous. She presented in spontaneous labor at 42 weeks' gestation and delivered after 13 hours of spontaneous labor. The monitor pattern was normal, but late second stage labor was not monitored continuously in the delivery room. Second stage lasted two hours, was difficult, and vacuum assist was used. The child weighed 8 lb 6 oz. There were no spontaneous respirations and no heart rhythm at birth. Resuscitation efforts continued for 45 minutes but were unsuccessful in obtaining other than a transient heart rate. An autopsy was refused. The delivering physician noted no physical abnormalities and considered an undiagnosed congenital abnormality as the cause of death; however, intracranial hemorrhage, premature placenta separation, and other causes could not be ruled out.

Another woman spontaneously delivered a 2,211-gram infant at term (Apgar 9/10). The mother had had ten prenatal visits, and no risk factors were identified except second pregnancy in less than 12 months. Intrauterine growth retardation was suspected prior to labor, but due to transportation problems a referral was not made. The child did well and was discharged with the mother.

Three infants weighing more than 4,500 grams were delivered without consult or referral. One required outlet assist. All mothers had been screened for diabetes during the pregnancy. All of these children did well in the nursery.

Only two infants delivered by the family physicians had five-minute Apgar scores of less than 7. The one perinatal death mentioned earlier had a recorded Apgar of 0/0. Another term child had variable decelerations on monitor and a tight nuchal cord twice coiled. The child's Apgar scores were 4 at one minute, 6 at five minutes, and 9 at ten minutes.

Birth weight for the 222 non-referred patients delivered by family physicians in rural hospitals averaged 3,505 grams (SD = ± 557 g; range, 2,211–5,188 g). With the exceptions noted in this section, all were delivered at between 38 and 42 weeks' gestation.

Locally Referred Pregnancies. Forty-eight pregnancies (16% of all pregnancies) were referred to local obstetricians, with deliveries occurring at a rural hospital. These cases were often team managed by both the family physician and obstetrician after referral. Indications for these referrals were previous cesarean section (23%), abnormal presentation (21%), abnormal labor pattern (17%), pre-eclampsia (10%), twins (6%), post dates (8%), abnormal monitor pattern (6%), and preterm (6%). Method of delivery was either cesarean section (73%) or vaginal birth (27%). There were no children with five-minute Apgar scores less than 7. There were only two neonatal problems. One neonate had transient tachypnea of the newborn, and one had hyaline membrane disease and was transferred to the tertiary care center. There were no neonatal deaths. Birth weights for locally referred babies averaged 3,335 g (SD = ± 921 g; range, 2,098–4,876 g).

Tertiary Care Center Transfers. Six patients (2% of all pregnancies) were transferred to tertiary care centers. One was at the request of the woman, who had had a previous cesarean section and preferred delivery at a major center. One transfer was for suspected intrauterine growth retardation, and the infant was delivered at 31 weeks' gestation due to decreased fetal activity. Another involved a twin pregnancy, with transfer due to premature labor at 33 weeks; the twins were delivered by cesarean section in the tertiary center and discharged in good health. One 17-year-old mother, after a failed induction at 42 weeks, was transferred for cesarean section at a tertiary center due to meconium stained amniotic fluid. One woman, whose pregnancy was complicated by hypertension, was transferred and delivered at term by cesarean section due to suspected intrauterine growth retardation. The only woman transferred who underwent vaginal delivery was an insulin-dependent diabetic who was followed and delivered at term in the tertiary center. The three premature neonates weighed less than 1,000 grams. The average birth weight of the other four neonates was 3,714 g (range, 2,430–4,876 g).

Cesarean Sections. Of the total 289 deliveries, 39 were by cesarean section, for an overall rate of 13.5%. Five of these were performed at the tertiary referral center. The cesarean section rates for study pregnancies at the three rural hospitals were 23%, 13%, and 9%. The highest rate was in the community with the most available obstetric specialists. Repeat cesarean sections accounted for 13 (33%) of these procedures. Other indications were failure to progress in labor (26%), abnormal presentation (23%), twins (5%), abnormal monitor patterns (5%), and prematurity (5%). At the time of this study, none of the study physicians were allowing trial labors after previous cesarean delivery.

Home Deliveries. One group of physicians cared for a large Amish community, and while they discouraged home deliveries, they did provide prenatal care for a number of women who later chose to deliver at home with the assistance of an Amish midwife. There were 13 women who delivered at home in this community. The average number of prenatal visits was 2.5, and none of the visits occurred before 30 weeks' gestation. All deliveries were at term, and all the babies did well, with an average birth weight of 3,662 g (range, 3,178–4,142 g). Birth weight was not recorded for five babies.

Perinatal Complications: Rural Deliveries. As reported, there was one fetal death at 31 weeks, one perinatal death at term, and no deaths in the referred pregnancies. There was only one other child with a tight nuchal cord twice coiled that had a five-minute Apgar of less than 7 but that did well in the nursery. There were 17 other neonatal complications that required intervention other than routine nursery care: elevated bilirubin (4), transient tachypnea of the newborn (3), elevated hematocrit (2), hip dislocation (2), fever (1), hydrocele (1), patent ductus arteriosus (1), Erbs palsy (1), scalp laceration (1), and undescended testicle (1). Five babies were transferred to tertiary centers with the following: transient tachypnea (1), hyaline membrane disease (1), elevated hematocrit (2), and elevated bilirubin (1). All of these children recovered.

Maternal Complications. There were five deliveries that were considered precipitous (less than three hours of labor), but none of these women were reported to suffer untoward consequences. There were three postpartum fevers plus one retained placenta that required curettage, and one postpartum hemorrhage that required transfusion. Of a total of five twin pregnancies, two sets of twins were delivered vaginally, and one mother was transferred at 33 weeks' gestation for premature labor and subsequent cesarean operation. No cases of placenta previa or abruptio placenta were reported in this series.

DISCUSSION

It is difficult to determine if one type of care is safe as compared to another in medicine. The large series required for this assessment often lack the homogeneity required for comparison. Care providers who follow established guidelines for process and achieve similar outcomes can be assumed to provide care that meets a standard considered to be safe. By reviewing every confirmed pregnancy to conclusion we have a unique description of the regional perinatal system when the entry is through the rural family physician. The review is descriptive, and the sample is too small to evaluate all outcome factors, but it can be used to examine process.

Table I compares the results of this study with data from several other sources. It may be concluded from the comparison that the rural family physicians in this study follow a process similar to that of other physicians doing obstetrics and obtain outcomes that are similar. Table I demonstrates a reasonable and conservative use of interventions by the study group. The frequency of office visits reflects the concept of continuity of care, with pregnant patients presenting to the same physicians for most of their health problems during pregnancy. In spite of this added attention, it would appear that one case of intrauterine growth retardation (birth weight <2,500 g) was not diagnosed until delivery.

TABLE I. Comparison of Select Study Findings with Previously Published Data

	Study	Literature*
Prenatal visits	12.9	8.1–10.4[15]
Referral/consult rate	36% (108)	25–30%[20]
Oxytocin use	25% (57)	10.7–46.8%[12,15,17]
Length of labor		
primipara	13 hr 19 min	10 hr 6 min[14]
multipara	10 hr 38 min	6 hr 12 min[14]
Analgesia use	15.3% (34)	17–30%[16]
Outlet assist	8.1% (18)	8–11%[16]
Episiotomies	59% (131)	48–84%[5,12]
Cesarean delivery	13.5% (39)	24%[18]

Length of labor exceeded the standard Friedman curves.[14] Other studies have indicated that women delivered by primary care physicians do have longer labors.[3,15,16] This may be the effect of a conservative approach to labor and the price paid for a lower cesarean section rate, and decreased use of oxytocin and outlet assist.[12,15,17] Whether the increased prenatal visits enhances care cannot be proven, but the relationship developed during these frequent visits may enhance the mutual trust needed to manage a long labor.

The overall cesarean delivery rate, including transferred patients, was 13.5%, compared to a rate of 24% for all of New York State in 1986.[18] The regional rate for Western New York rural hospitals with fewer than 500 deliveries a year was 23.2%.[18] The lower rate in this study may reflect a combination of several factors: Some women with previous cesarean delivery may not present to a family physician because of an established or anticipated need for an obstetrician; a patient cared for by a family physician may be given more opportunity to labor before primary cesarean operation is chosen; or the inherent presence of a second physician when a family physician consults with an obstetrician may make both less likely to choose cesarean delivery as an option. It is interesting that the highest cesarean section rate occurred in the hospital with the most available obstetric specialists. This observation most likely confirms that interventions are more strongly correlated with physician training and practice style than with indications.[19] It may also reflect a different threshold for obstetrical consultation when this service is readily available, or increased confidence in and readiness of a surgical team.

The family physicians delivered 80% of all pregnancies that did not end in abortion or home delivery. Physicians referred patients who required cesarean delivery, but continued to manage patients when oxytocin was used even though an obstetrician was always consulted. Including oxytocin use, the overall consultation/referral rate was 36%. This exceeds the observations in the British health system that 25–30% of women will require an obstetrical specialist during pregnancy or labor.[20] By utilizing a standard risk prediction scale, the large group of patients for whom pregnancy outcome is likely to be good can be separated from a high risk group, who are also likely to have good outcomes, but for whom adverse events are more likely to occur.[21]

This study and others suggest that high risk deliveries are, for the most part, being successfully identified and transferred for appropriate tertiary care. The one perinatal death in this series did occur at a rural site. Mortality in the first 24 hours of life in Western New York in 1986 was 4/1,000 live births.[18] Lack of autopsy limits the ability to comment extensively, but this case does dramatically demonstrate that the system is not perfect, though it seems unlikely that the outcome would have been altered with a different delivery site. One may consider that this traumatic event occurred in a supportive environment accessible to extended family and friends. The lack of risk factors would seem to make prediction of this death impossible. The resuscitation efforts were reviewed by the medical staff and were determined to be well directed. This case highlights the necessity for all birthing units to be prepared for resuscitation and stabilization of the newborn. Pregnancies without identifiable risk factors do not mean the absence of risk.[21] All pregnancies require attentiveness from an accessible health care provider, and all deliveries should have the advantage of a watchful, experienced team.

The rural hospital served as the birthing center for 98% of the pregnancies. Infants with neonatal complications occurring after delivery were successfully and routinely transferred to tertiary care centers where needed. The need to transfer did not seem to have an adverse effect on the outcome in these selected pregnancies. Maternal complications did not present unusual or untoward problems in the rural hospital. Cesarean delivery rates were well below state averages.

Obstetrical care has undergone a profound transformation since the 1950s. Once thought to be a natural life event, birth has become a medical condition.[22] This approach is appropriate for the high risk pregnancy but may lead to over-use of technology when applied to the routine low risk pregnancy. This trend has become particularly important to communities that must define a rural hospital's role in health care. It is evident that family physicians should not strive to be "mini-obstetricians," and that small rural hospitals should not strive to provide tertiary care.[23,24] Yet, rural hospitals and family physicians can provide quality, accessible, family centered pregnancy and birthing care within an organized referral system.

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The prevalence of subclinical hypothyroidism in adults with low-normal blood thyroxine levels

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ABSTRACT. **The purpose of this study was to determine whether subclinical hypothyroidism, defined as the state in which there is an elevation of thyrotropin blood level (TSH) with a low-normal blood thyroxine level (T_4), can be found in clinically healthy subjects. We identified, by screening of adults in an ambulatory patient care facility, 67 persons with T_4 at the lower range of normal (4.5–6.0 μg/dL) who were free of acute nonthyroidal illness and who were not on medications known to affect T_4, other than long term thyroxine therapy. TSH was determined in all 67 persons, whose ages ranged from 25 to 87 years. Nineteen persons in this population had a history of thyroid disease and 48 did not. Elevation of TSH was found in 13 subjects (19%), seven of whom had a history of thyroid disease. The prevalence of TSH elevation was 50% in those with T_4 of 4.5–<5.0 μg/dL, 26% in those with T_4 of 5.0–5.5 μg/dL, and 8% in those with T_4 of >5.5–6.0 μg/dL ($p < 0.02$).**

Three of these 13 patients had been on thyroxine therapy at the time of the survey, and their thyroxine doses were adjusted, whereas five others were started on thyroxine therapy subsequent to the survey. Treatment doses as low as 0.05 mg/day increased the T_4 and returned the TSH to normal in six patients. In two of five patients who went untreated, there was a substantial fall in T_4 and/or a rise in TSH. Four of these five had a thyrotropin releasing hormone (TRH) infusion test and exhibited a markedly exaggerated TSH response as seen in hypothyroidism.

These data lend additional support to the notion that TSH elevation is a marker of early thyroid gland failure, and suggest that subclinical hypothyroidism may be recognized with high frequency by TSH screening in persons with low-normal T_4 levels.

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It is generally recognized that elevation of thyrotropin blood level (TSH) is at present the most sensitive indicator of decreased thyroid gland function.[1,2] This is so because the pituitary is exquisitely sensitive to minute changes in serum thyroxine (T_4) levels, and will secrete increased amounts of thyrotropin in response to small decreases in T_4—decreases that may not cause observable alterations in any other clinical or laboratory variable.[3-7] A spontaneous rise in TSH is, thus, an endogenous physiologic market of thyroid gland failure. For this reason, the detection of an elevated TSH allows the physician to recognize failure of the thyroid gland even before the development of any symptoms.[8,9]

TSH measurements may be of clinical value in uncovering early thyroid gland failure in selected patients, including the aged,[9-11] those with a history of Graves' disease,[12-16] and those with Hashimoto's or autoimmune thyroiditis.[17-19] Tibaldi and Barzel[20] proposed that TSH elevation might also be found with high frequency among persons with "borderline low T_4," which they did not further define.

In previous studies of hypothyroid patients receiving adequate thyroxine replacement, it was observed that the mean T_4 varied between 9.01 and 9.40 μg/dL and the standard deviation varied between 1.51 and 1.16 μg/dL.[20] These data suggested that T_4 values below 6.0 μg/dL (ie, two standard deviations below the mean) could be described as "borderline low-normal."

In this study, we therefore examined TSH levels in a group of ambulatory subjects whose T_4 was found to be at the lower end of the normal range (6.0–4.5 μg/dL). Our findings suggest that screening for TSH elevation in patients with T_4 in the lower end of this range (<5.0 μg/dL) may indeed be productive and of clinical utility.

PATIENTS AND METHODS

At the Montefiore Medical Group, a large ambulatory care facility, determinations of T_4 were obtained on every new adult patient, on adults having routine physical examinations, and on other adult patients who did not have recent T_4 determinations. The Montefiore Medical Center laboratory records were reviewed daily, and patients with T_4 levels between 4.5 and 6.0 μg/dL were identified and their sera subjected to thyrotropin assay.

Patients with acute nonthyroidal illness and those on medications known to affect T_4 (dilantin, estrogens, steroids) were excluded from the study. Patients with a history of thyroid disease, radioactive iodine treatment, goiter, or clinical suspicion of thyroid disease were included in the study but analyzed separately. Long-standing thyroxine therapy for various thyroid disorders was not a cause for exclusion.

The Medical Group physicians were informed when TSH elevation was discovered in their patients, and used their clinical judgment in deciding whether to institute thyroxine therapy or to modify ongoing therapy. The patients' records were reviewed five months after completion of the survey. In cases in which treatment was modified or initiated, T_4 and TSH levels were determined. In other cases, a thyrotropin releasing hormone (TRH) infusion test was done as follows: baseline blood specimens for determination of T_4 and TSH were obtained five minutes apart, an intravenous bolus of 500 micrograms of TRH was then given, and blood was drawn at 15, 30, and 60 minutes for

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TSH determination.

Determinations of T_4 and TSH were made by methods previously described.[6] Normal range for T_4 is 4.5–12 μg/dL, and for TSH, <0.6–5.0 MIU/mL. Multiple regression analysis and the Mantel-Haenszel chi-square methods were used in the statistical analysis of the data.

RESULTS

T_4 within the range of 6.0 and 4.5 μg/dL was found in 81 persons (4% of the screened population). Of these, 67 met the criteria for inclusion in the study. The characteristics of the 67 subjects are described in Table I. The age range for the entire group was 25 to 87 years. TSH elevation was found in 13% of subjects without known thyroid disease, as compared with 37% in the group with known thyroid disease. The group without thyroid disease and with normal TSH was nearly equally divided between females and males (55% vs 45%), but there was a female predominance (83%) if TSH was elevated ($p = 0.02$). The group with a history of thyroid disease had female predominance whether TSH was normal or elevated (67% and 86%, respectively; $p = 0.03$).

In Table II, the data are analyzed in roughly 0.5 μg/dL T_4 intervals. There is clearly a trend for increasing frequency of TSH elevation as T_4 nears the lower limit of the normal range. When patients with and without a history of thyroid disease are combined, the frequencies of elevated TSH in those with T_4 in the ranges of 4.5–<5.0, 5.0–5.5, and >5.5–6.0 μg/dL are 50%, 26%, and 8%, respectively ($p < 0.02$).

Table III records details of the patients in whom TSH elevation was found, as well as follow-up information from two to nine months later. Therapeutic steps were taken by patients' physicians as a result of the survey findings in eight cases. Three patients had been on thyroxine therapy at the time of the survey. Their thyroxine doses had been increased by 0.025 mg/day (MP and GR) or 0.100 mg thyroxine per week (VM), and, subsequently, their T_4 levels rose and their TSH fell to within the normal range. In five cases, thyroxine administration was newly instituted. In three of these cases, a rise of T_4 and normalization of TSH were achieved by thyroxine doses varying between 0.050 mg and 0.150 mg per day. In two patients (GS and ZT), thyroxine therapy is as yet suboptimal as indicated by the continued elevation of TSH.

Follow-up T_4 and TSH levels were obtained in the five patients who were not given thyroxine therapy, and in four of these a TRH infusion test was completed. As indicated by a fall in T_4 and/or a rise in TSH, there was further progression of thyroid failure in two of these patients (MR and BH) during the follow-up period. Figure 1 compares the TSH response to TRH injection with that seen in a normal population of the same age. A markedly exaggerated response, typical for hypothyroid subjects, can be seen in each of the four cases, including three (JS, TA, and SL) in whom T_4 and TSH did not change significantly during the follow-up period.

DISCUSSION

Two basic concepts underlie this study. One is that routine screening of T_4 is justifiable, especially in the aged in whom both hypothyroidism and hyperthyroidism are difficult to recognize clinically.[9,10,20,21] The other concept is that every person has an inherent "set-point" of serum thyroxine level, and that if the endogenous production of thyroid hormone and serum thyroxine level (T_4) falls even minutely below this "set-point," thyrotropin secretion increases and the blood level of TSH rises. This concept is supported by clinical and experimental studies that demonstrate the sensitivity of the thyrotrophs to ambient thyroid hormone level.[1–7]

TABLE I. Characteristics of Patients with Low-Normal T_4* (4.5–6.0 μg/dL)

	Thyroid Disease Absent		Thyroid Disease Present	
	TSH† ≤5.0	TSH >5.0	TSH ≤5.0	TSH >5.0
Age range (years)	25–87	61–80	42–73	31–85
Total number of patients	42 (87%)	6 (13%)	12 (63%)	7 (37%)
Number of females	23 (55%)	5 (83%)	8 (67%)	6 (86%)

* T_4 = thyroxine.
† TSH = thyrotropin.

TABLE II. Number of Patients with TSH >5.0 MIU per mL/Total Number of Patients

	T_4 Range, μg/dL			
	4.5–<5.0	5.0–5.5	>5.5–6.0	
Thyroid disease absent	1/4	2/15	3/29	$p > 0.05$
Thyroid disease present	3/4	4/8	0/7	$p < 0.03$
Totals	4/8 (50%)	6/23 (26%)	3/36 (8%)	$p < 0.02$

The present study reveals that 50% of subjects with T_4 between <5.0 and 4.5 μg/dL and 26% of those with T_4 between 5.5 and 5.0 μg/dL have elevated TSH levels. Overall, 19% of subjects with low-normal T_4 of 6.0 μg/dL or less have elevated TSH. Thus, we can demonstrate that TSH screening in patients with low-normal T_4, especially at the lower end of the range (<5.0–4.5 μg/dL), is likely to uncover a significant number of elevated values.

Presently, a history of Graves' disease and of Hashimoto's or autoimmune thyroiditis[12–19] are indications for routine TSH screening. Existing data suggest that a significant number of cases with elevated TSH may be uncovered by TSH screening in the aged in general: In a screening of the original Framingham cohort of people older than 60 years of age, Sawin et al[9] found TSH elevation in 13.6% of the women and 5.7% of the men. In a community survey of Whickam, England, Tunbridge et al[11] recorded elevated TSH in 7.5% of the females and 2.8% of the males, but the prevalence of TSH elevation rose to 17.5% in females older than 75 years of age. In a geriatric screening clinic in San Francisco, on the other hand, only 2.6% of the patients had elevated TSH.[22] We believe that "low-normal T_4" should be considered a special justification for TSH screening, particularly in the elderly.

The condition in which patients have virtually no clinical findings of hypothyroidism but an elevation of TSH has been alluded to in the literature by a variety of names, including "limited thyroid reserve syndrome," "preclinical hypothyroidism," "premyxedema," and "subclinical hypothyroidism," with the latter being the term most commonly used. This condition represents early thyroid gland failure. Thyroid gland failure is a slow and insidious process which leads to the eventual development of hypothyroidism, but this failure may become clinically apparent only after it has progressed significantly. TSH elevation may be used as a marker of early and clinically unapparent thyroid gland failure.

Although the final number of cases with elevated TSH in this study is small, the data do provide further evidence that TSH elevation is indeed a manifestation of thyroid gland failure. This is clearly shown in those patients in

TABLE III. Clinical Data in 13 Subjects with Low-Normal T_4 (4.5–6.0 μg/dL) and Elevated TSH (>5.0 MIU/mL) at Baseline and After Two to Nine Months of Follow-up Observation

			First Observation				Follow-up Evaluation				
Patient	Sex	Age (yr)	Date	T_4	TSH	Therapy*	Date	T_4	TSH	Therapy*	Comment
MP	F	80	3/28	4.9	12.3	.075/d	11/8	6.0	1.5	.100/d	Partial thyroidectomy
GR	F	82	4/1	4.9	17.6	.050/d	9/9	8.5	4.7	.075/d	Graves' disease
VM	F	85	5/16	5.1	6.3	.50/wk	7/23	7.5	<0.6	.60/wk	Radiotherapy for thyroid cancer
RJ	M	77	2/23	5.3	60.0	—	7/12	10.2	<0.6	.150/d	Graves' disease
DS	F	78	3/15	5.5	7.8	—	6/27	8.5	2.1	.050/d	No history of thyroid disease
AK	F	61	5/13	4.9	10.0	—	9/9	10.7	<0.6	.100/d	No history of thyroid disease
GS	F	31	5/10	5.2	18.6	—	9/20	7.1	10.5	.050/d	Graves' disease
ZT	F	28	6/6	5.3	8.6	—	10/4	13.0[†]	17.5	.100/d	Thyroiditis
MR	F	66	6/6	5.6	20.0	—	10/29	4.3	63.3	—	No history of thyroid disease
BH	F	47	2/22	4.8	8.0	—	10/28	5.4	17.6	—	Graves' disease
JS	M	80	3/18	5.9	13.0	—	10/30	5.6	12.3	—	No history of thyroid disease
TA	F	70	4/15	5.6	7.5	—	10/29	6.5	6.2	—	No history of thyroid disease
SL	F	73	6/13	5.5	5.8	—	10/28	5.0	6.2	—	No history of thyroid disease

* Levo-thyroxine, mg.

[†] Patient has increased thyroxine binding.

whom treatment was initiated or modified and in whom clinical intervention increased the T_4 and corrected the TSH. (In subjects with normal TSH levels, the administration of thyroxine in the dosages used would not be expected to change the T_4 level.) Further evidence that TSH elevation is indeed a state of subclinical hypothyroidism is given by the cases in which no medical intervention was undertaken and in which the TRH infusion test was grossly abnormal and typical for the hypothyroid state. In fact, although these patients had no history of thyroid disease, their findings are identical with those patients with asymptomatic autoimmune thyroiditis classified as Grade III by Bastenie et al.[23] In the untreated group there was evidence of the progressive nature of the thyroid failure in the fall of T_4 and/or the rise of TSH over a relatively short period of observation in two cases. The sensitivity of the pituitary to minor changes in thyroxine input had been previously documented.[1–7] This sensitivity may decrease with age[6] or disease,[24] but it is clearly demonstrable in the case of the 85-year-old woman (VM) in whom the return to normal of a minimally elevated TSH was achieved by an increase in thyroxine replacement of 0.100 mg per week, or 0.01425 mg per day.

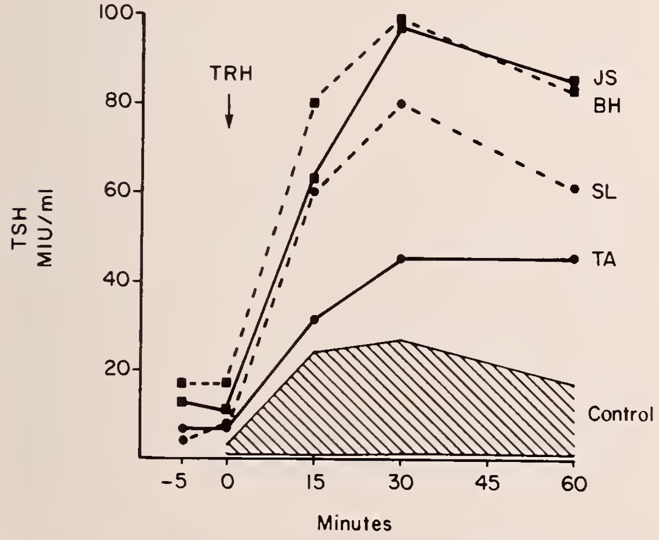

FIGURE 1. TSH response to the TRH test in four subjects with low-normal T_4 and elevated TSH. The normal response is depicted in the hatched area, and is based on 32 normal subjects with an age span of 28 to 87 years.[6]

Based on clinical findings, we believe that virtually all patients with a failing thyroid gland, ie, patients with elevated TSH and normal or near normal T_4, but in particular those with positive thyroid antibodies, should be given supplemental thyroxine in doses that would return the TSH to normal. Such patients should then be followed with periodic assessment of their T_4 and upward adjustment of the thyroxine dosage if a fall in T_4 and a rise in TSH occurs again. Using this strategy, which is generally safe and simple, the patient's thyroidal failure will remain fully compensated or "subclinical" at all times and clinical, symptomatic hypothyroidism will be prevented from emerging altogether.[20] Full replacement doses of thyroxin should be avoided in these states of early thyroidal failure, since some failing thyroid glands are still driven by abnormal thyroid stimulating immunoglobulin and are not subject to feedback inhibition, and, in such cases, thyrotoxic symptoms may be precipitated by full replacement doses. Supplemental thyroxine therapy avoids this complication.

In summary, TSH elevation is present in some people with low-normal T_4 levels (6.0 μg/dL or less). The likelihood of finding an elevated TSH level is higher at lower levels of T_4 (<5.0 μg/dL). An elevated TSH in this setting represents early thyroidal failure. Thyroxine supplementation is recommended as a way of preventing clinical hypothyroidism from emerging in patients with failing thyroid glands.

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SPECIAL ARTICLE

An overview of the activities of the New York State Board for Medicine

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With the establishment of licensure requirements for physicians in 1892, the medical profession became the first of the 32 licensed professions to be regulated by the Board of Regents. The State Board of Medical Examiners was established under Chapter 344 of the Laws of 1907. The first meeting of the board was called to order at 3:00 PM on June 24, 1907, by then Commissioner of Education Andrew Draper. In his opening remarks Commissioner Draper stated that the establishment of the board "had come to be inevitable. The doctors had tired out the legislature and the legislature had made up its mind that it would do something drastic some day to put an end to the eternal fuss that was coming up from the doctors."

The medical profession, as defined in the Education Law of New York State, now encompasses allopathic and osteopathic physicians, physician's assistants, and various types of specialist's assistants. Currently there are approximately 60,000 New York State registered physicians. Of this group about 58,800 are allopathic physicians and 1,200 are osteopathic physicians. There are 2,572 registered physician's assistants and 44 specialist's assistants. In addition to providing advice concerning standards of medical licensing, the State Board for Medicine addresses matters concerning undergraduate and graduate medical education, medical manpower supply and demand, medical practice issues, and lifelong learning or continuing competency in the medical profession.

COMPOSITION OF THE STATE BOARD FOR MEDICINE

Presently, the State Board for Medicine is composed of 20 physicians, including two osteopathic physicians, and two public members. The specialties represented on the State Board cover a broad spectrum: anesthesiology; family practice; internal medicine; acupuncture; obstetrics/gynecology; ophthalmology; pathology, pediatrics; neurology; psychiatry; public health/preventive medicine; radiology; surgery; and nuclear medicine. Each of the state's judicial districts is represented on the board. The Board for Medicine is one of the most diversely represented boards. Four of the board members are female and nine board members are minority group members. Seven of the board members are engaged primarily in academic medicine, including Eli Stark, an Assistant Dean for Clinical Studies and Post Graduate Education at the New York College of Osteopathic Medicine; and Philip Holtzapple, Assistant Dean for Curriculum at State University of New York Health Science Center at Syracuse. The other State Board members are in private practice or are involved in other professional activities. Four board members are graduates of foreign medical schools, representing National Taiwan University in Taipei, Yokohama University in Japan, University of the Philippines in Manila, and Shiraz University in Iran.

The full board usually meets three times a year. On the evening before each full board meeting, the executive committee, comprised of the chair, vice chair, immediate past chair, and the senior public member, meets to review the agenda materials. In addition, ad hoc committees are frequently established and asked to address a variety of important issues. Such issues include limited licensure, continuing competency, acupuncture, academic licensure, geriatrics in medical education, and graduate medical education. For example, in 1989 a committee of the board reviewed the First Annual Report of the Council on Graduate Medical Education. The committee's recommendations were presented to the regents in July 1989. The State Board currently is reviewing the Second Annual Report of the Council. In 1988 the report of the board's Committee on Geriatric Medical Education became an integral part of the interagency Long Term Care Policy Coordinating Council's (LTCPCC) report on Geriatrics in Medical Education. LTCPCC is an interagency task force, cochaired by the Commissioner of Health and the Director of the State Office for the Aging, charged with reviewing issues related to long term care and providing recommendations to the governor's office. During 1987, LTCPCC studied the current extent and methods of educating physicians in geriatric care. Department staff and board members played key roles in the development of that report as members of the LTCPCC subwork group.

Board members also serve as members of peer panels to review moral character and restoration cases. In 1989, board members participated in 12 restoration hearings and six moral character hearings. This resulted in board member service for 48 days. In addition, a majority of

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board members are active in local, state, and national organizations. These organizations include the Federation of State Medical Boards, National Board of Medical Examiners, Medical Society of the State of New York, Associated Medical Schools of New York, American Medical Association, Liaison Committee on Medical Education, and various specialty board activities. Dr Margaret Colgan, chairperson of the board, is a member of the Federation Licensing Examination (or FLEX) Test Review Committee of the Federation of State Medical Boards, Dr Patrick Carone, vice chairperson of the board, is a member of the Nominating Committee of the Federation of State Medical Boards, and Dr Pascal James Imperato is editor of the *New York State Journal of Medicine.*

OFFICE OF THE STATE BOARD FOR MEDICINE

The primary mission of the Office of the State Board for Medicine is to advise and assist the Board of Regents and the Department of Education on matters related to medical licensure, practice, and education. In accomplishing this mission, the Office of the State Board for Medicine establishes and maintains effective liaison between the medical profession, the State Board, the Education Department, and the Board of Regents. It is a principal component of the Office of the Professions and provides a variety of services for the Office of the Professions and in support of the State Board for Medicine. The board office prepares the agenda for each meeting of the board and brings before the board issues of state and national importance. The board office arranges for national and state leaders in medical education and department staff to meet with the board to discuss issues related to their respective areas of responsibility and concern. During the past few years representatives of the National Board of Medical Examiners and the Federation of State Medical Boards have met with the members of the State Board for Medicine to discuss topics of mutual interest; such topics have included the uniform examination pathway to medical licensure and the development and use of the Special Purpose Examination (SPEX). SPEX is a cognitive examination designed to assist licensing jurisdictions in the assessment of the knowledge base of practicing physicians. In 1987, members of the State Board for Medicine and former Regent Dr Salvatore Sclafani reviewed the SPEX and recommended to the Board of Regents that the SPEX be used in the case of certain individuals seeking restoration of medical licensure. At their May 1988 meeting, the Board of Regents accepted these recommendations and amended Section 28 of the Rules of the Board of Regents to permit the use of a proficiency examination such as SPEX in restoration cases. In addition, the executive director of the Associated Medical Schools of New York, the executive director of the Statewide Geriatric Resource Center, the co-chairman of the New York State Advisory Committee on Physician Recredentialing, the chairperson of the New York State Hospital Review and Planning Council, and officers of the Medical Society of the State of New York have met periodically with the State Board to discuss issues such as geriatric medical education and continuing competency. Staff from all areas of the Education Department work closely with staff of the board office and frequently meet with the board to discuss such issues as school health programs, licensing procedures, the disciplinary process, manpower planning, and improving the access of minorities to the medical profession. Interprofession dialogue and communication is important; to that end members of other state boards, such as Nursing and Physical Therapy, meet on occasion with Medicine to discuss topics of mutual interest. These topics have included such issues as proposals for direct access to physical therapy services and the development of alternative criteria for the certification of nurse practitioners.

Staff of the State Board office includes the executive secretary, two professionals, and three support staff members. Daily, staff spend a considerable but necessary amount of time responding to written and telephone inquiries on a wide variety of issues related to medical practice, undergraduate and graduate medical education, and licensing requirements. During the first quarter of 1990, the State Board office handled 3,565 telephone calls, 2,728 pieces of correspondence, and 85 licensing cases.

The board office assists the Division of Professional Licensing Services by reviewing the applications of persons who do not clearly satisfy the licensing requirements established in Part 60 of the commissioner's regulations. Many of the cases involve applicants who request credit for postgraduate medical education completed outside the United States and Canada, or who have engaged in fellowship training or training that was not devoted exclusively to clinical medicine. The executive secretary reviews these cases and either recommends action to the division based on precedent or refers those cases raising issues not previously considered to the full board for review and recommendation. In 1989, the board office evaluated approximately 500 cases and referred 35–40 of them for review by the full board. The executive secretary, in collaboration with selected board members, also reviews all applications for acupuncture licensure and certification. This includes the review of research protocols submitted by physicians or dentists seeking acupuncture certification.

The board office is also responsible for reviewing the proposals of physicians with service obligations under the Regents Physician Shortage Area Scholarship Program and the New York State Medical Contract Program. As they near the completion of professional training, Regents Physician Shortage Area Scholarship recipients submit proposals to the Higher Education Services Corporation and participants in the Contract Program submit proposals to the department's Coordinator of Professional Practice to fulfill their service obligations. The corporation and coordinator subsequently forward the respective service proposals to the board office. Staff determine if the location of an individual's proposed practice is in a shortage area. If a proposed practice is in a shortage area, the board office writes to the individual indicating approval of the proposed practice plan. If the proposed practice is not in a shortage area, the board office informs the individual that his or her practice cannot be approved and that appropriate steps will be taken to initiate repayment.

For the past ten years the evaluation of unregistered and unaccredited medical schools (basically, all foreign medical schools) seeking clerkship training for their students in New York State has been a major responsibility of the board office.

In 1981 the regents approved regulations governing the circumstances under which students enrolled in foreign medical schools may engage in clinical clerkships in New York State. Those regulations provide that students enrolled in medical schools outside the United States and Canada may perform a maximum of 12 weeks of clerkships on two conditions: First, the student's foreign medical school must certify that the student is enrolled and currently matriculated; and second, the New York State teaching hospital must certify that it has agreed to accept the student for clerkship training.

If foreign medical students wish to engage in more than 12 weeks of clerkships, they must be enrolled in a medical school that has been reviewed and approved for such purposes by the State Education Department. Immediately following the 1981 regents action, the staff developed and implemented a system for reviewing foreign medical schools. This system has gained national renown as an effective assessment instrument. Several other states now rely upon New York as the primary resource for information concerning foreign medical schools. The system relies on a data base document that schools seeking approval are required to complete. They next provide extensive information about the institution's program of medical education. Staff also developed an instructional guide for use by consultant evaluators and an extensive list of pertinent academic questions for use by team members during the site visit. An information guide for foreign medical schools that express interest in seeking department approval for their programs is available.

A foreign medical school seeking department approval must submit a formal application to the State Board office. In addition to completion of the data base document provided by the office, the applicant institution must submit a current catalog, course syllabi, and faculty and student handbooks or other documents setting forth policies related to faculty and students. All expenses connected with the evaluation are borne by the applicant institution. No state funds are involved in the evaluation of foreign medical schools.

A site visit by staff and consultants is critically important to the process. The major purpose of the site visit to the campus in the country in which the school is located is to effectively evaluate the institution's program, primarily the basic medical sciences, which prepares students for clinical training. As part of the evaluation, visits are also made to the teaching hospitals in New York State that have agreed to affiliate with a foreign medical school for clerkship training. The purpose of the affiliate visit is to evaluate the proposed system of clerkship training, specifically the integration of such training within the foreign medical school's total program of medical education and the respective roles of the foreign medical school and the hospitals in faculty appointments, student selection, supervision, coursework, and academic evaluation.

Since 1981 eight foreign medical schools have submitted applications for approval: Universidad Del Noreste, Tampico, Tamps, Mexico; Universidad Autonoma de Guadalajara, Guadalajara, Jal, Mexico; Ross University, Dominica, West Indies; American University of the Caribbean, Montserrat, West Indies; St George's University School of Medicine, Grenada, West Indies; Aguinaldo School of Medicine, Cavite, Philippines; Far Eastern University, Manila, Philippines; and Fatima College of Medicine, Manila, Philippines. The applications of the Universidad Autonoma de Guadalajara, St George's University School of Medicine, and Fatima College of Medicine were approved. Universidad Del Noreste was approved with conditions but approval was subsequently withdrawn following a determination the conditions had not been fulfilled. The applications of Ross University and the American University of the Caribbean were not approved. The applications of Far Eastern University and Aguinaldo School of Medicine lapsed for a variety of reasons, but primarily because they were unable to affiliate with a teaching hospital in New York State.

Teams have also visited the American University of Beirut, Beirut, Lebanon (1982), and the Sackler School of Medicine of Tel Aviv University, Ramat Aviv, Israel (1987), for purposes of program registration. Both institutions are chartered by the regents and the Sackler School of Medicine participates in the New York State Medical Contract Program.

The evaluation teams are composed of four or five medical educators and practitioners from New York and other states who are experienced in medical program evaluation. A member of the State Board for Medicine is included in each team. Each team also includes a graduate of a foreign medical school and individuals who are fluent in the language of the country in which the school is located. The executive secretary and the associate have participated in each site visit. Comprehensive reports of each evaluation are maintained in the Office of the State Board for Medicine. The information available in the board office is unavailable anywhere else in the world and has become an important source of information for licensing bodies throughout the United States. Telephone and written requests for this information are frequently received and staff have been called upon to provide testimony and consultant expertise to a variety of agencies including the General Accounting Office, the American Medical Association, and other state licensing boards.

The board office is also responsible for issuing certificates to students enrolled in approved foreign medical schools authorizing them to engage in more than 12 weeks of clerkships in teaching hospitals affiliated with their school if they have achieved a passing score on a satisfactory proficiency examination. Since the currently used proficiency examination is Part I of the Foreign Medical Graduate Examination in the Medical Sciences, the authorizing certificate is commonly known as the "FMGEMS Certificate." The board office also issues "Letters of Eligibility" to students enrolled in both approved and unapproved foreign medical schools for clerkships of 12 weeks or less. Students enrolled in approved foreign medical schools may engage in up to 12 weeks of clerkships in teaching hospitals not affiliated with their medical school. Students enrolled in unapproved medical schools may perform a maximum of 12 weeks of clerkships in any teaching hospital in the state.

In 1989, 197 letters of eligibility of this type were issued for 191 students from 53 foreign medical schools representing 25 different countries, serving 29 different clerkships in 34 hospitals in the state.

During the past few years the executive secretary and associate have also participated in the evaluations of every medical school and physician's assistant training program in New York State. This includes the programs of medical education at 12 medical schools and ten physician's assistant programs. All of the site visits have been coordinated with program reviews by national accrediting bodies. In addition, the executive secretary reviews acupuncture training programs as a result of recent changes in the commissioner's regulations related to acupuncture certification requirements and the establishment of a new exempt category of practitioners.

The staff in consultation with members of the State Board also advise the department on the development of regents' legislative proposals and positions concerning bills being considered by the state legislature. During the current legislative session, staff developed six regents' legislative proposals and reviewed approximately 200 bills that had been introduced.

An important component of the executive secretary's responsibilities includes working closely with local, state, and national medical organizations. He meets regularly with county or district medical societies and serves in an ex officio capacity on various state medical society committees. In addition, he is an ex officio member of the Continuing Medical Education Council of New York State, which includes in its membership the Medical Society of the State of New York, the Associated Medical Schools of New York, and the Association of Medical Specialty Societies of New York. Several board members and the executive secretary have also recently been appointed to and serve on the Governor's Advisory Committee on Physician Recredentialing.

At the national level staff and board members work closely with representatives of the American Medical Association (AMA), Administrators in Medicine Society (AIMS), National Board of Medical Examiners (NBME), and Federation of State Medical Boards (FSMB). During the past few years the executive secretary has served on the FSMB's Committee on Fraudulent Medical School Credentials, Long Range Planning Committee, and the board of directors. Involvement with national organizations such as the AMA, NBME, and FSMB has been critical in addressing medical education, practice, and licensing issues of national importance.

One of the most important topics recently discussed by the AMA, FSMB, and NBME has been the development of a uniform examination pathway to medical licensure. An information item on this topic was presented to the regents in the Deputy's Report on March 22, 1990. It was the lead topic of discussion at the 1990 annual meeting of the Federation of State Medical Boards which was recently held in Birmingham, Alabama. This topic has been the object of considerable discussion and debate at the state and national levels for the past two years.

The large number of foreign medical graduates who seek licensure in New York and the regents' policy to promote access to the professions and encourage equity in the licensing process make it critically important for New York to participate in this national debate. Several legislative leaders, including Senator Donovan, have been strong advocates of a uniform examination pathway. We have had positive dialogue on this issue with various national organizations, including the Federation of State Medical Boards; the Association of American Medical Colleges (AAMC); the American Medical Association; and the Educational Commission for Foreign Medical Graduates (ECFMG).

The FSMB, along with a variety of other concerned organizations and groups, has taken an active role in addressing this issue. Department staff, primarily the executive secretary for medicine, have participated in many discussions with the FSMB and concerned groups.

At its January 6, 1989, meeting, the New York State Board for Medicine unanimously passed a resolution endorsing the concept of developing a uniform examination pathway to medical licensure. This resolution was later endorsed by the FSMB's Long Range Planning Committee, and at the April 1989 annual meeting of the FSMB, a motion to consider the resolution was subsequently introduced and unanimously passed. In September 1989 Parts I and II of NBME became available to graduates of foreign medical schools for the first time.

The decision to make available Parts I and II of the NBME to foreign medical graduates is actually only the beginning of a process which should result in a true uniform examination pathway to medical licensure. Accordingly, we anticipate that an entirely new examination sequence will evolve within the next two to four years. We expect that the uniform examination pathway to medical licensure will initially include Parts I and II of NBME as they presently exist and a yet-to-be developed Part III which is intended to serve as a clinical competency component.

LICENSING REQUIREMENTS

In 1989, 4,202 physicians were newly licensed in New York State. Of this group approximately 1,151 were graduates of foreign medical schools. The New York system for licensing physicians relies on a structure referred to as the "three pillars of medical licensure"—education, experience, and examination. Each of these pillars is important and, without any one of them, the viability of the entire licensing process becomes questionable.

In addition to New York's general requirements for medical licensure such as age and good moral character, there are specific requirements of education, professional experience, or postgraduate training. The requirements of experience or postgraduate training and examination differ for graduates of unaccredited or unregistered schools and graduates of accredited or registered schools. The educational requirements for graduates of all types of medical schools are identical: completion of at least two years of acceptable preprofessional education; completion of a program of medical education of not less than 32 months; and receipt of an acceptable degree. The program of medical education either must be approved by an accrediting organization within the United States or Canada which is acceptable to the department, that is, the Liaison Committee on Medical Education or the American Osteopathic Association, or must be recognized by the appropriate regulatory authorities of the country in which the school is located.

In addition to satisfying the educational requirements and passing the appropriate licensing examination, graduates of accredited or registered programs must complete a year of approved postgraduate training. Graduates of unaccredited or unregistered programs, that is, most foreign medical schools, must pass an acceptable proficiency examination (the ECFMG, VQE, or FMGEMS), complete three years of approved postgraduate training, and pass FLEX.

The purpose of the three-year postgraduate training requirement for foreign medical graduates is to assure that all licensees have developed a readiness for independent practice and patient management. The assumption that underlies this requirement is that graduates of accredited or registered programs have reached an acceptable level of clinical performance based upon graduation from an education program which has been determined to have met agreed upon national standards of excellence. Information concerning medical education programs in most other countries is not readily available and the three-year postgraduate training requirement is intended to remediate any real or perceived deficiencies which may exist in the undergraduate medical education program. Within the context of the current New York licensing requirements, two major objectives have been achieved: the public health and welfare are protected while high quality medical care is becoming increasingly available, and licensing applications are reviewed on an equitable and reasonably timely basis.

OTHER PROFESSIONS

Chapters 1135 and 1136 of the Laws of 1971 created the professions of physician's assistant and specialist's assistant. The responsibility for implementing the legislation, which became effective April 1, 1972, is shared by the State Education Department and the Department of Health. Under the provisions of Section 6540 of the Education Law, the State Education Department is responsible for evaluating and approving physician's assistant and specialist's assistant educational training programs. Under the provisions of Section 3700 of the Public Health Law, the Commissioner of Health has the power and duty to "promulgate regulations defining and restricting the duties which may be assigned to physician's assistants and specialist's assistants by their supervising physician, the degree of supervision required and the manner in which such duties may be performed."

Physician's assistants, which differ from specialist's assistants, are qualified by education and training to elicit a complete medical history, perform a comprehensive physical examination, perform or order diagnostic laboratory tests and interpret the results, perform therapeutic procedures, and respond to common emergencies. It has been estimated that physician's assistants can perform 70% of the clinical procedures routinely performed by physicians in general or family practice.

There are currently 2,572 physician's assistants registered in New York State. A physician's assistant is considered a dependent practitioner working under the supervision of a licensed physician who is responsible for the actions of the physician's assistant. The supervising physician may delegate to the physician's assistant any medical procedures and tasks that are routinely performed within the normal scope of the physician's practice.

The physician's assistant/specialist's assistant statute created a unique professional category in New York State. Unlike other licensed, certified, or registered health professionals, physician's assistants are provided no functions or activities of their own but act solely on delegation from the supervising physician. The physician's assistant is entitled to use his or her medical skills and knowledge in the performance of medical acts, functions, and services only on delegation from and on behalf of the supervising physician.

The statute and implementing rules and regulations provide that medical acts, functions, and services delegated to the physician's assistant must be within the scope of practice of the supervising physician and which the physician's assistant is qualified to perform. It is the responsibility of the supervising physician to assure that the physician's assistant is competent to perform that which is delegated. In the private office setting it is largely the judgment of the supervising physician that determines the functions and activities of the physician's assistant. In the hospital setting the governing authority of the hospital may place limits on the functions and activities that may be delegated by the supervising physician.

The statute, rules, and regulations specifically permit the physician's assistant to function at a distance from the supervising physician. The supervising physician need not see each patient prior to the physician's assistant's providing services. The physician's evaluation of the medical knowledge, skills, and judgment possessed by the physician's assistant and the nature of the problem presented for management are major determinants of the "degree of freedom" permitted by the supervising physician.

In the hospital setting the supervising physician may, if permitted by the hospital, delegate the writing of medical orders, including controlled substances, to the physician's assistant. Medical orders written by the physician's assistant must be countersigned by the supervising physician within 24 hours, but countersignature is not required prior to execution of the order. The procedures for prescription writing are specified in Part 94 of the Administrative Rules and Regulations of the Department of Health.

There are currently 44 specialist's assistants registered in New York State. Specialist's assistants are highly skilled in a group of clinical procedures that comprise a specialized clinical service. New York State has designated four categories of specialist's assistants: orthopedic assistant, urologic assistant, radiologic assistant, and acupuncture assistant. Qualified individuals are eligible for registration as specialist's assistants in the specialty category appropriate to their qualifications.

The requirements for registration as a physician's assistant or specialist's assistant include the following. At the time of application the applicant must: (1) be at least 21 years of age; (2) be of good moral character; (3) successfully complete a four-year course of study in a secondary school approved by the Board of Regents or pass an equivalency test; and (4) satisfactorily complete an approved program for the training of physician's assistants or specialist's assistants.

The approved program for the training of physician's assistants shall include not less than 40 weeks of supervised clinical training and 32 credit hours of classroom work. The Commissioner of Education is empowered to determine whether an applicant possesses equivalent education and training, such as experience as a nurse or military corpsman, which may be accepted in lieu of all or part of an approved program. In addition, certain individuals, such as military corpsmen, who have not completed an approved educational program may satisfy the educational requirement by passing the examination administered by the National Commission on Certification of Physician's Assistants.

In addition to the above registration requirements, additional requirements for registration as a specialist's assistant in each of the designated categories follow.

Orthopedics. Specialist's assistants registered in orthopedics must:

1. satisfactorily complete a program for the training of orthopedic assistants approved by the New York State Education Department, or
2. possess equivalent education, training, and experience. Training and experience while in military service which led to an orthopedic specialist, orthopedic cast room technician, or orthopedic clinic technician rating and two years of satisfactory experience as an orthopedic assistant working under the supervision of an orthopedic surgeon within the past five years; or completion of medical corps school and five years of satisfactory experience as an orthopedic assistant working under the supervision of an orthopedic surgeon within the past eight years may be considered equivalent education, training, and experience for the purpose of registration in this category, or
3. achieve passing scores on the National Board for Certification of Orthopedic Physician Assistant's Examination (NBCOPA).

Urology. Specialist's assistants registered in urology must:

1. satisfactorily complete a program for the training of urologic assistants approved by the New York State Education Department, or
2. possess equivalent education, training, and experience. Training and experience while in military service which led to an urology surgical technician or urological technician or clinical specialist rating and two years of satisfactory experience as a urologic assistant working under the supervision of a urologist within the past five years; or completion of medical corps school and five years of satisfactory experience as a urologic assistant working under the supervision of a urologist within the past eight years may be considered equivalent education, training, and experience for the purpose of registration in this category.

Radiology. Specialist's assistants registered in radiology must:

1. be licensed as a radiologic technologist by the New York State Department of Health, and
2. satisfactorily complete a program for the training of radiologic assistants approved by the New York State Education Department.

Acupuncture. Specialist's assistants registered in acupuncture must:

1. graduate from a school of medicine or a school of traditional oriental medicine, herb medicine, or acupuncture acceptable to the New York State Education Department;
2. provide evidence of five years of experience in the use of acupuncture; and
3. meet the English proficiency requirement of Section 59.3(e) of the commissioner's regulations.

The department may require successful completion of an examination in acupuncture as an alternate qualification for any applicant unable to submit satisfactory evidence of education and training.

A specialist's assistant in acupuncture may practice only in an approved institutional setting and only under the supervision of a physician or dentist certified to practice acupuncture.

In addition to registered specialist's assistants in acupuncture there are presently three other categories of acupuncture practitioners recognized in New York State: physicians and dentists certified to use acupuncture; licensed or independent acupuncturists; and those individuals practicing under the exemption provisions of Chapter 663 of the Laws of 1988.

Physicians and dentists seeking certification to use acupuncture must:

1. possess a New York State license and current registration to practice medicine or dentistry;
2. complete at least 300 hours of instruction in programs in acupuncture registered by the department, or in programs determined by the department to be the equivalent of registered programs; and
3. practice within the provisions of an approved research protocol.

There are currently 497 physicians and dentists certified to use acupuncture in New York State. Individuals who are not licensed physicians or dentists may also seek licensure to practice acupuncture as licensed acupuncturists.

Licensed or independent acupuncturists must:

1. have a license to practice acupuncture which is acceptable to the State Board for Medicine as a doctor of acupuncture, herb physician, or doctor of traditional Chinese medicine duly licensed by the licensing board of any state or foreign country. In judging the acceptability of licenses, the criteria, requirements, and administration of the applicable licensing procedures are evaluated. Licenses which represent levels of competence and skill in the practice of acupuncture and which represent the sub-

stantial equivalent of those standards required for the licensing of professions in New York may be accepted;

2. submit satisfactory evidence of having practiced acupuncture for at least ten years; and
3. meet the English proficiency requirement of section 59.3(e) of the commissioner's regulations. There are currently 145 licensed acupuncturists in New York State.

Chapter 663 of the Laws of 1988 permits individuals who are otherwise unlicensed or uncertified but have completed an educational training program acceptable to the Education Department to practice acupuncture under supervision in detoxification programs approved by the Division of Alcoholism and Alcohol Abuse, Division of Substance Abuse Services, or the Department of Health. Chapter 772 of the Laws of 1990 establishes new licensing criteria and procedures for acupuncturists effective May 1, 1991.

ROLE OF THE STATE BOARD FOR MEDICINE WITHIN THE OFFICE OF THE PROFESSIONS

The State Board has considered and advised the regents and the department on a wide variety of important issues during the past few years. These issues include licensing and clerkship requirements as they relate to foreign medical schools, establishing criteria for the training of acupuncturists, assuring the continuing competence of physicians, geriatric medical education, minority access to the medical profession, guidelines for reviewing postgraduate training in the US and abroad, use of the special purpose (SPEX) examination, academic/limited licensure, rural health care, and a uniform examination pathway to licensure.

The board has also advised the department on such matters as the scope of practice of occupational and physical therapy; nurse practitioners; diagnostic services performed by clinical laboratories; the use of topical drugs by optometrists; homeopathic and orthomolecular medicine and chelation therapy; administration of IV therapy and the structure and content of the department's application for medical licensure. In addition, board members and staff have worked with departmental and interdepartmental committees to review such issues as geriatric medical education, manpower studies, and rural health needs.

Board members and staff have participated in reviews of the Foreign Medical Graduate Examination in the Medical Sciences examination; the National Commission on Certification of Physician's Assistant examination; the National Orthopedic Assistant examination; the FLEX examination; and the SPEX examination to assess the appropriateness of their use as proficiency/licensing examinations in New York State. All of these examinations were subsequently determined to be acceptable by the department and the regents.

The board is currently reviewing and discussing a number of issues of state and national importance. These issues include:

1. The development of a uniform examination pathway to medical licensure. This issue continues to be of considerable importance. Staff and a member of the State Board recently participated in discussions on this topic at the Federation of State Medical Board's meeting in Birmingham, Alabama. Some time during the next couple of years it may be necessary to consider amendments to Part 60 of the commissioner's regulations in order to accommodate a new examination system;
2. Geriatric medical education. The board has discussed this topic in the past and it continues to be a concern of the board. This topic was revisited at the September 1990 state board meeting which was attended by Dr Butler of Mt Sinai School of Medicine and other representatives of the medical education community;
3. The development and refinement of guidelines for the review of postgraduate or residency training obtained abroad and fellowship training obtained in the United States and Canada;
4. The issue of lifelong learning or continuing competency. Board members currently serve on the Advisory Committee on Physician Recredentialing and work closely with staff to achieve the regents objective of assuring the continuing competency of all health care professionals including physicians;
5. The structure and content of programs of graduate medical education (ie, residencies). Staff and board members are now working closely with the Council on Graduate Medical Education and reviewing the council's Second Annual Report;
6. Current physician licensing requirements. The board's recommendations may be forthcoming concerning suggested changes in Part 60 of the commissioner's regulations as they relate to the current postgraduate training requirement;
7. The importance of attempting to establish standards of practice and developing a system of quality assurance. These increasingly important areas will require increased interaction with professional associations, specialty societies, and the educational community;
8. The need to increase opportunities for members of underrepresented minority groups to enter the medical profession. Members of department staff and representatives of the Associated Medical Schools periodically meet with the board to discuss this topic. Board members would like to become increasingly active in developing strategies to address this problem; and
9. Issues related to scope of practice and the delivery of services by members of the health care team. Recently, members of the State Board worked closely with department staff and members of the State Board for Nursing to resolve issues related to the use of licensed and nonlicensed personnel on intravenous therapy teams. The roles of licensees and nonlicensees as members of various treatment teams will continue to require periodic clarification. The State Board expects to work closely with department staff and the members of other boards in resolving these issues.

The allocation of scarce medical resources*

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On October 1, 1989, the Department of Social Services of New York State began a new policy that did not require approval of the legislature; this policy limits Medicaid patients to no more than 14 visits a year to a doctor. New York State also limits the number of visits to dentists, podiatrists, and laboratories as well as the number of prescription drug purchases that a Medicaid beneficiary can make. The Committee on Bioethical Issues of the Medical Society of the State of New York believes that these rationing measures, designed to curtail fraud at the expense of patient care, are immoral.

The Committee believes that rationing of health care services by government is not a solution to the problem of scarce medical resources. Our health care is worth the cost, and we should maintain it. This approach will require an apolitical reassessment of state and federal priorities and resources, with maintaining and strengthening health services as a high priority. A social climate for rationing (or denying) medical care should not exist.

More and more, money is the scarce resource that determines the level, extent, and complexity of medical care for our citizens. Complete, modern medical care is a luxury that some believe we may not be able to afford.[1] In the United Kingdom, the National Health Service budget is decided on by the government. Part of the decision making process is based on competing claims for available funds between education, welfare, defense, transportation, housing, and other social needs. In the United States, the federal government and the states are increasingly seeking to limit or decrease their health care expenditures. The diagnosis related group (DRG) method of prospective payment for hospital care is one such effort to reduce costs of health care. Landmark legislation rationing health services to the poor has recently been adopted in Oregon.

Rationing of health care has been vigorously debated in the 1980s and will continue to be debated into the 1990s. A reduction in payments for health care affects the quality of patient care. This report examines medical, ethical, social, economic, political, religious, and philosophical aspects of the rationing of medical care and expensive medical resources.

TRIAGE

Triage is concerned with the rationing of scarce resources and is usually based on the discernment of those who are beyond treatment, those who are likely to survive without treatment, and those whose lives can in all probability be saved by prompt treatment. Triage becomes necessary when medical resources are limited.[2] The limitations may be in personnel, equipment, support services, or money. Two people in the desert with water sufficient for only one of them to survive pose a classic challenge in the allocation of a scarce resource, water. Two dying patients with medicine sufficient to save only one is another example of the dilemma of choosing "who shall live and who shall die." Thirty people on a sinking ship in the middle of the ocean with a single lifeboat sufficient for only ten people pose the same ethical dilemma because of the shortage of lifeboats.

HEMODIALYSIS: A CLASSIC EXAMPLE

The insufficient dialysis facilities in the United Kingdom continue to attract the attention of the media. The reason is simple: If a fatal condition can be effectively treated, failure to treat some patients means that they will die.[3] More people are allowed to die of chronic renal failure in the United Kingdom than in any comparable European country.[4]

Since dialysis is extremely expensive, fiscal justification for its widespread use is the expectation that many beneficiaries would, by virtue of the treatment, return to full employment and enjoy a markedly improved quality of life. With this expectation, the United States Congress legislated in 1973 that all patients with kidney failure who need hemodialysis or renal transplantation should have access to them, and that Medicare would assume the cost for the entire End Stage Renal Disease Program. However, most patients who now undergo dialysis in the United States are too old and too sick with other serious medical problems to be able to work.[5]

Before 1973, dialysis was a scarce resource, and its availability had to be rationed. Decisions as to who would

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receive it were made in two steps. First, rules of exclusion narrowed the number of potential recipients. Second, rules of selection provided a basis for choosing among the remaining patients. The reasons for exclusion included age, lack of ability to pay, low social worth of the patient, bias on the part of the physician, medical diagnosis (eg, diabetes), inability or unwillingness of the patient to travel to the site of dialysis, preference for a particular physician or hospital, psychological instability of the patient or family, and hospital orientation (eg, veterans' hospitals excluded non-veterans). A list of medical, economic, and psychosocial reasons was then used to select the patients who would receive dialysis from those remaining in the pool.

Each of these rules of exclusion and selection for allocating scarce resources can be criticized on moral grounds. Rescher[6] has suggested an approach that combines the exclusion and selection criteria into a single set of medical and social criteria, such as constituency (eg, children's hospitals exclude adults), progress of science, prospect of success, life expectancy, family role, potential future contributions, and past services rendered.

Selection on the basis of first-come, first-served seems to be the simplest method of allocating a scarce resource. However, ethical problems still arise. Should not the President of the United States or a brilliant scientist receive preference over a person with far less apparent social value? Should a mother of small children be dialyzed before a single person? Should a young person be dialyzed in preference to an old person? Practical necessity and the public conscience may require exceptions to the first-come, first-served standard.

These issues arise at the policy level as well. Moral questions pertain to intent as well as to process. Is it worthwhile to dialyze all patients with end stage renal disease? Should scarce financial and personal resources be used for a chronic dialysis program for social outcasts rather than for well-baby clinics, teenage pregnancy programs, hypertension screening and treatment activities, or other critically needed health care programs? What comes first and who comes first when resources are limited because funds are in short supply?

One reason that society has not elected to support fully the care of other groups of chronically ill people is that the End Stage Renal Disease Program has outgrown its original cost estimates and, for most patients, provided only a minimal quality of life. But who is to judge one's quality of life?[7] Patients believe the quality of their lives during its prolongation by dialysis to be higher than it seems to be to others.[5]

Funding of Organ Transplantation

Which is more valuable: to provide a very expensive liver transplant to an ailing child of indigent parents; or to use that same money for prenatal care for a large number of expectant mothers? When a congressional inquiry was launched in April 1983 by Albert Gore, Jr, to evaluate current methods of securing human organs for transplantation, the legislators found themselves confronted by an array of social, economic, medical, political, and ethical questions, such as, "Who shall live?" "Who shall pay?" and, "Who shall decide?"[8]

In 1978, the Medicare End Stage Renal Disease Program was amended to include provisions designed to increase the frequency of renal transplantation. In 1984, Congress passed the National Organ Transplant Act, which authorized grants to organ procurement agencies and established a national organ sharing system. The act also established a task force on organ transplantation to examine the medical, legal, ethical, economic, and social issues raised by the procurement of human organs. The task force prepared two reports.[9-10] In the Omnibus Budget Reconciliation Act of 1986, Congress added another incentive to renal transplantation by mandating Medicare coverage of immunosuppressive drugs for one year after the transplant. The effect of transplantation on the Medicare End Stage Renal Disease Program was recently reviewed by Eggers,[11] who concluded that the financial costs of maintaining patients with functioning kidney grafts are only one third of the costs for maintaining patients on dialysis. Furthermore, the quality of life is much better for transplant patients than for patients on dialysis. Thus, the best clinical practice for patients with end stage renal disease is also the least costly.

With the increasing success of organ transplantation, the question is frequently asked, how much should we as a nation spend for transplantation compared to other technologies? According to Perry and Chu,[12] the American public acknowledges the threat posed by escalating health care costs, but will not easily be persuaded about the value of delaying or denying beneficial new technologies such as organ transplantation. These authors conclude that the savings realized by containing spending on organ transplantation may never be steered toward more worthwhile causes.

In the spring of 1987, the Oregon legislature decided to curtail funding for organ transplantation. In a painful decision necessitated by lack of funds, Oregon noted that over the following two years, Medicaid could either extend its funding for basic health care to include about 1,500 persons not covered previously, or continue to fund a program of bone marrow, heart, liver, and pancreas transplantation for a projected 34 patients. Oregon decided on the former. Welch and Larson[13] chronicle the events leading to the decision, the public reaction, the state's response, and several lessons drawn from the experience in Oregon. These lessons include the following: financial resources for medical care are limited; the need for acute care is more visible to the public than is the need for preventive care; new medical therapies are more likely targets for cost containment than those already established; and new therapies must replace older, less effective ones if total health expenditures are to be held constant. The message from Oregon pertains to all public policies:

> Budgetary constraints are real, and they apply to health care. Physicians are welcome to join the debate over choices, but choices must be made. We must look beyond our own vested interests and no longer assume that medicine is any more important than schools, roads, safety, water, or other public programs. Policy makers need our views in order to establish sensible priorities for medical care. . .[13]

Rationing of Medical Care

The era of cost containment is here. Increasingly, federal and state governments are seeking to contain or reduce health care expenditures. This often means reduced benefits or services for patients, or increased out-of-pocket expenditures. Cost cutting by government has sparked an intense debate about its effect on the quality of medical care. Health care rationing is inextricably linked with cost containment. "The only way to cut costs is to deny benefits to some people or deny benefits for some diseases."[14] Some people give prenatal care, nutrition, immunizations, birth control, and abortion services a high priority for funding, whereas organ transplants and cosmetic surgery are given a low priority. Many health experts argue that the United States no longer has the financial resources to provide unlimited medical treatment for all those who need it. The only solution, some say, is to ration health care under clearly established rules and with community support.

The specter of denying medical benefits has provided the impetus for many suggestions on how to avoid rationing: reducing demand for hospital services, better use of outpatient and clinic services, emphasis on healthful life styles and illness prevention, and the acceptance of some limitations on the treatment of the elderly and the terminally ill. The latter suggestion, espoused by Callahan[15] in his controversial book, has aroused the anger of senior citizens, who feel that society should not set an age limit for health care.[16] Callahan argues that, "Medicine should be used for the further extension of the life of the aged, but only for the full achievement of a natural and fitting lifespan and thereafter for the relief of suffering."[15] Callahan also blames our inability to pay for "routine" health care on recently developed, extremely expensive technologies for diagnosis and treatment, such as magnetic resonance imaging and artificial hearts.

A recent Brookings Institution publication calls for explicit criteria for excluding patients for whom no ultimate benefit can be predicted.[17] The authors would not treat or would severely limit treatment for patients with "rapidly progressing fatal illness, a large proportion of patients for whom survival is extremely improbable, and patients for whom iatrogenic risks exceed the likelihood of potential benefits." They also propose eliminating do-not-resuscitate orders in favor of "positive consent" for cardiopulmonary resuscitation and other standard intensive care interventions before a patient is admitted to an intensive care unit (ICU). Such strict ICU admission criteria, argue the authors, are necessary to avert the rationing of intensive care.

Griffin and Thomasma[18] explore the allocation dilemma in pediatric critical care. They report a case in which physicians debated whether to treat a deeply comatose, critically injured five-year-old child or to withhold treatment to save societal resources. Although the patient was comatose for several months, during which time she continued to require intensive care, including a respirator and hyperalimentation, she recovered to the point that she is now a normally alert child, but one who has only minimal neurologic function below the level of C-4. Griffin and Thomasma feel that physicians must be protected from the dangerous dual role of determining allocation criteria and, at the same time, making decisions about what critical care to give, since such a role is intrinsically contradictory. They suggest that society should determine how much of its resources are to be invested in critical care, while physicians should continue to provide their patients with the best available care for the patients' medical needs.

La Puma et al[19] discuss the physician's role in resource allocation for the treatment of endocarditis. They point out that, "in the face of relatively scarce medical resources, physicians can maintain the integrity of their relationships with patients and with society by developing a professionally and ethically acceptable framework for decision making." These authors suggest that those with clinical judgment and a primary commitment to patient care are best equipped to articulate the ethical values relevant to allocation of resources. On the other hand, Morreim[20] asserts that every medical decision is now subject to scrutiny for its economic as well as its medical wisdom. Wray et al[21] discuss the process of deciding about withholding medical treatment from the severely demented patient because of costs.

Increasing efficiency, the elimination of waste, and the reduction of medical care costs are desirable goals, but rationing is surely not the answer. Since 1980, the United States Congress has cut nearly $20 billion from Medicare Part A, which pays hospitals, and nearly $14 billion from Medicare Part B, which pays physicians.[22] Several methods are used to ration hospital care, including an increase in the deductible amount by third party payers, the assignment of hospital payments according to diagnosis related groups (DRGs), and the limitation allowances for lengths of hospital stays. All these cost cutting schemes place an unfair burden on the poor and the elderly. "The longstanding principle of medical ethics that physicians should be strong advocates for their patients is seriously undercut by the rationing of hospital care."[23]

The imposition of expenditure targets by Congress is a newly proposed rationing device which puts the onus for rationing on doctors. This cost cutting plan, if enacted into law, would limit the money available to pay for Medicare services, no matter how necessary those services are. Physicians would be expected to stay within a limited national budget. If they did not, the target budget for all Medicare physician services the next year would be reduced. The American Medical Association is strongly opposed to expenditure targets, since they would undoubtedly lead to rationing of medical care. People may then be denied services because of age, or wait days or weeks for tests or surgery, as is now the case in the United Kingdom and in Canada. The American Medical Association has proposed other ways to control Medicare spending, ways that do not threaten necessary care.[24] The imposition of expenditure targets in the United States would lead to drastic changes in the health care system. Congress, states the AMA, should not approve an experiment that would adversely affect the nation's elderly.[25]

"Rationing can be summarized as a system of deliberate choices about the sharing of health care resources among persons (ie, who gets what care, and in what order of priority) on grounds that go beyond an individual patient's clinically defined needs; the criteria specifically include both comparative medical need and social equity." [26] In this framework, rationing is not identical to cost containment, but the latter can lead to the former.

POLITICAL CONSIDERATIONS

American values focus on the importance of the individual. They emphasize autonomy, self-determination, privacy, and justice for all. Individuals are free to act as long as they do not harm others. While these principles often overlap, they sometimes impede one another. Policy makers must allocate scarce resources fairly. Legislators must make difficult choices: whether to give more money to health and education or to allocate those funds to the arms race, housing, museums, and highways.

Over the last decade, efforts have been made to distribute health resources rationally, but actually, the distribution of health resources has never been determined by an objective measurement of need. The issue is more complicated than that. The government, with its limited monetary resources, attempts to fund a health care program that is beneficial to both poor and wealthy alike. American culture is predisposed to take advantage of high technology, and Americans believe in medicine's role as the invincible conquerer of disease. If policy makers consider this national orientation, they face the question, Is it ever politically prudent to ration care in order to permit the greatest number to benefit, at the cost of neglecting some? Two examples that illustrate this problem are organ transplants and the treatment of disabled neonates. These situations epitomize the way in which advanced but expensive medical technologies and medical knowledge can produce difficult ethical and political dilemmas.

Legislators attempt to satisfy needs rather than to fulfill all wishes. Acceptable legislation is that which benefits the greatest number of people and harms the least. This doctrine is known in politics as the fairness of markets.[27] Unfortunately, market theory is not totally commensurate with the cost-benefit principle. We have yet to put a price on the quality of a human life. It is estimated that by the year 2000, health care expenditures will exceed 15% of the gross national product (GNP), and that allocations of scarce resources are likely to force policy makers to make very difficult decisions.

When facing the size and complexity of the American health care system, it is of paramount importance that we understand that health professionals, government agencies, consumer organizations, and special interest groups interact with each other and sometimes have opposing priorities.

With expenditures projected to reach $2 trillion by the year 2000, the stakes are very high and the economic situation, more than the other major social issues, may well become the top priority in establishing health care policy. Hospital costs, physician reimbursement, nursing home care, the care of women and children, dental care, and the acute and chronic care of patients with the acquired immunodeficiency syndrome are bringing the health care establishment to the brink of catastrophic deficits. Despite the fact that 90% of the population has third party insurance of some type, 28% of the total costs of health care are still paid directly by patients. In addition, more than 25 million Americans do not have any type of insurance for health care, although most have access to health care. For these reasons new strategies are needed for allocating scarce medical resources—strategies that do not involve rationing medical care.

PHILOSOPHICAL CONSIDERATIONS

The problem of scarce medical resources arises in a number of different situations that are often confused with each other. Cases of absolute scarcity include heart, liver, and lung transplants, for which there is an absolute shortage of organs for transplantation. Temporary scarcity occurs in situations in which there is no absolute scarcity but, at times, resources are not available. For example, a hospital may have an 85% occupancy rate, but at certain times of the year the hospital may be fully occupied, causing a delay in obtaining a bed and thus competition for a bed by various parties. Geographic scarcity is the unavailability of a resource in certain places. For example, computerized axial tomographic (CAT) scans may not be available in rural areas, although sufficient CAT scanners are present in the country for everyone.

When a medical resource is absolutely scarce, a just formulation for its allocation is needed. However, there are different forms of justice. Aristotle distinguishes between corrective justice, which governs transactions between individuals, and distributive justice, which determines the manner in which the state should distribute the assets of the community.[28] Rescher[29] lists seven criteria of distributive justice that are commonly advocated: equal distribution; or distribution according to need, moral merit, effort, productive contribution, or requirements of the common good (social utility); or distribution according to the nature of one's service in terms of supply and demand. Vlastos[30] adds the criterion, "according to the agreement made." Perelman[31] notes that in practice, distribution or allocation is often made "according to rank."

How do these notions apply to the problem of scarce medical resources? How does one decide who is to be allocated the ICU bed or who is to receive the kidney transplant? Rank or social utility is often considered. Shatin[32] argues that scarce medical resources should be allocated on the basis of an individual's social worth. Basson agrees.[33] Other authors argue for a system of allocation based on utilitarian factors.[34]

Nozick[35] strongly criticizes the concept of distributive justice as intrinsically "unjust" and advocates corrective justice, based on the notion of an individual's claims or rights.[35] A person has the right not to donate his kidney for transplantation, or to specify that his kidney be given to his close relative. On the other hand, people often fail to exercise some rights in favor of other rights. When people donate blood to a blood bank, they usually allow the hospital to allocate the blood to the most needy patient; however, individuals donating platelets for a family member with leukemia can restrict the allocation of the platelets to that patient. The platelets are directed to the patient with the understanding that if not needed, they can be used for any patient who needs them. We respect the right of individuals to donate platelets to the person they choose in preference to someone more needy or more worthy. Hospitals encourage these "restricted donations" both for this reason, and because they know that otherwise donations of blood products would often not be made.

Some formulations of distributive justice can also be criticized because they violate other principles we normally associate with justice. Individual actions, motivations, and responsibilities seem to be unimportant in determining whether an individual's actions are just or unjust. Strict implementation of the rules of distributive justice has led to injustices against many individuals. Moreover, many of us feel that an individual's life is of infinite value. It is, therefore, unjust to remove a patient from an ICU merely because that ICU bed could be better used by another person whose social value is greater. One is not permitted to sacrifice one human life to save another.[36]

RELIGIOUS CONSIDERATIONS

Catholic tradition adheres to the principle of the sanctity of human life, understood in the context of life as a gift from the Creator, and to the belief that human beings are created in the image and likeness of the Creator (Genesis 1:27). This scriptural teaching asserts that humans are indeed the Creator's surrogates in the world as He commissioned them to subdue the earth and have dominion over it (Genesis 1:28). Having made the world for human beings, the Creator entrusts the world to them. This approach is clearly stated in the Vatican II teaching: "God intended the earth and all that it contains for the use of every man and people. Thus, as all men follow justice and unite in charity, created goods should abound for them in a reasonable basis."[37]

Some Catholic theologians argue that the basic equality of all must be recognized, since all human beings possess a rational soul and are created in the Creator's likeness, have the same nature and origin, and enjoy the same divine calling and destiny. Thus, "Every human being has the right to life, to bodily integrity, and to the means which are necessary and suitable for the proper development of life."[38] Human physical life is of infinite, though not of absolute, value, a principle recognizing the transcendence and absolute authority of the Creator.

In the allocation of resources, some Catholic theologians examine three dimensions of basic justice: commutative justice, distributive justice, and social justice. Commutative or corrective justice calls for fairness in all agreements and exchanges between individuals or private social groups. Distributive justice requires that the allocation of income, wealth, and power in society be evaluated in light of the effects on persons whose basic material needs are unmet. Social justice implies that persons have an obligation to be active and productive participants in the life of society, and that society has a duty to enable them to participate in this way.[39]

In the Judeo-Christian tradition, the justice of God is displayed in care for the poor, exemplified as the marginal, the neglected, the widows, and the orphans. The biblical narrative extensively reflects this bias toward the poor. In 1973, the Christian Medical Commission of the World Council of Churches applied this principle to the health care needs of the marginal members of society. "Whatever health services are available shall be equally available to all: Departures from equality of distribution are permissible only if those worse off are made better off." Their recommendations include a floor or minimum of health services for all, distribution according to need of available resources above the floor, distribution of services to the least favored and most disadvantaged when health care resources cannot be divided, and participation in the discussion about allocation by the people likely to receive health care.[40]

In Jewish tradition, a physician is given specific divine license to practice medicine. According to Maimonides and other codifiers of Jewish law, a physician is obligated to use his medical skills to heal the ill. Not only that, but the patient is obligated to care for his own health and life. Man does not have full title over his life or body; he is charged with preserving, dignifying, and hallowing that life. He must eat and drink to sustain himself, and must seek healing when he is ill.

Another cardinal principle in Judaism is that human life is of infinite value. The preservation of human life takes precedence over all Biblical commandments, with three exceptions: idolatry, murder, and forbidden sexual relations, such as incest. To preserve a human life, the Sabbath and even the Day of Atonement may be desecrated, and all other rules and laws, save the three exceptions, may be suspended.

The corollary of this principle is that one is prohibited from doing anything that might shorten a life even for a very brief time, since every moment of human life is also of infinite value. How does Judaism resolve the dilemma of two patients dying of kidney failure and only one available dialysis machine? Who takes precedence? How does one decide? Is one not condemning one patient to death by offering the dialysis machine to the other patient?

The Jewish sources that deal with the dilemma of allocating scarce medical resources are detailed elsewhere.[41] Rabbinical statements seem to suggest that qualitative distinctions can be made on the basis of social worth, personal dignity, or religious status. The rule that "one may not sacrifice one life to preserve another" seems to imply that no qualitative distinctions should be allowed. A person can will his organs for transplantation to save the lives of others because "his blood is no redder than theirs" (Pesachim 25b). Therefore, in the allocation of scarce medical resources, a lottery system or a first-come, first-served approach, or pure medical criteria, might be preferable in Judaism.

An approach based on medical criteria is suggested by at least one contemporary scholar, Rabbi Eliezer Waldenberg,[42] who states that a patient with a potential for cure takes precedence in the allocation of scarce medical resources over a patient whose illness would, at best, be controlled only temporarily. This rule, however, applies only when the incurable patient is passively neglected in favor of the curable patient. It is forbidden to terminate the therapy of the incurable patient so that the curable patient can be given the care.

So far as society goes, Judaism teaches that "thou shalt open thine hand unto thy brother, to the needy, and to thy poor, in thy land" (Deuteronomy 15:11). Does this sequence also apply to matters of life and death and to the allocation of scarce medical resources?

The infinite worth of man, a Judaic axiom already cited earlier, is compromised by limited or finite resources. Why should a resident physician have to decide which patient gets the intensive care bed? Why not create more intensive care beds rather than support museums? Why not build more dialysis machines rather than buy more tanks or battleships? These are political decisions. One may legitimately ask whether allocations and axioms affecting individuals apply equally to society's problems. Is society a separate being and more than the sum of its component parts? According to Rabbi Moshe Tendler,[43] the answer is strongly affirmative. Societal ethics are indeed different from individual ethics. Society or government is needed to prevent man from destroying his fellow man. Therefore, society is not bound by the same ethical principles that bind individuals, such as the infinite worth of man. Support for this view can be found in several Jewish writings.

The Talmud (Gittin 3:6) asserts that captives should not be redeemed for an exorbitant amount of money for two reasons: to discourage kidnappers, and to prevent society from becoming impoverished by having to divert its resources to the ransoming of captives. The resources of society are finite, and the needs of the members are infinite. Society cannot provide for all the needs of all the people all the time. In accordance with the Talmud, the Codes of Jewish law rule that society may not expend excessive money to redeem captives. However, a husband may, using his own resources, ransom his wife for any amount, and a father, similarly, may ransom his son or daughter for any amount. Societal ethics are different from individual ethics.

Society must be concerned about future generations, long range planning, and the long range effects of its actions. This is not so for individuals. The physician in the intensive care unit must make decisions that affect individual patients here and now. Two individuals in the desert with a single pitcher of water should share that water, even if it belongs to neither, because they have to be concerned with preserving their lives here and now. They may be rescued tomorrow. The allocation of a medicine or treatment to one of two patients when there is not enough for both is a decision necessary for the here-and-now care of one or both patients.

However, one cannot totally divorce individuals from their responsibility of considering future generations when allocating resources. This point is best depicted in the talmudic story of a righteous man called Choni who was journeying on the road and saw a man planting a carob tree (Taanit 23a). Choni asked him, "How long does it take for this tree to bear fruit?" The man replied, "Seventy years." Choni then asked him, "Are you certain that you will live another seventy years?" The man replied, "I found ready-grown carob trees in the world. As my forefathers planted these for me, so I too plant these for my children."

Judaism views each person as being of supreme and infinite value. It is the obligation of both individuals and of society to preserve, hallow, and dignify human life, and to care for the total needs of individual citizens in order to enable them to be healthy and productive members of society. This fundamental principle should help guide both physicians making life and death decisions at the bedside for individual patients, and governmental bodies deciding short term and long term health needs and priorities for the population as a whole.

CONCLUSIONS

With this eclectic review of the literature, the Committee on Bioethical Issues of the Medical Society of the State of New York concludes that the allocation of scarce resources is a complicated, multifaceted issue associated with many moral and political problems. Many systems have been proposed to distribute finite medical resources equitably and to make these decisions consonant with basic principles of ethics. No system is perfect; all have positive and negative attributes. The criteria used by an individual or a clinic or hospital department may differ from those used by a large institution or government.

If one is to allocate scarce medical resources wisely and fairly, one must take into consideration the broad scope of medical care. This not only includes the care of individual patients, both acute and chronic, but also public health, preventive medicine, health education, and research and development. Each of these facets of medicine must be considered both from the aspect of its own individual cost and long term benefit to individuals and society, and also from the point of view of how spending in one area may affect or jeopardize other areas. Physicians must take a leading role in explaining the value of medicine, in evaluating diagnostic methods and treatments, and in insuring the protection of the rights of individuals as well as the rights of society.

With the continuing development of costly technology, care must be taken to evaluate thoroughly the efficacy of new techniques prior to their widespread application. Scientific evaluation of the harm/benefit ratios of new techniques is often inadequate. Is it enough to determine that a therapy produces an improvement in the clinical condition, or must it also prolong life? We need critical analysis of therapeutic effectiveness as well as cost effectiveness. We must not confuse efficiency and economy. We need to assess the instances in which preventive care is no longer appropriate, and when therapies with excellent short term results but dubious long term outcomes are appropriate. Physicians must participate actively in scientific trials aimed at answering questions about efficiency and economy in the delivery of health care, and in the development of criteria for the use of costly treatments.

Palliative care and comfort care are among the most difficult treatments to address in any system of allocation. At what point should a therapy be withheld because it is too costly for a person with a limited life expectancy, even though it may improve the quality of life? If we could quantify the benefits of therapy, we might be able to answer these questions. Experience seems to be the best teacher. The provision of education to patients in the outpatient and clinic areas is important. The provision of continuing care experiences for medical students may better prepare the latter to make difficult decisions about allocation. Patients are becoming sophisticated consumers of health care. Doctors must be prepared to spend time addressing their concerns. Doctors must be prepared emotionally to give honest appraisals based on sound data regarding palliative and comfort care for the terminally ill as well as for therapy aimed at cure.

Physicians must address issues of cost. They must be aware of the cost engendered by any test or treatment. Physicians must question the cost effectiveness of their individual behavior. Information should be provided to physicians about economy in the care of patients. For certain highly technical therapies, for which volume is necessary for good research results, centers of excellence may be the best solution. For very costly diagnostic tests and treatments, resources need to be shared. Physicians must take responsibility in helping to control costs. Peer review can help establish priorities of care.

Medical research and development must be preserved by adequate funding and protection of individual creativity. Targeted research can be useful, but individual investigators pursuing their own unique ideas have been most productive throughout history. Training of physician-scientists has been greatly curtailed over the last decade. Do non-physician-scientists have the same perspective on clinical problems as physicians? Allocation of health care resources cannot overlook or minimize the need for excellent scientific research performed by physicians.

Viewing individual rights as of prime importance, the Committee on Bioethical Issues of the Medical Society of the State of New York believes that decisions regarding the allocation of scarce medical resources by hospitals and physicians should be made on medical grounds and not for social reasons. The essence of good medical practice is that the physician be an advocate for the health and safety of his or her patients. Physicians should not be required to endorse or to participate in patterns of allocation that are inherently unjust.

The doctor is and must always be the advocate for the patient, seeking for the patient treatment that can improve or prolong life. Although physicians must participate in planning and education regarding the allocation of health care resources, specific decisions to limit types of therapy to particular populations must be at a level above that of the individual doctor-patient relationship.

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Primary signet ring cell carcinoma of the uterine cervix

Ute M. Moll, MD; John C. Chumas, MD; William J. Mann, MD; Bruce Patsner, MD

The frequency of adenocarcinoma of the endocervix is approximately 10% of all cervical carcinomas, with rates as low as 5%[1] and as high as 16%[2] having been proposed. Recently, a sharp increase in the proportional frequency has been observed, and rates up to 34% are being reported in the literature,[3] possibly due to a relative decrease in squamous cell carcinoma of the cervix.[4] In a series of 203 patients with cervical adenocarcinoma, Hopkins et al[5] distinguished five histologic subtypes: endocervical (46%), adenosquamous (mixed) (33%), papillary (11%), clear cell (8%), and mucoid (4%). Signet ring adenocarcinoma is an unexpected pattern in the cervix, and its presence should raise the possibility of metastasis. We report a case of signet ring adenocarcinoma of the cervix which appears to be primary. While the impact of the histologic subtype on survival is still a matter of dispute, the distinction between a primary neoplasm and a metastasis to the endocervix is eminently important for treatment and prognosis.

Case Report

A 50-year-old woman, gravida 4 para 3, presented with a two-month history of postcoital vaginal bleeding and menometrorrhagia. Tissue obtained by a fractional curettage showed a poorly differentiated carcinoma with focal signet ring features in the endocervix and with invasion of endometrial stroma. Based on the signet ring features, evaluation for an extragenital tumor was performed prior to definitive treatment. The patient had no history of gastrointestinal symptoms. An upper gastrointestinal study showed a normal mucosal pattern and peristalsis, and a barium enema demonstrated diverticulosis. Sigmoidoscopy performed before surgery was negative. Intravenous pyelograms revealed a pelvic mass, representing an enlarged uterus with compression of the left ureter but no obstruction. A breast examination and mammograph were within normal limits. The patient had never received any previous radiotherapy.

The patient underwent a radical hysterectomy with bilateral salpingo-oophorectomy and lymph node dissection. During the operation, a thorough examination of the stomach, intestinal tract, pancreas, and urinary bladder failed to reveal evidence of an extrauterine primary cancer. The patient received postoperative whole pelvic irradiation. After an interval of eight months, she presented again with signs of urinary obstruction and multiple bone metastases. Repeat radiologic studies for a breast or gastrointestinal tumor were negative. She died following progression of the tumor ten months after the initial diagnosis. Permission for autopsy was refused.

Pathologic Findings. The enlarged uterus weighed 300 g. The endocervical canal was completely obliterated by a firm, tan, tumoral mass which extended deep into the cervical walls and involved the entire posterior lip of the ectocervix. The endometrial cavity was filled with blood. The right ovary measured 4 × 5 × 1.5 cm. In addition to a simple cyst with a flat smooth lining, sections revealed multiple tan, soft nodules ranging from 2 mm to 5 mm within the stroma. Some of them were present at the surface.

On histologic analysis, the majority of the endocervical tumor consisted of poorly differentiated infiltrating carcinoma growing in sheets and trabeculae. The tumor cells had vacuolated cytoplasm and small, angulated nuclei which were often crescentic and displaced to the periphery of the cell (Fig 1). A single large intracytoplasmic vacuole occupied the cytoplasm of most cells. The tumor cells were uniformly positive by the periodic acid-Schiff reaction (diastase stable). Mucicarmine and alcian blue (pH 2.5) gave variably positive results. Small polypoid regions of the tumor showed disordered, but readily recognizable glandular differentiation (Fig 2) including replacement of native endocervical glands by neoplastic cells and focal microinvasion (Fig 3).

The ectocervix was covered by an atrophic squamous epithelium without atypia. The endometrial stroma and the myometrium including subserosal lymphatics as well as one out of two para-aortic lymph nodes and a small serosal plaque in the cul-de-sac were infiltrated by tumor. None of 50 sections showed any squamous component. The right ovary showed multiple nodules of metastatic adenocarcinoma consisting of a pure population of well-developed signet ring cells (Fig 4). The left ovary and both fallopian tubes were free of tumor. Metastatic carcinoma was present in one of two para-aortic and one of seven left pelvic lymph nodes. Although the vaginal margin was free of tumor, vaginal lymphatics were permeated by carcinoma.

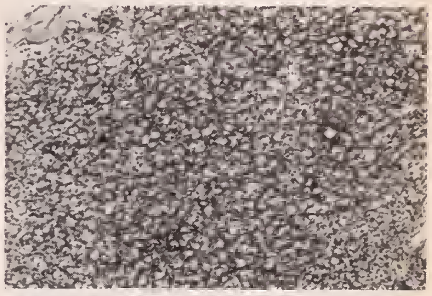
FIGURE 1. Endocervical carcinoma composed of sheets and files of cells with vacuolated cytoplasm and signet ring forms (original magnification × 100).

Discussion

Primary endocervical adenocarcinoma with true signet ring differentiation is unexpected. Although the existence of a signet ring subtype has occasionally appeared in histologic classifications,[6-8] we were unable to identify a

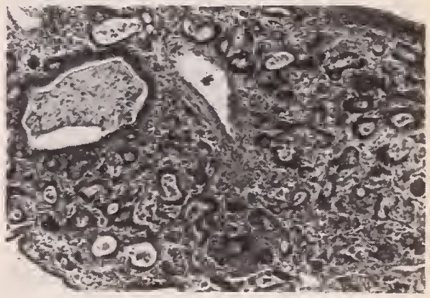
FIGURE 2. Gland forming component of endocervical tumor. Area of endocervical type differentiation (original magnification × 50).

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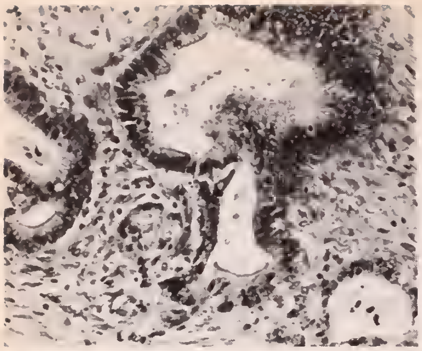

FIGURE 3. Glandular component with in-situ carcinoma and early stromal invasion (original magnification × 150).

fully documented case in the literature. For example, a recent study of 203 cases of uterine cervical adenocarcinoma does not indicate an example of signet ring carcinoma.[5]

Mucin-containing cells are not uncommon in cervical carcinomas with squamous differentiation,[9-11] and such cells may have a signet ring morphology.[9,10] De la Vega[12] described a case of signet ring cell carcinoma of the cervix in a 52-year-old woman, which arose from an anaplastic carcinoma after irradiation therapy. However, that tumor was negative for all mucin-detecting histochemical stains including periodic acid-Schiff after diastase digestion, mucicarmine, and alcian blue, which led the author to conclude that the signet ring morphology was the result of irradiation and might represent a degenerative change rather than true adenocarcinoma.

Signet ring carcinomas in the female genital tract are most commonly seen in the ovary and are nearly always metastatic, usually from the stomach, but also from the breast, colon, or appendix, with other sites being exceedingly rare.[13]

Regarding metastases to the uterus more specifically, Kumar and Hart[14] found that the majority of extragenital carcinomas metastasizing to the uterine corpus were from breast, colon, stomach, and pancreas. Carcinomas from any of these sites may have signet ring morphology and, indeed, Kumar and Hart[14] illustrated a case of metastatic signet ring carcinoma from the breast.

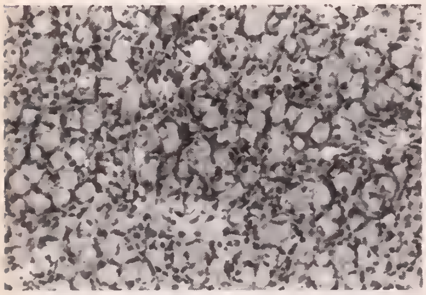

FIGURE 4. Signet ring metastasis in ovary (original magnification × 250).

Likewise, extragenital carcinomas that metastasize to the uterine cervix are most commonly from the colon,[15] stomach,[16,17] and breast.[17]

As signs and symptoms relating to a primary carcinoma may remain clinically silent until months or years after the presentation of metastatic disease, the diagnosis of an unusual histologic tumor type as primary should be made with caution. Several clinicopathologic features indicate that the carcinoma reported here is primary in the cervix. There were no symptoms relating to the gastrointestinal tract, and radiologic studies were normal, including repeat studies performed ten months after the diagnosis of the cervical cancer. The gastrointestinal tract was free of tumor by palpation at the time of hysterectomy. The breasts were normal to palpation and by mammography. Based on histologic findings, the tumor included areas of adenocarcinoma in-situ, a feature that suggests a local carcinogenic field effect, and which has been used to support cervical origin of adenocarcinomas when that determination is uncertain.[17,18] Finally, the tumor exhibited the biological behavior of cervical carcinoma, with lateral extension causing ureteral obstruction and hydronephrosis. Nonetheless, the unlikely possibility of an occult (eg, gastric) neoplasm cannot be completely excluded in the absence of an autopsy. Breast and colon as primary sites are more confidently excluded by the diagnostic studies that were performed on this patient.

The presence of signet ring carcinoma in the ovary merits comment. The distribution of multiple tumor nodules in the ovary with pure signet ring histology is highly suggestive of a metastatic lesion. The observation that cervical adenocarcinomas may have a propensity to metastasize to the ovaries has recently been emphasized[19-21] and is illustrated in this case.

While the tumor reported here was poorly differentiated, the histologic subtype may be unimportant in assessing survival.[5] However, the determination of whether a tumor is primary or metastatic has profound prognostic implications. This case report should serve to heighten awareness that cervical carcinomas with signet ring morphology might be primary rather than metastatic.

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Intestinal obstruction secondary to bezoars

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Small bowel obstruction secondary to bezoars has been recognized for many years. DeBakey and Oschner[1] in 1938 reported several hundred cases of bezoars, with a high percentage becoming obstructed in their attempted passage through the small bowel. Mangold et al,[2] Cooper and Tracey,[3] Rumley et al,[4] Storck et al,[5] and others have all recorded cases in which obstruction has occurred due to the failure of passage of a bezoar through the small intestines.

The composition of bezoars greatly varies according to the curious habits of some people who swallow or ingest peculiar substances or strange combinations thereof. Hair, metal objects, seeds, vegetables, and cereals, for example, are all capable of agglutinating in the stomach to form bezoars, some of which may remain there indefinitely or may eventually pass through the entire alimentary tract. Others exit the stomach only to become lodged in the small bowel. While in former years the diagnosis of bezoars located in the stomach was made quite accurately based on radiologic findings, the advent of endoscopy has made identification easier, and this technique reveals more precise information on the composition of bezoars.

The two patients reported here are interesting because of the odd composition of their bezoars and the circumstances that prevented their passage through the intestinal tract.

CASE REPORTS

Case 1. An alert, well-oriented 75-year-old man was admitted to the Cabrini Medical Center in September 1989 because of diffuse abdominal distention, pain, nausea, and vomiting of three days' duration. He gave no history of having ingested anything out of the ordinary or of having choked on anything that he had swallowed. His medical history was significant for an appendectomy for a perforated appendix approximately two years previously. His recovery from the surgery had been uneventful, and there had been no remarkable sequelae.

Physical examination showed the patient to be markedly dehydrated. Blood chemistry studies, however, were in the near-normal range. Abdominal examination revealed moderate distention with diffuse tenderness and rebound on palpation. Auscultation demonstrated diminished bowel sounds. No masses were palpable on abdominal or rectal examination. The stool was negative for blood.

Plain abdominal radiographs showed three air-fluid levels in the small intestines, but there was no evidence of a foreign body (Fig 1).

A diagnosis of small bowel obstruction was made, and, after fluid replacement and the insertion of a Swan-Ganz catheter, the patient was taken to the operating room for exploratory laparotomy.

At operation, many adhesions between dilated loops of ileum were found in the right lower quadrant, and these were lysed. A foreign body was palpated within the lumen of the ileum at the point of obstruction. Attempts to advance the foreign body so as to pass it through the ileocecal valve were unsuccessful. Accordingly, an enterotomy was carried out over the mass, and an undigested wedge of lemon measuring 7 × 4 × 1 cm was extracted.

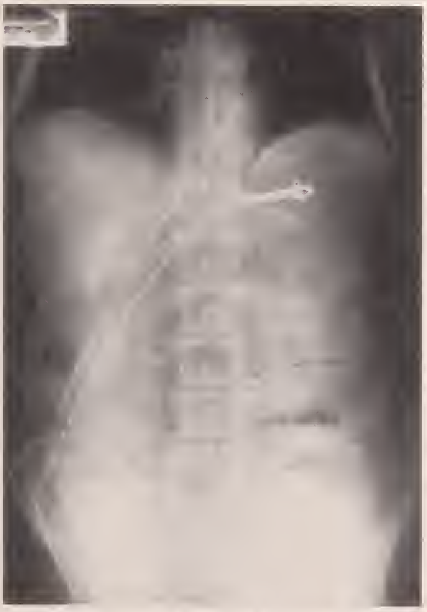

FIGURE 1. Plain film of the abdomen showing air-fluid levels secondary to small bowel obstruction.

Case 2. A 45-year-old woman was admitted to Cabrini Medical Center in September 1989 because of the acute onset of periumbilical colic-like pain in the abdomen accompanied by several episodes of vomiting and diarrhea. Her medical history was essentially negative. Her surgical history revealed that she had undergone a myomectomy approximately five years previously, complicated by the formation of many adhesions and necessitating a laparotomy for small bowel obstruction three years later.

The patient stated that to combat weight gain she had recently gone on a high fiber, low calorie diet. It consisted, among other things, of eating three slices of "wood pulp bread" three times daily. The packaging claimed that the bread was high fiber and low calorie but did not actually contain wood pulp. (The product is no longer on the market.) In order to diminish her appetite, the patient said that she chewed approximately three slices of chewing gum a day, and after finishing with the chewing gum, she would swallow it. This practice went on for three days at a time, with intervals of several days between resumption of the practice.

On admission, the patient was alert and cooperative but in severe abdominal pain. Her vital signs were within normal limits. Abdominal examination revealed moderate distention with tenderness on palpation in the periumbilical area. No masses were palpable. High pitched bowel sounds were present. Rectal examination was negative. Flat plate roentgenograms of the abdomen showed small bowel distention but no foreign body.

A diagnosis of small bowel obstruction was made, and the patient was taken to the operating room. At laparotomy, markedly dilated loops of distal jejunum and proximal ileum, along with many adhesions, were found. The adhesions were lysed. A small area of torsion was noted and a bezoar, measuring approximately 20 × 2 cm in diameter, was palpated through the dilated bowel wall at the point of obstruction. After the obstruction had been released through the cutting of adhesions, it was felt that the bezoar, because of its semisolid consistency, could be advanced beyond the ileocecal

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valve without the need for an enterotomy for its extraction. This maneuver was successfully carried out.

The patient made an uneventful recovery, and on the fourth postoperative day passed the bezoar per rectum. It was found, not unexpectedly, to be a conglomeration of undigested "wood pulp bread" intertwined with masses and strands of chewing gum.

DISCUSSION

Bezoar obstruction of the small bowel has been reported by many investigators within recent years.[1-11] The majority of the reports deal with an obstruction caused by the large size of the offending foreign body. A second cause of bezoar-induced small bowel obstruction mentioned frequently in the literature is atony of the intestine secondary to previous gastric surgery. In this connection, Moriel et al[11] reported on 72 patients with phytobezoar obstruction, all of whom had undergone truncal vagotomy plus pyloroplasty or partial gastrectomy for peptic ulcer disease. A third cause of bezoar obstruction, often ignored in the literature, is the presence of adhesions in the area of the small bowel secondary to previous abdominal surgery. This report primarily addresses this last group of patients.

In diagnosing the etiology of small bowel obstruction, one should be certain to take a careful dietary history. With the increasing popularity of foods containing large quantities of nonabsorbable fiber, one should be alert to the possible formation of bezoars.

Plain radiographs of the abdomen often do not reveal a bezoar, but Verstendig et al[8] were able to diagnose seven jejunal bezoars in nine patients by the administration of water-soluble iodinated contrast material. Dreznik[10] demonstrated bezoar ileus in seven patients who had been given a water-soluble contrast meal. These roentgenologic studies are advisable provided they do not promote long delays in starting therapeutic measures to relieve the obstruction.

It is well recognized that one of the leading causes of small bowel obstruction is adhesions from previous surgery. In a series of 289 patients, Mucha[6] reported that nearly 50% of their cases were secondary to postoperative adhesions. Brolin[12] stated that postoperative adhesions accounted for 79% of the 91 cases of small bowel obstruction admitted to his insititution.

In the two cases described in this report, the bezoars were of such size and composition that one might have expected them to have traversed the small bowel without difficulty. However, each patient had undergone previous abdominal surgery resulting in adhesions and kinking of the ileum of insufficient proportions to have routinely caused obstruction. Obstruction only took place when the bezoars arrived in the area.

The treatment of acute small bowel obstruction is so well known that it does not require detailed discussion here. Suffice it to say that if the obstruction cannot be relieved by conservative means within 24 hours, surgical intervention is indicated. It is especially important to intervene if bezoar obstruction is suspected, because many bezoars are composed of metallic materials that have a tendency to perforate the bowel.

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Metastatic cancer of the spinal intradural extramedullary space

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The most common primary tumors of the spinal canal arise in the intradural extramedullary space.[1] Metastatic tumors of the spinal column, on the other hand, are almost invariably extradural. Intradural extension of these metastases is uncommon, and an intradural extramedullary primary location of a metastatic deposit is rare.[2-5] This report presents two cases of metastases to the spinal intradural extramedullary space, along with an extensive review of the literature.

CASE REPORTS

Case 1. A 44-year-old woman presented with severe low back pain of six weeks' duration. The pain was sharp and radiated to the right leg initially. Later it involved the left leg with equal intensity. Her medical history was significant for a left radical mastectomy for carcinoma of the breast eight years earlier. This was followed by oophorectomy and chemotherapy as indicated for the stage of her disease. One and a half years prior to the present admission, she received further chemotherapy for recurrent hepatic disease. At that time she also had skeletal lesions on her left iliac bone and vertebral bodies at T7 and T10. She had had a partial response, with decrease in the size of her liver and resolution of all osteolytic lesions. She had remained asymptomatic since that time.

On physical examination, the patient was alert and oriented. A complete neurologic

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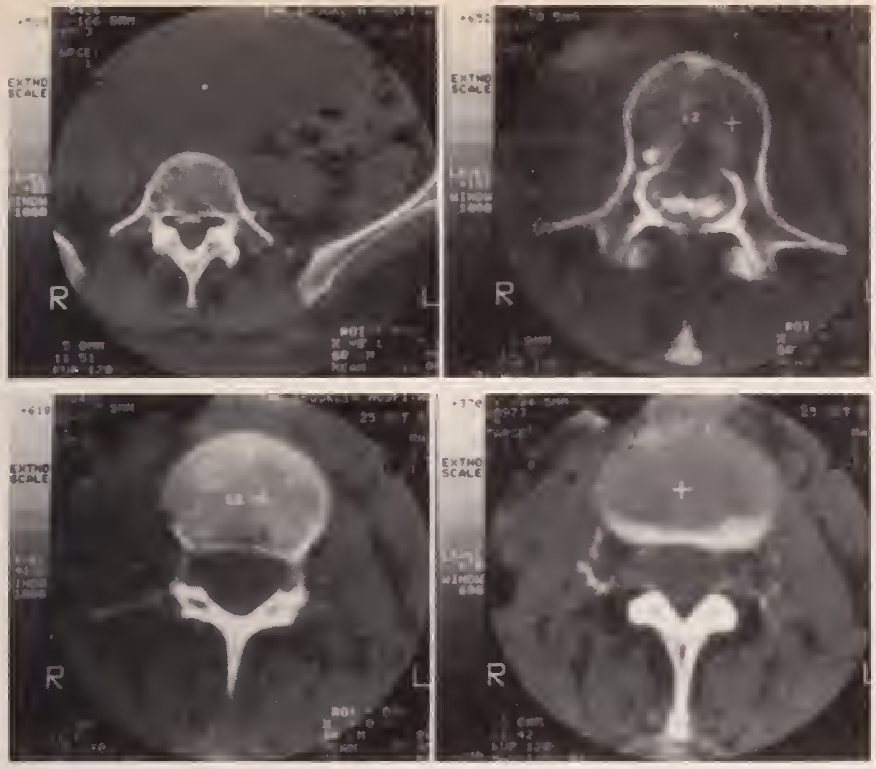

FIGURE 1. Post-myelogram CT scan (Case 1).

examination showed normal cranial nerves. Power and reflexes in the upper extremities were unremarkable. She had severe pain on movement of both lower extremities, which she was able to raise against gravity for a short time only. Sensory examination revealed a hypersensitive reaction to pinprick in both lower extremities. Her deep tendon reflexes were equivocal.

A post-myelogram computed tomographic (CT) scan demonstrated the presence of an intradural soft tissue mass at the level of L2, producing cauda equina compression (Fig 1). There were no signs of bone destruction. A total myelogram confirmed that there was a complete block of the contrast column at the level of the L2 vertebra (Fig 2).

Shortly after admission, the patient showed rapid progression of pain and weakness, necessitating emergency surgical decompression. The dura was opened at the L2 level, and a friable hemorrhagic mass was found measuring 3 × 3 × 2 cm. It was firmly adherent to the nerve roots and was resected using an operative microscope. Histologic evaluation confirmed that the tumor was metastatic adenocarcinoma, similar to her previous primary breast tumor.

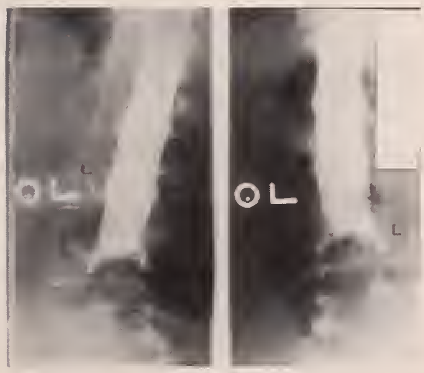

FIGURE 2. Myelogram (Case 2).

The patient experienced significant improvement after the surgery. Her lower limb strength improved and the preoperative back pain as well as radiculopathy disappeared almost immediately. Two and a half weeks after the surgery, the patient was walking without assistance and palliative radiotherapy was initiated.

Case 2. A 54-year-old woman presented with a two-day history of a sudden onset of inability to walk. She was known to have diabetes and hypertension and to have smoked two packs of cigarettes per day for 40 years. Her medical history was significant for a posterior cranial fossa tumor, eight months earlier, that on excision turned out to be metastatic adenocarcinoma of unknown origin. A complete work-up including CT scans of the chest, abdomen, and pelvis; a mammogram; and a barium enema failed to reveal the primary lesion.

On physical examination, the patient was alert and oriented. Neurologic examination revealed bilateral lower extremity weakness with powers of $\frac{1}{5}$ and $\frac{2}{5}$ in the right and left lower extremities, respectively. There were bilateral Babinski toe signs and bilateral hyperreflexia of the lower extremities. Sensory level to pinprick was at T1. Her cranial nerve and upper extremity examinations were unremarkable. An emergency myelogram revealed a high-grade obstruction at the T1 level from possible extrinsic compression (Fig 3).

The patient underwent emergency decompression. At surgery, three small friable gray brown masses were found within the dura attached to the arachnoid matter at the C_6-C_7, C_7-T_1, and T_1-T_2 levels. The lesions were excised using an operative microscope, and decompressive laminectomies were performed.

Following the surgery, the patient had gradual return of power, and she was able to walk with assistance. Repeat CT scans of the chest, abdomen, pelvis, and head demonstrated a 1-$\frac{1}{2}$-cm nodule in the left upper lobe of the lung with the suggestion of hilar adenopathy. The liver also showed multiple low density lesions. She received a course of radiation to the spine, with a short course of steroid therapy. Pathologic evaluation showed a poorly differentiated adenocarcinoma, with histologic findings similar to those for the lesion previously removed from the posterior fossa.

DISCUSSION

Based on findings, most spinal tumors present with back pain and signs and symptoms of progressive spinal cord or nerve root compression.[6,7] In their study, Perrin et al[2] found that clear-cut sensory loss and sphincter dysfunction were present twice as often with intradural as with extradural metastasis. The second patient discussed here was unusual, in that she presented

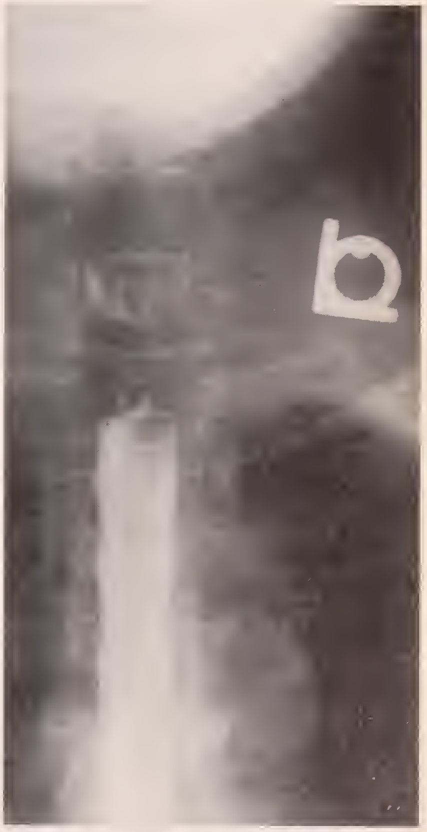

FIGURE 3. Myelogram (Case 2).

with sudden onset of paraparesis as the sole symptom, unaccompanied by back pain.

Plain films of the spine are typically normal in patients with intradural lesions.[2] Myelography and CT scans may help in the differentiation of intradural lesions from vertebral lesions.[3,5,7-9] Myelography may show a complete block with cap-like deformity.[10] Magnetic resonance imaging (MRI) may also be valuable,[11] but often the exact diagnosis is made only at surgery.[3] A recently developed diagnostic tool is gadolinium-DTPA enhanced magnetic resonance imaging. Its advantages include detection of small iso-intense intramedullary tumors and the ability to differentiate tumor from cord syrinx, gliosis, or arachnoid scarring.[12] MRI with flow compensation and cardiac gating may help eliminate intrinsic artifacts from cardiac pulsation, respiration, and cerebrospinal fluid flow.[1]

The most common location of these tumors seems to be the cervicothoracic spine[6]; but similar lesions may also occur in the lumbosacral spine.[2,3,7] The most common primary lesion is carcinoma of the breast, but metastatic tumors have been reported from lung, adrenal, colon, kidney, and ovary cancers, as well as melanoma and Hodgkin's disease.[5-7,13]

Intraoperative spinal sonography may be a useful adjunct in defining the location and extent of these lesions.[14] Perrin et al[2] have suggested, based on their experience, that since most of these patients have associated intracranial lesions, total neuraxial radiation and chemotherapy should be considered as adjunctive treatment.

Recovery of neurologic function in paraplegic patients is often unsatisfactory.[2,15] In both of the patients presented here, however, a good functional result was obtained. They were ambulatory on discharge from the hospital. We believe that even though the overall prognosis is grave, aggressive decompression may be warranted, since it may afford excellent palliative treatment and the opportunity for accurate tissue diagnosis.

Intradural extramedullary spinal metastasis from non-neuraxial tumors is a rare occurrence[2]—only three out of 100 reported cases of spinal metastases.[15] Most of the reports in the literature are isolated case reports, such as this one.[3-5,8,16] Many of these patients have also had secondary brain tumors, which gives rise to the speculation that the spinal metastases may indeed be tertiary deposits due to spread via the cerebrospinal fluid.[2,17] The second patient presented here had had previous removal of a similar tumor from the posterior cranial fossa. Her spinal lesions may have represented drop metastasis from this lesion. Other mechanisms proposed include spread along perineural lymphatics,[8,18,19] by way of the vertebral venous systems,[5,20] and transdural invasion,[4,5,16] which is unusual.

The reasons given for the rarity of these lesions as compared with cerebral metastases include the relative difference in the mass of the brain compared with the spinal cord, and the fact that the blood supply to the cord consists of small, late arterial divisions and small veins.[4] Furthermore, the dura almost always forms an effective barrier to the spread of extradural lesions.[7]

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LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Health and health care challenges for New York City in the 1990s

TO THE EDITOR: To the list of challenges described in Dr Imperato's informative commentary[1] one might add a truly pernicious challenge: the politicization and distortion of public health measures by the appearance of the human immunodeficiency virus (HIV). This has occurred at both the New York City and New York State departments of health. The implications of this may only be fully assessed by some future historian who may conclude that the Orwellian Era of Medicine began in the decades of the 1980s and 1990s in New York State. The essential facts are as follows:

The state has decided that the sexually transmitted HIV virus is not a sexually transmitted disease (STD). It has suspended normal public health measures in the hope of supposedly better containing the epidemic. The appointment of a New York City Commissioner of Health was challenged by a miniscule but vocal minority. Their challenge was based on objection to the possibility that the appointee would follow normal and usual measures to combat the spread of the HIV virus.

If our military were only issued blank cartridges or accountants forbidden trial balances by fiat, what would the result be? Should the sensibilities of political factions alter case finding and treatment? Distortions owing to the need to contain costs are perhaps understandable, but outright suppression of medical procedures beneficial to and protective of the public's health should not be tolerated. It may already be too late to halt the politicization of public health and its parent, medicine, but it need not go unchallenged.

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1. Imperato PJ: The health and health care challenges for New York City in the 1990s. *NY State J Med* 1990; 90:51-52.

The New York State Department of Health's Part 405 regulations

TO THE EDITOR: The New York State Department of Health's (DOH) Part 405 regulations are part of its efforts to micromanage health care in New York. They will cost hospitals some $475 million a year.[1] Despite promises to the contrary, only a fraction of these costs will be reimbursed to the hospitals.

The Part 405 regulations are a striking example of misplaced priorities by a government bureaucracy. At a time when most of New York's hospitals are experiencing enormous operating losses each year, and suffering shortages of nurses, technicians, and other persons needed for direct patient care, the DOH has chosen to impose numerous costly new personnel requirements and practices. These will certainly detract from our ability to take care of patients.

In an effort to prevent doctors, nurses, hospital employees, volunteers, etc, from injuring patients, the Health Department Part 405 regulations now mandate an annual physical examination for every health care provider. In the case of Schenectady's Ellis Hospital, this numbers 1,700 persons. The hospitals are also required to review and reassess these files at least once a year, at great cost, without reimbursement from the DOH. There presently exists a legal responsibility for all physicians to report behavior of other physicians and hospital personnel which they regard as suggesting impairment of their ability to take care of patients, by reason of drug or alcohol abuse or ill health. The Part 405 regulations add an undefined physical examination which is supposed to accomplish the same objective. Such an examination could not possibly be effective. Many of us believe that its real purpose is to gain control over the major privilege of medical licensure—the right to care for patients in the hospital—while forcing the hospital to become the health department's enforcer. Presently, licensing powers rest with the state department of education. Moreover, access to such confidential health files by many levels of hospital as well as DOH personnel virtually places them in the public domain.

The Part 405 regulations further require hospitals to provide language interpreters and persons "skilled in communicating with vision and hearing impaired individuals." The former must be regularly available for non-English speaking groups comprising more than 1% of the hospital's service area population. All must be made available to inpatients and outpatients within ten minutes of a request made to the hospital by the patient, patient's family, or provider of medical care. In short, these regulations require many hospitals in New York State to be prepared to produce within minutes in their emergency rooms at 3:00 AM a Korean or Spanish translator, or a person who can sign for the deaf. Failure to do so would result in facing the consequences. And far too frequently in the past, the consequences have been draconian penalties levied by the DOH.

The failure of the DOH to provide reimbursement for many of the services it mandates means that other, vital hospital services and programs will have to be terminated or curtailed to divert funds to comply with the requirements. Patient care will suffer, and further injury will be inflicted on many of New York's hospitals, a large majority of which are already in precarious financial condition.

Schenectady's Ellis Hospital, for example, had operating losses totalling more than $6 million during the past ten years (G. B. Serrill, personal communication, August 16, 1989). Two years ago, it had to close its excellent alcoholism unit at a time when the statewide demand for such treatment programs far exceeds the supply. More recently, its immensely useful poison control center was shut down, also for financial reasons. Schenectady's other community hospital, St Clare's, lost $2.2 million over the past ten years (J. G. Stewart, personal communication, February 23, 1990). Albany Medical Center had a deficit of $14 million in the fiscal year ending June 30, 1989, alone,[2] and is facing stringent austerity measures which threaten patient care.

Two great hospitals in New York City, Columbia-Presbyterian and Mount Sinai, were recently forced to close outpatient pharmacies which were subsidizing medicines for the poor at a cost to them of more than $1 million a year.[3] Presbyterian Hospital, which had operating losses of $25 million in 1988, had to lay off 300 staff members in a time of severe personnel shortages.[3] Characteristically, Health Commissioner David Axelrod's compassionate comments about the pharmacy closures were, "It is unfortunate that these major hospitals have chosen to terminate primary care services for the poor, compared to other cost-cutting actions they could take."[3]

An additional consequence of the DOH's new regulations is that they greatly increase a hospital's exposure to malpractice liability. This occurs at a time when hospitals are already burdened enormously by malpractice insurance costs. This will result in the inability of many hospitals to comply with these state mandates for financial reasons, as well as the nonavailability of necessary personnel.

The Hospital Association of New York State (HANYS) has filed a lawsuit against the entire Part 405 regulations. Schenectady physicians like myself overwhelmingly support the HANYS position. On June 30, 1990, with the financial support of more than 150 Schenectady physicians and several dozen physicians from other counties, I filed suit in state supreme court against the DOH over the physical examination provision of Part 405, which is the only provision for which we have legal standing to sue. But the suit has a much broader purpose: to help forge a coalition of doctors, nurses, hospital personnel, and patients to obtain legislative relief.

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1. Sisto D: Hospital association files litigation contesting Part 405. *Hospital News, Capital District*, May 1989; pp 7-8.
2. *Albany Medical Center News Release*, December 15, 1989.
3. French HW: 2 Manhattan hospitals shut pharmacies used by needy. *NY Times*, March 9, 1989, p 9.

Gender and sex

TO THE EDITOR: "Gender" is a grammatical term. "Sex" is the correct biological term. Even in English inanimate objects have gender—albeit neuter. When we come to French, German, Italian, Spanish, and the like, gender becomes significant. As a matter of fact, it becomes a marker of the pupil's knowledge of the language. We should insist upon correct terminology.

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In reply. Dr Kolisch raises an interesting point. At one time the word "gender" was restricted to grammar and the word "sex" to biology. However, this is no longer the case. "Gender" is now interchangeably used with "sex" in the biological field, thus blurring distinction. Recent English language dictionaries define gender firstly as sex and secondly as "any of two or more subclasses within a grammatical class of a language (such as noun, pronoun, adjective or verb) that are partly arbitrary but also based on distinguishable characteristics . . . and that determine agreement with and selection of other words or grammatical forms."[1,2]

Noah Webster would no doubt have preferred retention of the distinction. However, common usage is a powerful force that is constantly changing the English language.

PASCAL JAMES IMPERATO, MD
Editor

1. *Webster's New International Dictionary of the English Language*. Springfield, Mass, G&C Merriam Co, 1971, p 944.
2. *Webster's Ninth New Collegiate Dictionary*. Springfield, Mass, Merriam Webster Inc, 1985, p 510.

Cross-dressing successfully treated with fluoxetine

TO THE EDITOR: Cross-dressing is part of the group of paraphilias. Patients with these disorders have much in common with patients suffering from obsessive-compulsive disorder (OCD) though they are differentiated by the fact that their obsessive thoughts and compulsive actions are not ego-alien to them.

OCD has recently been thought to be a psychobiological illness by such investigators as Lieberman[1] and Zohar and Insel.[2] This view is further supported by several studies in which obsessive-compulsive symptoms have yielded to treatment with serotonin-specific second-generation antidepressants.[3-5]

In this report, a case of cross-dressing in a 60-year-old married man is described, in whom treatment with fluoxetine hydrochloride resulted in marked improvement. While other nonpsychopharmacologic variables such as the effects of life circumstance, the passage of time, and some intercurrent psychotherapy prevent attribution of success solely to the use of the drug, the outcome is of interest. It was not deemed ethically advisable to discontinue the drug to test for symptomatic relapse. A traditional randomized drug trial, or a group outcome study would serve to determine whether this is a reproducible phenomenon or an isolated occurrence.

Case Report. A 60-year-old married man, who had recently retired from business, presented himself for treatment upon the urging of his wife. He had recently appeared to her in women's clothing, complete with pocketbook and high heeled shoes, declaring that the woman inside of him wanted to come out. If prevented from donning female clothing, he became extremely anxious. He wanted to go shopping like a woman and be accepted by other women as their equal. Over the last four years, the patient had taken a dress from his wife, as well as shoes, lingerie, and accessories, but kept this secret until recently. During the last few weeks preceding evaluation, his obsessive thinking had escalated into a compulsion to appear openly as a woman. His wife had distanced herself from him, becoming depressed and suicidal over their increasingly deteriorating relationship. The couple described their sex life as gratifying until recently. The patient's relationship with his mother was unusually close and a source of friction in the marriage.

In his boyhood, the patient had shown no atypical genderal behavior, thus differentiating his development from those who take on a homosexual orientation.[6] However, from nine or ten years of age, he had wanted to be a girl for periods of months or years, each time putting the idea aside. His mother, apparently a mentally disturbed woman, had often told him he had the body of a girl, slim and graceful. Her clothing had been always strewn about and he was encouraged to try it on. (The patient repressed whether or not he ever used the female clothing to stimulate himself sexually, or whether his practice of masturbation was associated with it.) The patient reported the mother as neglectful, and claimed she would not buy food; and would put all her time and money into shopping for clothing in a compulsive manner, bringing home bags of it that were later stored in a separate apartment with the tags still attached.

The father enjoyed baseball in his free time, and took his son to a few male activities, but was otherwise passive. The boy tried to emulate his father and tried to build up his slender body by joining athletic teams in high school. At age 17, he joined the Navy where he felt accepted and useful, and no longer felt the need to be a woman. He had had crushes on girls and felt no attraction to males. He was a virgin when he married, but had no problem consummating the union or becoming a father. The urge to cross-dress subsided during those years of marriage but never quite left him. Normal periods dominated until the patient was in his 50s. During the last few years, especially since retirement, and since his wife moved out of the bedroom, he felt lonely and started to don a dress in the evening or night time. Once he considered a sex change operation.

The mental status examination revealed a tall, slim, intelligent man dressed in conservative male attire and free of effeminate behavior. His affect was rich, his mood neutral. He was tense, and told his story in a matter-of-fact way without showing shame or guilt. At times he spoke as if delusional. "She wants to come out and dominate. She is in competition with my wife." On closer questioning, he stated that he knew this was not real. There was no true thought disorder and no true delusions, nor was he believed to be psychotic. His judgment was intact about all matters other than cross-dressing. Cognitive functioning was unimpaired. Insight was limited.

The diagnostic impression was that the cross-dressing, practiced for the relief of tension and gender discomfort, had developed into a compulsion, though not one that was experienced as ego-alien as in most cases of OCD. In establishing a differential diagnosis, consideration was given to the possibility that the patient's behavior could simply be the result of a subclinical depression, but the patient's long-standing periodic gender identity confusion and the absence of depressive symptoms made the diagnosis of atypical and/or combinant paraphilia and OCD more likely.

The patient was placed on fluoxetine hydrochloride, 20 mg daily, and insight-oriented psychotherapy, first twice, then once a week.

The patient responded with astonishing speed. By the evening of the first day, he was calmer, less insistent on donning feminine attire. A week later, it was sufficient for him simply to put on a woman's nightgown; he would then feel completely relaxed and able to sleep. After approximately four weeks, the patient lost the urge to cross-dress. The possibilities for insight-oriented psychotherapy became limited; the patient remained resistant to seeing any connection between his present difficulties and the symbiotic relationship with his mother. He now merely wished to forget the past. His wife, also, after brief biweekly therapy, was able to reestablish a positive relationship with her husband.

Discussion. The patient described here, exhibiting a symbiotic relationship with a mother he perceived as neglectful, under the stress of retirement, advancing years, and the unconscious fear of abandonment, developed a wish to be a woman, which escalated into a compulsion to be acted upon in the form of cross-dressing. The latter was completely controlled in a matter of weeks, with the abandonment of the wish to be a female. It is suggested that the use of fluoxetine hydrochloride was critical in effecting this recovery in such a short time. His marriage has remained stable and he has undertaken employment in a recently selected new career. This treatment alternative has been presented for consideration in other cases where cross-dressing is used for tension relief, and where gender identity confusion is of lesser severity. The simple intervention with fluoxetine in a rather low dose may prove efficacious, although this has not been established to date in a controlled study.

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Unnatural causes: America's three leading killer diseases

Unnatural Causes: The Three Leading Killer Diseases in America. Edited by Russell C. Maulitz, MD. 212 pp, illustrated. New Brunswick, NJ, Rutgers University Press, 1989. $37.00 (clothcover)

While discussing the three leading killer diseases in America—heart disease, cancer, and trauma—this collection of articles raises intriguing issues and yet coalesces on a unifying theme. Issues raised can be phrased as questions and include the following:

- What is an "unnatural cause" of death?
- Will applications of advanced technology reduce mortality among the leading killer diseases in America?
- Are life style and behavior changes most important in impacting upon the leading killer diseases?

This last question also relates to the unifying theme specifically noted in each of the 13 contributions to this volume. As I read the articles, my mind kept returning to my study of Greek and Roman literature in college. Ancient essayists and dramatists consistently employed the concept of rewards for those who followed the middle road—who were moderate in all areas and did not engage in excesses. Theognis typified the sentiment about 550 BC in *Sententiae:* "Nothing in excess; moderation is best in all things."

Relative to heart disease, cancer and trauma, this volume certainly advises control of excess in terms of dietary intake, cigarette smoking, alcohol use, exercise, occupational hazards, and assorted environmental risks. Overeating, overdrinking and under exercising—these so-called "sins of civilization"—may contribute more to the mortality statistics than the combined actions of bacteria and viruses.

After telling a patient that his carcinoma of the prostate gland was treatable and that he would go on for several more years and probably die of natural causes, Dr Russell C. Maulitz wondered: "Is there such a thing as a 'natural cause'"? In the introductory chapter, as editor of this 1987 symposium collection commemorating the 200th anniversary of the College of Physicians of Philadelphia, Maulitz cites three conceptual viewpoints:

- Fatalistic—Death by any instrumentality is natural.
- Triumphalist—Natural death is one foreordained by the natural running down of the biological clock.
- Aesthetic—Natural death is the good death, the cleanly anticipated death.

Furthermore, Maulitz comments that there are few fatalists and aesthetes in the late 1980s. Logically, that leads us to consider the "intellectual toolbox" provided in this book "to grapple with some of these difficult issues." Tools in this volume include molecular biology, behavioral science, epidemiology, and political economy. Pointedly illustrated with 28 figures and 12 tables as well as relevant references, the articles in this collection are clearly instructional.

Pointing out the fluidity patterns of disease, Maulitz recalls that critics suggest that "we merely trade today's pestilence for tomorrow's plague." With high expectations, Maulitz comments that the authors in this volume "are characterized by unconventional thinking and fresh insight into the diseases on which they concentrate."

Still in the introductory section, Dr Daniel M. Fox reviews 60 years of health policy and changing epidemiology in the United States relative to chronic disease. He begins with the analytical and promotional activities of a few people in the 1920–1933 period creating an awareness of the shock of chronic disease. A purposeful definitional ambiguity may have helped to avoid resolution of the problem. In the years 1933–1940, national policy seeds were implanted with the passage of the Social Security Act and the data from the National Health Survey. World War II was the dominant factor between 1941 and 1945 and earlier activities stimulated priority changes among public agencies, hospitals, and the research community. Coalitions formed between 1945 to 1960 fluctuated between growth and frustration, with funding a major issue. In the 1960–1972 years, the first national policy for the costs of chronic disease emerged in the form of federal legislative mandates and the enactment of Medicare and Medicaid. From 1973 to the present, cost containment influenced political and scientific decisions without arriving at an acceptable consensus on paying for care.

Moving into the section on cardiovascular disease, Dr Stanley J. Dudrick and his colleagues discuss the experimental reversal of clinical and experimental atherosclerosis. These researchers elucidate on animal and clinical dietary experiments, and on the use of total parenteral nutrition (TPN) at home. Table 3.3 lists the amino acids, trace elements, multivitamins, optional additives and all the ingredients in the average daily anticholesterol solution administered during TPN. Advantages of using a totally intravenous diet to reduce cholesterol levels include the following: diet components are finitely defined and administered into the bloodstream for immediate biochemical reactions; cholesterol lowering effect results in 40% to 65% reductions—unprecedented with any other single form of anticholesterol therapy; patient receives prompt symptomatic benefits and gratification; arrest and reversal of atherosclerosis can be documented indirectly and/or directly and will improve as measurement techniques are developed.

Citing a number of references, Dudrick et al conclude that "it appears that reducing elevated total plasma cholesterol levels can significantly decrease the risk, or is associated with a decreased risk, of myocardial infarction secondary to atherosclerotic coronary heart disease."

Dr Bernard Lown reflects on sudden cardiac death and the interactions between the brain and the heart. Ischemic heart disease (IHD) is "the most common class of cardiac disorder in which occluded arteries promote damage to the heart muscle." Critical treatment advances in the 1960s included techniques for cardiopulmonary resuscitation, defibrillation, and cardioversion.

Theoretically, Lown posits: "Ventricular ectopic activity, among patients with ischemic heart disease, is a risk indicator for sudden cardiac death." Electrophysiological abnormalities may be triggered by anatomic or physiological factors such as altered tone or spasm of a coronary artery, increased blood viscosity, enhanced platelet aggregability, or transient embolic or thrombotic obstructions. How is stress determined? Lown is surprised that there is a paucity of studies of the role of the brain in affecting the heart and predisposing to sudden cardiac death.

Four dynamically interrelated sets of conditions are identified that contribute to the occurrence of malignant ventricular arrhythmias in a large number of patients: electrical instability of the myocardium; a psychological state stressful enough to burden daily life; a psychological triggering event; the presence of potassium deficiency, hypokalemia.

Future therapeutic directions include neuropharmacology to reveal psychobiological relations in humans. Attention to neurotransmitters such as serotonin and modification by dietary means may be important since serotonin affects ventricular fibrillation. Lown also talks about the use of tryptophan, phenelzine and carbidopa relative to ventricular fibrillation. "Recent advances in neurochemistry and neurophysiology, as well as the advances of computers, make decisive leaps possible in an area which has been limited by the complexity of the terrain."

A section on cancer opens with a molecular biology analysis of oncogenes and tumor suppressor genes by Dr Robert A. Weinberg. Oncogenes are "specific genes within cells that force normal cells to assume the traits of malignancy. These genes often acquire the ability to induce malignant traits after suffering damage at the hands of carcinogenic agents." Recombinant DNA procedures allowed for the isolation and study of oncogenes. Data indicate that oncogenes are of indigenous origin, being altered states of normal cell genes—often termed proto-oncogenes. Oncogenes may be created by a distinct genetic change in nucleotide, by transformation of chromosome, or by simple amplification in the gene copy number of a proto-oncogene. H-ras oncogene is identical to the bladder carcinoma oncogene; myc oncogene participated in the chromosomal translocation of Burkitt's lymphoma; and the erb B oncogene was found to undergo amplification in the human A431 epidermoid carcinoma cell line.

Manipulation of the gene using anti-oncogenes as suppressors to inhibit tumor formation appears to be the next step. Restoration of an entire human chromosome 11 to Wilm's tumor cells causes them to lose their tumorigenic abilities. "If retinoblastoma tumor cells are tumorigenic because of the absence of functional copies of the Rb gene, then tumorigenicity may be lost when functional versions are reintroduced into these cells." Anti-oncogenes like Rb "seem to exercise their normalizing effect by coupling a cell with an environmental signal regulating growth."

Combining the knowledge of genetic engineering and biochemistry, data on the interactions of genes in balance and unbalance will accumulate. Information on malignancy formation can produce new insights to battle cancer and to devise cures.

Drs Bruce A. Chabner and Marcia J. Browne of the National Cancer Institute detail the prospects for the drug treatment of cancer. These researchers discuss the new concepts of "polyresistant" cells, biological response modifiers, protein kinase C, manipulation of the immune system through immunotherapy and monoclonal antibodies, and antimetabolites and antifolates. However, a sentence about the bottom line impact is not comforting. "Despite these major advances, the number of patients actually cured by chemotherapy remains less than 10% of the total cancer population; and most patients who succumb to cancer do so after failing to respond to chemotherapy."

In an analysis of risk factors and mortality, Dr Philip Cole makes a strong declaration on cancer prevention. "Virtually half of all malignancies in this country today are of known etiology, and the etiologic factors are environmental and thus susceptible to favorable manipulation." Tobacco is a major target for control, followed by diet (excess fat, fiber deficiency, caloric excess) and occupational exposures to chemicals and other risks. Treatment success also continues to move upward from a 39% survival rate in 1960 to a 50% rate in 1980.

With life expectancy increasing rapidly, with prevention strategies improving moderately, with detection and treatment improving slowly, cancer rates are stable or declining slowly and disability increasing moderately. Dr Cole predicts that "By 1998 it [cancer] will be the major cause of mortality and, perhaps, of morbidity."

Dr Edward J. Sondick reviews the progress in cancer prevention and control and concludes: " . . . the age-adjusted mortality statistics over the last 50 years for many of the major cancer sites have experienced relatively little change." Three exceptions are noted: lung cancer rose precipitously; stomach cancer had a great decline; and cervical cancer mortality also declined. Prevention activities involve chemoprevention, diet and nutrition, and smoking and tobacco use cessation. Cancer management programs include screening and detection, community oncology, rehabilitation, and continuing care. Resources development includes training, cancer centers, construction, and organ system research. As others have done, Sondick emphasizes the fact that the toll of cancer can be reduced with the information already on hand. Estimates from the National Cancer Institute claim that "anywhere from 25 to 50% of cancer mortality can be reduced by the year 2000 if our full measure of knowledge is applied as effectively as possible."

Opening the section on trauma, Susan P. Baker calls injuries in America a national disaster. "Trauma is the number one cause of death in this country in individuals between one and 44 years of age." Trauma risk is distributed unevenly among the population and may vary by race, age, income, geographic location, and other factors. Furthermore, specific risks seem to impact differently such as homicide, suicide, falls, fires, motor vehicle accidents, poisoning and so on. However, it appears that predictable risks may be acceptable to society. Suicide and homicide among certain population groups do not engender societal reactions. On the other hand the randomness of risk may arouse public emotions. When foods or drugs are tampered with, anybody could be affected.

Automatic protection such as passive seat belts, fuses, and design safeguards on machinery provide sure and swift reductions in trauma. Yet, Baker feels that the United States tolerates conditions that amount to a national disgrace in many areas. We tolerate scalding hot water in residential facilities for the elderly; we tolerate children's play equipment high enough to cause serious injury in a fall; poorly designed trucks and autos almost guaranteed to inflict injury in a frontal collision; we tolerate glamorous public areas designed without regard for safety; and we tolerate flammable materials in the home capable of being ignited. Raising more questions than answers, Baker poses a nagging question: "Can a national campaign against trauma be *this* far behind?"

Dr Mark L. Rosenberg states that violence is a public health problem. Risk variables include sex and race in one telling example. "The lifetime probability of homicide victimization for a baby born in the United States in 1981 was 1:450 for a white female baby, and 1:28 for a black male." For white males, the probability was 1:164. In terms of years of potential life lost (YPLL), homicide and suicide rank fourth and fifth, respectively. In economic terms, suicides in the 15–24 year age group in direct costs and lost earnings was about $2.3 billion in 1980. Paradoxically, intentional injuries are a large part of the problem but receive a disproportionately small part of injury resources and research attention. What are the biochemical-neurophysiological bases of violence and aggression?

Homicide and suicide prevention programs have received a great deal of attention but there are significant barriers to overcome. A beginning behavioral inhibitor requires measures to decrease the cultural acceptance of violence as normal. "Spare the rod and spoil the child" is an example of cultural acceptance of spanking. There is a long history of aborted efforts to institute gun control legislation in this nation over the traditional cries for the citizen's right to bear arms.

Finally, Dr G. Tom Shires discusses the organizational and physiological considerations in the care of the injured in America. Data are presented on the physiology of trauma, hemorrhagic shock, and septic shock. Of interest, a Center for Injury Control (CIC) has been established in the Centers for Disease Control initially. However, the start-up budget was only $10 million when Shires estimates $100 million was needed.

Less than 2% of the National Institutes of Health budget ($34 million out of $6.2 billion) is spent on injury control research. "Injury thus remains a neglected disease of modern society, an epidemic that we are not adequately addressing." As an example Shires notes that only three nonmilitarily-funded burn centers exist in the entire United States.

In concluding views, Dr Alvin R. Tarlov delves into the spectrum of health and the matching of medical education to medical realities. He raises three questions for discussion:

- What is health?
- What are the determinants of health?
- What are the points of intervention in health most appropriate for tomorrow's physicians?

After noting that there is a continuous spectrum from perfect health to ill health, Tarlov divides determinants into health care, individual behavior, and surrounding influences. Impacting on the determinants are statistics showing that tobacco use, alcohol abuse, motor vehicle crashes and other injuries, and substance abuse together account for 52% of the economic costs of illness. A high proportion of the remaining 48% is accounted for by dietary factors, lack of physical fitness, and unattended hypertension.

In view of the above, what are the medical intervention points? Starting at the bottom of Tarlov's intervention pyramid, the list includes cultural beliefs, laws and regulations, commercial practices, social norms including fads and customs, behavioral risk factors, physiological risk factors, and finally ill health. Tarlov comments that the physician intervenes primarily at the top of the pyramid when ill health is evident. He calls for physicians to broaden their understanding and to start in medical education. "By itself, however, biology as a basis for medical practice is insufficient . . . We need to understand health in sociocultural and behavioral, as well as biological, terms, and to join biological concepts with those stemming from social, economic, governmental, and cultural phenomena."

In the final article in this book, Dr Paul D. Stolley presents the epidemiologist's perspective looking toward the 21st century. He identifies the three leading causes of death in American as heart disease, cancer, and stroke. Cigarette smoking, alcohol addiction, substance abuse, homicide, and AIDS are linked to behavior disastrous to the health of Americans. Environmental threats include water supply contamination, ozone layer depletion, acid rain, and disturbances in ecology. "The greatest single action Americans can take to improve the nation's health would be to stop smoking cigarettes."

Some 38 "Health for All" targets include attention to providing health, to life styles, to environmental changes, to health care systems, and to support for health development. However, Stolley echoes earlier views: "The promise of high technology applied to the medical field will not have a large effect on the three major killers over the next decades . . . Understanding the necessary health strategies for the next century will require not just a knowledge of medicine but familiarity with history, ecology, and economics."

What is evident from all 13 articles in this book on "unnatural causes" of death is that physicians must expand their scope of concern. Most of the major killers in America are not caused by a specific microbe or virus. Life style, patterns of behavior, and environmental factors are the evident culprits. Beginning in medical school and other health care educational centers, providers have to integrate their scientific learning within a broader base of information that can help their patients to help themselves to better health. Both Tarlov and Stolley agree on that in their summary comments.

This book provides a mixture of philosophy, scholarly scientific updates, generally useful statistical data, hope for the future, pessimism about the impact of technology, and crystal ball gazing into the future. While an often uncomforting mixture of concepts and ideas, this book is recommended for those who wish to find out what is happening as well as for those who want to influence what is about to happen.

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FROM THE LIBRARY

DO YOUR PATIENTS REALLY LIKE YOU?

Nothing is a safer topic for conversation than doctors and the medical profession. A group of people may have had violent disagreements, but when the discussion is shifted to doctors, most of the people present will probably agree. Unfortunately, in most cases they will agree on negative facts about the medical profession. Even those people whom you would not consider capable of unfairness and subjective attitudes will join in gleefully to report incidents from their own experience where doctors were wrong, overcharged, drove around in Cadillacs after only one year in practice, split fees, and a long list of comparable horror tales.

From a public relations viewpoint and to the researcher in human motivations, a big question mark poses itself. How come? Why, from among all the various possible scapegoats in modern human society, are doctors picked, and what can be done about it? How can we make patients like their doctors more? . . . I have conducted a number of . . . experimental studies to determine in what ways the doctor-patient relationship can be improved.

. . . In summarizing our findings, it was shown that what had happened was that while the world was changing and changing very rapidly; while the patient in this world was changing at least at the same pace; while all the medical equipment, medical knowledge, and drugs were developing at an ever-increasing rate, the human aspect in the doctor-patient relationship had fallen behind. A psychologic lag had taken place. We discovered that most doctors choose their profession for idealistic reasons. Although a good income is usually expected, and this factor does enter into the picture, the idea of helping humanity and doing something worth-while, uncommercial, and out of the doldrums of everyday life, of devoting oneself to helping others is very strongly accented. Something vital and very important happens to the young doctor during the course of his major studies and particularly during his internship. The young doctor discovers the harsh realities of life. These realities very quickly clash with his initial, highly exaggerated idealism.

The patient, as well as the doctor, becomes aware of this conflict. The doctor often exaggerates his idealism to overcompensate for the recognition that in many ways he really has to behave like a businessman. The patient, on the other hand, is asked and expected to pay respect comparable to the kind of reverence that one pays to a saint, and then a few days later he receives a bill; again a conflict is the result. A further difficulty stems from the fact that while the modern patient is catered to by most other businesses and professionals and even large corporations, the medical profession as a whole has failed to acknowledge that this patient has been growing up, is insistent on his right to be treated as an equal, and spoken to in clear understandable language. . . .

I think that the answer to improved public relations will not lie in insistence on rugged individualism and absolute freedom nor in federalization and socialization of medical care. The answer will lie in a new democratic form of cooperation where the individual gives up some of his prerogatives, accepts the responsibility of the health of the community, recognizes the right of his patients, and learns to cooperate with them. The answer to how to make your patients like you more lies in recognizing the fact that this world is a changing one, that the patients are changing, and that you too must learn to change.

ERNEST DICHTER, PhD
(*NY State J Med* 1954; 54:222—226)

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AIDS morbidity and mortality

The rate of increase in new AIDS cases has slowed in New York State, not only among cases attributed to homosexual/bisexual activity, but also among cases linked with intravenous drug use. When all case reports are filed, the 1989 AIDS case total is expected to increase by only 3% above the 1988 total to slightly over 6,700 cases.

The reasons for this slowing of AIDS incidence are under review, and may include decreased rates of HIV infection in prior years, reduced numbers of infected people developing full-blown AIDS because of improved therapies and changes in reporting patterns.

New York continues to have the highest cumulative number of AIDS cases in the nation, accounting for 23% of the US case total. Compared with the rest of the country, New York has a higher proportion of cases among intravenous drug users (IVDUs), minorities, women and children.

Geographic Distribution

Of all AIDS cases reported in New York State (excluding cases diagnosed among state prison inmates), 87% were residents of New York City. Of the cases elsewhere in the state, 90% were residents of counties in the New York City vicinity, mid-Hudson Valley and upstate urban centers.

While AIDS cases are highly geographically clustered, cases have been reported from nearly every county in the state. In New York City, cumulative incidence rates per 100,000 population ranged from 101.7 on Staten Island to 738.3 in Manhattan. Outside of the city, the cumulative rates ranged from 0 to 72.4 per 100,000 population.

Risk Factors

Since 1988, AIDS cases diagnosed among intravenous drug users have exceeded the number of new cases diagnosed among gay and bisexual men in New York State. The proportion of AIDS cases among homosexual/bisexual men has decreased from 60.0% of all cases prior to 1986 to 44.8% as of December 31, 1989. They accounted for 37.0% of cases reported in 1989.

Male and female IVDUs now account for 38.3% of New York AIDS cases compared with 20.6% in the US. The percentage of AIDS cases among IVDUs has risen in the state from 29.3% prior to 1986. Over half (61.6%) of all women with AIDS in New York State reported intravenous drug use. Of the 613 pediatric AIDS cases, 77.7% were born to mothers who were IVDUs or sexual partners of IVDUs.

Of all AIDS cases in New York State, 3.4% were attributable to heterosexual contact. Of these, 95.6% were female sexual partners of people at risk for HIV infection. Of all heterosexual contact cases, 84.0% of the at-risk partners were IVDUs.

The percent of AIDS cases in other risk groups include hemophiliacs (0.2%), foreign born—NIR (no other identified risks) (1.9%), transfusion (1.0%) and pediatric (2.2%). Nearly 4% of AIDS cases have undetermined risk.

Age/Race/Ethnicity

So far in New York State, 88% of AIDS cases have been reported in young and middle-aged adults between the ages of 20 and 49. However, the recognized long incubation period between infection and serious illness may indicate that many of these people became HIV infected in their late teens or early twenties.

Minorities are carrying a disproportionate burden of the AIDS epidemic in New York State. Of residents diagnosed with AIDS through the end of 1989, 34% were black and 26% were Hispanic. Only 38% of cases were among whites, who comprise 75% of the state's population. Most women and children with AIDS are minorities. Of all female cases, 52% are black, 30% Hispanic and 17% white. Pediatric AIDS cases are 55% black, 34% Hispanic and 10% white.

Mortality

Overall, 65% of people reported with AIDS in New York State are known to have died since diagnosis. The death rate, which increases with time since diagnosis, ranged from more than 95% for people diagnosed through 1983 to 13% for those diagnosed in the second half of 1989.

In New York City, AIDS is the leading cause of death among 30–49-year-old males and 20–39-year-old females (Table 1). It is the second leading cause of death among New York City children aged 1–9 years. In the rest of the state, AIDS is among the top five causes of death in 20–49-year-old males and 30–39-year-old females. AIDS is the leading cause of death among state prison inmates in New York State.

While the death rate remains high, survivorship appears to be improving slightly. The percentage of homosexual/bisexual AIDS patients who have survived one year or more from date of diagnosis has increased from 43.4% of cases diagnosed in 1984 to 58.4% of cases diagnosed in 1987. This gain may be due to earlier diagnosis of AIDS and new, more effective treatment methods.

HIV Seroprevalence

Since late 1987, the State Department of Health has tested more than 620,000 anonymous blood samples for HIV antibodies, representing the most comprehensive effort by any state to identify HIV infection rates and to use such data to design and target prevention activities. Groups from which blood specimens are being tested blindly include: newborns (women giving birth), family

TABLE I. Ranking of AIDS Among the Five Leading Causes of Death by Population Group and Age, New York State, 1988

Age	<1	1–9	10–19	20–29	30–39	40–49	50–59
New York City Males							
Total	4	2		2	1	1	3
White	4	5*		1	1	1	3
Black	3*	2		2	1	1	3
Hispanic	4	2*		2	1	1	3
New York City Females							
Total		2		1	1	3	
White		3*		3	2	5*	
Black	5*	2		1	1	3	
Hispanic		2		1	1	3	
Rest of State Males							
Total				4	3	4	
White				4	5	5	
Black		2*		3	1	3	
Hispanic		1		2	1	1	4
Rest of State Females							
Total					5		
White					5		
Black				5	4	4	
Hispanic			4*		1		

* Tie with another cause of death.

planning clinic clients, sexually transmitted disease clinic clients, people in drug treatment, state prison entrants and homeless youth (Table II). The department also monitors HIV-infection rates among blood donors, hemophiliacs, military recruits and people who voluntarily seek HIV antibody testing through anonymous test sites, medical clinics and private physicians' offices (Table III).

Rates of HIV infection within the groups tested blindly vary significantly, from 26%, or one in four drug treatment clients in New York City, to 0.5%, or one in 200 clients in family planning clinics. Overall, the data confirm the higher risk of infection among people who reside in economically impoverished urban neighborhoods where drug use is prevalent. Rates of HIV infection are also elevated among men and women who are being treated for other sexually transmitted diseases. In general, HIV infection rates are consistently higher among residents of New York City compared with the rest of the state; in people between the ages of 20 and 39; and among blacks and Hispanics.

AIDS Case Projections

The Department of Health updates its AIDS case projections annually based on the most current and relevant

TABLE II. HIV Antibody Seroprevalence Among All Groups Tested in Blind Seroprevalence Studies, New York State, through December 1989

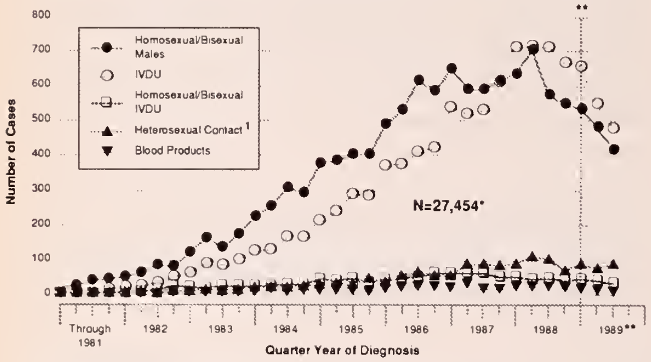
	Tested Through December 1988			Tested During 1989		
	Number Positive	Number Tested	Percent Positive	Number Positive	Number Tested	Percent Positive
Newborn Infants	1,964	298,392	0.66	1,822	283,213	0.64
New York City	1,671	133,603	1.25	1,537	127,415	1.21
Rest of State	262	158,996	0.16	260	150,699	0.17
Family Planning Clinic Clients	65	12,840	0.5	55	11,570	0.5
New York City	54	6,476	0.8	40	5,350	0.8
Rest of State	11	6,364	0.2	15	6,220	0.2
STD Clinic Clients[1]	90	4,039	2.2	93	4,132	2.3
Drug Treatment Clients[2]	268	708	37.9	443	1,674	26.5
Male Prison Entrants	86	493	17.4	—	—	—
New York City	69	341	20.2	—	—	—
Rest of State	17	152	11.2	—	—	—
Female Prison Entrants	84	448	18.8	—	—	—
New York City	79	334	23.7	—	—	—
Rest of State	4	97	4.1	—	—	—
Homeless Youth[3]	86	1,351	6.4	51	1,091	4.7

[1] All participating STD clinics are outside of New York City. Data is for 4th quarter only of 1988 and 1989.
[2] All participating drug treatment clinics are in New York City. Data from 1988 overrepresent high seroprevalence programs and are not directly comparable to 1989 data.
[3] All homeless youth tested are in New York City.

TABLE III. HIV Antibody Seroprevalence Among Groups Tested With Their Knowledge, New York State, through December 1989

	Tested Through December 1988			Tested During 1989		
	Number Positive	Number Tested	Percent Positive	Number Positive	Number Tested	Percent Positive
First Time Blood Donors	127	161,768	0.08	92	107,470	0.09
Downstate[1]	115	81,519	0.14	82	52,765	0.16
Upstate[2]	12	80,231	0.01	10	54,705	0.02
Military Recruits[3]	393	111,775	0.35	91	30,525	0.30
New York City	304	35,613	0.85	73	9,993	0.73
Rest of State	89	76,162	0.12	18	20,532	0.09
Anonymous Test Site Clients	6,022	57,272	10.5	4,112	35,330	11.6
New York City	3,991	26,736	14.9	3,564	23,924	14.9
Rest of State	2,031	30,536	6.7	548	11,406	4.8
Medical Practice	33,007	165,572	19.9	17,600	122,022	14.4
New York City	24,296	93,366	26.0	11,520	52,020	22.2
Rest of State	8,711	72,206	12.1	6,080	70,002	8.4
Sexually Transmitted Disease Clinic Clients	1,050	8,934	11.8	1,265	13,099	9.7
New York City	880	4,349	20.2	1,032	5,472	18.9
Rest of State	170	4,585	3.7	233	7,627	3.1
Hemophiliacs				281	431	65.2

[1] New York City, Long Island and Hudson Valley Blood Services.
[2] Albany, Syracuse, Rochester and Buffalo American Red Cross Blood Services.
[3] An additional 149 recruits who tested negative are not included due to missing county of residence.
Notes: Categories are not mutually exclusive, some HIV-infected individuals may be represented in more than one group.
Sources: New York State Department of Health, New York City Department of Health, U.S. Centers for Disease Control.



* One homosexual/bisexual and one IVDU are not included due to missing year of diagnosis
**Data for 1989 are incomplete.
[1] Includes people who have had heterosexual contact with a person with AIDS or at risk for AIDS and people without other identified risks who were born in countries in which hetersexual transmission is believed to play a major role

FIGURE 1. Adult AIDS cases by risk factor, New York State, December 31, 1989.

information available. Given the uncertainties of predicting the future course of the AIDS epidemic, the department has developed two sets of five year projections, using two different approaches and modeling techniques. One set of projections, based on historic trends, predicts that cumulative AIDS cases will approximate 77,500 by the end of 1995. An alternative approach, which assumes that female sexual partners of intravenous drug users may become a leading risk group over the next five years, projects a cumulative AIDS case total of 100,600 cases by 1995.

This article was adapted from *AIDS In New York State 1989*. For copies of this report, call (518)474-5422, or write New York State Department of Health, Public Affairs Group, Room 1455, Corning Tower, Empire State Plaza, Albany, NY 12237.

Safe use of capillary blood sampling devices

Health care workers should be aware of a potential risk of hepatitis B transmission associated with *improper* use of spring-loaded lancet devices in obtaining capillary blood samples. While the Department of Health has not received reports of cases of hepatitis B or other blood-borne diseases associated with misuse of such devices in New York State, recent reports in the medical literature underscore the importance of avoiding cross contamination when using these units.[1,2]

One such report described an outbreak of 16 cases of hepatitis B associated with *improper* use of a spring-loaded lancet. Investigation revealed that health care personnel were replacing the hemolance, but not the platform after each use which resulted in the patient's exposure to the previous patient's blood.[1]

When used correctly spring-loaded lancet devices are effective and safe. Capillary blood samples are obtained by finger or heel sticks using hand-held single-use lancets or automatic spring-loaded devices. These devices are in widespread use in hospitals, clinics and private physicians' offices. Spring-loaded devices typically have two disposable parts: the lancet, used to puncture the skin, and the platform or endcap, used to control the depth of the puncture. Health care workers are generally aware of the necessity to replace the lancet after each use, but may fail to replace the platform or endcap, which may become contaminated with the patient's blood.

Health care workers using spring-loaded lancets should be instructed to:

- replace the lancet and the platform or endcap after each use;

- always order an equal number of platforms or endcaps when reordering;
- dispose of both lancets and platforms in an appropriate sharps container;
- not use spring-loaded lancets that do not have replaceable platforms.

Should you become aware of hepatitis B virus transmission in association with such capillary blood sampling units or any medical device, please immediately report the circumstances to the local health department and to the Bureau of Communicable Disease Control, New York State Department of Health, at (518) 474-3186.

References

1. Douvin C, Simon D, et al: An outbreak of hepatitis B in an endocrinology unit traced to a capillary blood sampling device. *N Engl J Med* 1990; 322:57–58.
2. Drinka P: Spring-loaded lancets. *N Engl J Med* 1988; 321:1762.

THE WORK AND PLAY OF WINNICOTT

By Simon Grolnick, MD. 232 pp. Northvale, NJ, Jason Aronson, Inc, 1990. $25.00 (hardcover)

Simon Grolnick, MD, offers us a tribute to D. W. Winnicott, and a synopsis of his major contributions. Well-versed in contemporary psychoanalysis and deeply committed to the study of Winnicott's ideas, Dr Grolnick brings an impressive combination of rigor and passion to his book. After a brief description of the cultural and analytic climate in which Winnicott worked, we are taken on a tour of Winnicottean principles, given a chapter describing the mother-and-child dyad, and introduced to Winnicott's developmental lines. This is followed by further description of what Winnicott called "the intermediate world," several chapters on reading Winnicott and applying his ideas to clinical practice, and closing comments about the future of Winnicottean therapy.

The Work and Play of Winnicott confronts the challenge inevitable to all works of tribute. How does Dr Grolnick, the student, convey his enormous respect without idealizing or reifying Winnicott the teacher? Dr Grolnick veers repeatedly toward idealization, and at one point he even presents us with the paradox that "Winnicott was not always Winnicott" (page 174). Although this may be an example of the author's playfulness, it also demonstrates a split between an idealized man who personifies certain abstractions, and a beloved but imperfect man. Nevertheless, it is hard not to admire Winnicott, whose compassion and wisdom are so poignantly captured in any number of quotes and vignettes. Thus, it was relatively easy for me to overlook Dr Grolnick's lapses toward idealization.

Throughout the book, we are immersed in the concepts of play and spontaneity, as well as the need for a dialectical perspective which can provide comfort and nurture our creativity in the "intermediate" realms—between reality and fantasy, between drive theory and self-psychology, between the unilateral nature of the therapeutic relationship and the necessity for some degree of mutuality. Perhaps it is with conscious attention to parallel process that Dr Grolnick creates a collage of Winnicott's ideas, forcing the reader away from an obsessive, intellectual relationship with his principles or developmental lines, and into a personal dialogue with these interwoven concepts. This way of presenting his material is at the same time a strength—teaching by example—and a limitation. The reader is not offered a critical analysis of each of Winnicott's ideas, nor is there an overall map which organizes the interrelationships of these developmental lines.

The book came together for me in chapter 9, devoted to the practice of therapy. It is as though Dr Grolnick is, in the very structure of his book, showing us again that ideas crystalize not in the abstract, but in the living interaction of therapist and patient. Dr Grolnick's writing takes on a particular clarity and poetry as he describes his work with patients, and it becomes quite clear how the principles presented in his earlier collage shape clinical interventions of great sensitivity and power.

This is an ideal book for students of theory and therapy who are interested in getting to know Winnicott and his ideas. It is readable, inviting, and provides an excellent introduction. More generally, the book should be useful and interesting to all of us who are concerned about the interplay of early development and later healing, theory and practice, patient and therapist.

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BRIEF THERAPY APPROACHES TO TREATING ANXIETY AND DEPRESSION

Edited by Michael D. Yapko, PhD. 384 pp, illustrated. New York, Brunner/Mazel Publishers, 1989. $39.95 (hardcover)

This book consists of 17 chapters that represent papers presented at the Milton H. Erickson Institute Conference at San Diego, California, in the winter of 1988. The chapters were written by some of this country's leading experts on the Ericksonian approach to psychotherapy, which looks to hypnosis and other brief therapies to treat mental disorders.

Dr Martin E. P. Seligman, Professor of Psychology at the University of Pennsylvania, and the "originator" of the "learned helplessness" model of depression, has written a concise and interesting chapter on predicting depression. Ernest L. Rossi, PhD, is the author or editor of 14 books on various psychological topics. His chapter on "Conciousness . . . .and the Mind Gene Connection" attempts to add knowledge to the field of psychobiology. Russell A. Bourne, Jr, PhD, is Director of the Counseling Center at Randolph Macon College, and his chapter on treatment of anxiety and panic disorders clearly goes over the Ericksonian viewpoint. Dr Michael D. Yapko, the book's editor and the Director of the Milton H. Erickson Institute of San Diego, has written a brief but interesting chapter on a seldom used treatment for depression involving future orientation. Robert Schwartz, PsyD, Director of the Institute for Advanced Clinical Training in Philadelphia, discusses the treatment of anxiety and depression in pain states.

Generally the chapters are presented in an interesting and understandable fashion. Enough background is given for the novice reader to understand and appreciate the subject matter.

This type of book gives a reader a chance to enhance knowledge in an area of psychotherapy that is often not fully understood. Those readers who are interested in expanding their expertise will probably find this to be an engaging and educational book.

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CHILD PSYCHIATRY: A PRIMER FOR THOSE WHO WORK CLOSELY WITH CHILDREN

By R. Dean Coddington, MD, and Mollie M. Wallick, PhD. 254 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1990. $37.50 (paperback)

This book is designed, as its senior author states, to "aid the counselors, teachers and other front-line soldiers" (ie, those who work with children) in understanding children and their behaviors. It is not written for child psychiatrists, highly trained psychologists, or social workers. Thus the authors have a dilemma—how to present complex material in sufficient authoritative detail and yet maintain the simplicity needed in a book for relatively untrained people. The dilemma unfortunately is not solved. There is a superficial quality to this book, a tendency for dogmatic statements, a simplicity which in fact may lead to erroneous concepts.

R. Dean Coddington is an eminent child psychiatrist who for many years was director of child psychiatry at Ohio State University at Columbus and professor emeritus of psychiatry at Louisiana State University at New Orleans. For this book he has the help of Mollie Wallick, who holds a PhD degree in special education. Together the authors have edited chapters by pediatricians, child psychiatrists, and social workers. The major portion of the book, however, is written by Dr Coddington.

This primer is divided into three sections, the first an overview of normal child development from infancy and early childhood to adolescence. A chapter on families (William Sze) completes the first section. The second section of this book is designed to teach the reader what to look for when assessing a child, and how to recognize anxiety, depression and internal conflict. Coddington believes that anxiety and depression are key symptoms in childhood. He makes a clear distinction between depression as a symptom of an adjustment process and depression as an endogenous biological state. This is an important distinction, one with which child psychiatrists daily grapple and which has implications for reducing the reflex prescription of antidepressant drugs in childhood. Unfortunately, the difficulty in making the distinction is not stressed, resulting in an unrealistic expectation on the part of child care workers. Coddington also rightly describes individual coping mechanisms in response to stress and he attempts to quantify stress with a "life-events scale." The themes presented in this section are worthy of more than a superficial presentation.

The final section of the book is a clinical one, describing a spectrum of specific psychological disorders of childhood, including mental retardation, attention deficit hyperactivity disorder (ADHD)/learning disability, conduct disorders, psychophysiological disorders, eating disorders, developmental disorders, stereotyped movement disorders, pervasive developmental disorders and autism, schizophrenia, and substance abuse. This section is a mixed one. Recognizing the difficulty of presenting clinical material to a relatively untrained audience, the authors try to do too much, squeezing complex concepts into a few pages, using dogmatic, and in some instances, such as equating ADHD with learning disability, incorrect concepts. Rather than attempting to cover all childhood pathology, the authors would have made a better contribution if they restricted their material to the first two portions of this book, to normal development, to the genesis of key symptoms such as anxiety and depression, and to the ways in which children cope with their experiences.

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COMPUTER APPLICATIONS IN PSYCHIATRY AND PSYCHOLOGY

Edited by David Baskin, PhD. 192 pp, illustrated. New York, Brunner/Mazel, 1990. $23.95 (hardcover)

As the forenote to this book states, "Psychiatry is in a state of flux." So, too, is the computer industry, which makes it a risky venture at best to write books about the interface of these two rapidly changing worlds. Nevertheless, this book is a good place to start for the novitiate in computers as well as for the clinician who, like this reviewer, might be conversant with computer applications per se but not well versed in the clinical applications of these machines that are so changing our world. The book is generally well-written and easy to read. The fact that each chapter exists independently makes it a book that can be read in fits and starts with no loss of continuity.

The book deals with two broad topics: clinical applications of computers and mental health information systems. As such, it should be of interest to clinicians as well as to administrators who have to deal with large amounts of data and detailed reporting requirements.

I found the sections dealing with the clinical use of computers to be a useful "broad brush" introduction to what can prove to be a useful tool. There were discussions of many aspects of the use of computers in the clinical setting. One question that is frequently asked is "how do patients accept the use of a computer in the clinical setting?" Having written and used a large relational database application that comprised the entire clinical record for about 100 patients making multiple visits over a period of two years, I was interested to find that my experience was also replicated by others in the finding that there was little resistance to the use of a computer during the interview process.

As one who has started using computer-based test interpretations within the past six months (MMPI-2 and MCMI-II through NCS), I was especially interested in the material dealing with this topic. Having personally found these tests clinically useful (with no special training in psychology), I thought some of the caveats a little overdone. However, the material is presented in a clear, relevant, and clinically useful manner, and is certainly worthwhile reading for anyone even considering the use of these tests. The tests are easily administered in an office or hospital setting, and can be scored by mail without ever touching a computer, or in just a few minutes by phone with the use of a modem attached to a computer. They offer a quick, inexpensive, and I believe effective aid in diagnosis and can help in formulating a treatment approach. I do not think they are a substitute for a thorough psychological evaluation done by a competent psychologist, and they should not be viewed as such.

The material on computers as patients and computers as psychotherapists was interesting. The discussion of computer-generated patient histories, wherein a patient sits at a computer alone or with an aide, and answers questions as they appear on the screen, with the computer then generating a history, was thought provoking.

I was less satisfied with the portions of the book dealing with information management. It would seem that anyone with information to manage seems to believe that their information is special and unique and therefore needs to be dealt with uniquely. This invariably leads to specialized applications and confusion. In my view, the most effective way to set up and use information management systems and more specifically, relational database systems, is to start to think of data in terms of fields, records, and files. Once these concepts are absorbed and integrated, the rest follows easily. One of the best ways to absorb these concepts is to read examples of what others in similar circumstances have done to implement computerized handling of their data. There are several good examples in the book of systems that others have set up. They alone would justify reading the book and should prove worthwhile to anyone implementing or thinking of implementing a computerized information management system.

What the book includes, it handles well. There are some obvious omissions and they are in areas that portend much change for all of medicine. I would have liked to see discussions of artificial intelligence, especially in the areas of diagnosis, and possibly for emergency department screenings, etc. Compact disk read only memory (CD-ROM), another new technology just starting to have an impact, holds the promise of making vast amounts of information instantly available. The disks are similar to audio CDs, and can contain about 550 megabytes each (about 275,000 typewritten pages on a single CD) at a manufacturing cost of less than $5 each. At present it is possible to instantly access the entire PDR in the office in seconds via CD-ROM. On-line searching is another current technology that makes it possible to use any computer to search databases such as the National Library of Medicine via a modem and an ordinary telephone line. These services are generally not cheap to use but offer considerable savings of time once they are mastered.

Implications of computer usage might have been dealt with more. For example, while I am an enthusiastic computer user, and see the machines as offering ways to do many things to the benefit of both users and patients, there are reasons to be concerned and uneasy. Medicine is becoming more and more "cookbook" every day, and the burden of proof in the clinical setting is continually being placed on the clinician to justify any deviation from that cookbook. What also is of concern is that once the leap to electronic record keeping or even to typewritten records is accomplished (effective optical character recognition is not far off), it becomes a trivial exercise for Big Brother to monitor clinicians much more closely. Every record could be screened by machine for compliance. The implications are mind-boggling. (There is a certain safety in handwritten records! Machine reading of handwriting appears to be a long way off at this time.)

There will unquestionably be a significant role for computers in the future of medicine, which includes psychiatry and psychology, and it is coming whether or not we are ready, and whether or not we like it or want it. We can only hope that computers can become useful tools for clinicians who are trying to help patients, and that their use to enforce cookbook medicine can be limited. Which way things will go, if indeed it is an either/or situation, is not clear at this time. What is clearer is that very significant directions will be established in the not too distant future, and these paths will determine what eventually happens. It will be largely up to the current generation of clinicians to determine how that future is shaped. This book provides a start.

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STRESS MANAGEMENT: AN INTEGRATED APPROACH TO THERAPY

By Dorothy H. G. Cotton, PhD, 288 pp, illustrated. New York, Brunner/Mazel Publishers, 1990. $30.00 (hardcover)

The purpose of this book is "to help clinicians from all disciplines to conceptualize and control stress in patients seen in a clinical setting." Dr Cotton has attempted to present an integrated approach to stress management as a therapeutic modality, with portions of the book describing this integrative approach and suggesting methods of charting problems and treatment options. In addition, the book addresses stress issues particularly as they relate to gender, with recommendations regarding women's concerns.

Dr Cotton is cited as having extensive clinical and consultative experience and, based upon the comments by the series editor and the editorial board which is involved in the book review process, she is a very well qualified author. Her integrated paradigm for assessing and treating stress is based upon this clinical expertise, and is supported by various aspects of research in the area. She has written a general, readable text that offers practical information to the therapist. She includes criteria whereby physicians might refer patients for stress management and offers insights to the physician as to how stress might be affecting various aspects of a patient's behavior. The author also carefully outlines various treatments for stress including physiologic, cognitive, and behavioral approaches. Case studies offer a systematic presentation of three patients (two females and one male) included at the conclusion of each chapter. These case studies provide a means of further demonstrating the actions of the therapist within the context of the author's integrated approach to stress management.

Easily readable and clinically applicable books on the subject of stress management are very important to the medical community. Stress management books frequently delve into extensive detail for more research-oriented practitioners who are experts in stress management and treatment of stress-related disorders. They are therefore written at a level of detail that is more than is required by therapists with a more clinical focus, or physicians and other health professionals interested in learning how stress may affect various aspects of their client's care. While this is a strength of the book, it may also require that one seek other sources for more detailed information.

The author begins the book with a general overview of stress management and continues with definitions of stress. She carefully cites various authors in the field, offering their perspectives. She then discusses the physiologic aspects of the stress response, relating them to cognitive as well as physiologic changes. The next section addresses stressor related illness from the perspective of various paradigms (eg, psychosomatic) with particular emphasis on the medical model. Specific illnesses associated with stress such as tension headaches and arthritis are discussed. Chapter 4 lists measurement approaches from the perspective of measures of stress, the stress process, and means of assessing physiologic, cognitive, and behavioral responses to stress.

Chapter 5 covers a major perspective of the author's regarding interventions for stress management. The author reviews a cognitive-behavioral approach to stress management with treatment focusing on developing coping skills rather than working only to eliminate stressors. Given that one coping skill may relieve many stressors, or one stressor may require many coping skills, Dr Cotton suggests a planning grid which can be utilized to categorize stressors and plan specific treatment strategies. The final four chapters of the book are concerned with actual stress management therapies including relaxation therapies; life style management, including smoking cessation and alcohol issues; and cognitive and behavioral strategies. In each of these chapters, Dr Cotton has highlighted major therapies within the field and addressed them from an overview perspective. The reader is thus aware of the therapies, has some perspective as to their use, but may not always be privy to the details whereby they might be carried out.

The content of the book is accurate, with references provided for each chapter. In some cases, further discussion and/or referencing of material, eg, smoking cessation literature, would be helpful to readers interested in details regarding particular strategies and available resources.

This is a clinically oriented book written in a clear manner. The author begins with careful discussion of definitions of terms and paradigms addressing particular clinical interventions, making this an appropriate text for the physician with expertise in areas other than stress management.

As previously noted, many of the other texts regarding stress management are written more from a research perspective. Dr Cotton's approach is respectful of what research has identified as important to management, and is focused on clinically applicable aspects of care. The author meets her stated objective of aiding the therapist to recognize problems of stress and proposing a model of stress management.

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TRAUMA AND THE VIETNAM WAR GENERATION: REPORT OF FINDINGS FROM THE NATIONAL VIETNAM VETERANS READJUSTMENT STUDY

By Richard A. Kulka, PhD, William E. Schlenger, PhD, John A. Fairbank, PhD, Richard L. Hough, PhD, B. Kathleen Jordan, PhD, Charles R. Marmar, MD, and Daniel S. Weiss, PhD. 352 pp. New York, Brunner/Mazel Publishers, 1990. $19.95 (hardcover)

This book is valuable for the practitioner wishing a better understanding about post-traumatic stress disorders in Vietnam veterans. In 1983, the United States Congress mandated that a study be conducted to examine the ongoing effects of the Vietnam war on its veterans. In response to this directive, the National Vietnam Veterans Readjustment Study (NVVRS) was undertaken. This volume presents the results of the study. It provides overwhelming evidence for the existence and prevalence of post-traumatic stress disorders among Vietnam veterans. More so than any other book published about Vietnam veterans and post-traumatic stress disorders, this one brings together a number of consultants, collaborators, and colleagues who are noted experts and authorities in the field of post-traumatic stress as it applies to Vietnam veterans.

This is an excellent publication for all individuals who have a need to know more about the effects of post-traumatic stress disorders. Mental health professionals, physicians, nurses, and even lawyers (who are involved with legal problems) will gain insight into the problems experienced by this population. Additionally, this publication provides an excellent overview of those who served during the Vietnam war, their stress reaction symptoms during the war, clear guidelines for treating post-traumatic stress disorders, a historical insight into the development of post-traumatic stress disorders, and differential diagnoses regarding post-traumatic stress disorders. It also addresses readjustment problems that many veterans have gone through during the last 20 years, including physical problems that might present to a health practitioner. This book also looks toward the future with some insights as to where we will go with the population that survived the Vietnam conflict, family members, and children of the survivors of the Vietnam war generation. Research that follows this study should address racial and ethnic differences in postwar adjustment and the impact on family systems.

Perhaps one of the best chapters in this whole interesting publication is the epilogue offered in the last chapter. Dr David Grady, a decorated and twice wounded Vietnam marine rifleman, offers this chapter as a self-help guide. Dr Grady has done extensive professional mental health work with the Vietnam veteran population regarding readjustment problems. He discusses how most Vietnam veterans have successfully readjusted after their wartime experience, reviewing symptomatology and intrusive thoughts. He goes on to discuss, in probably the most comprehensive manner ever presented in a publication of this kind, treatment resources for outpatient clinics, mental health clinics, day hospitals, and day treatment centers available through the Veterans Administration, the Disabled American Veterans, and the Paralyzed Veterans Association. He also discusses in detail the veteran centers that are currently available and provides much detail such as addresses, names of contact individuals, and their phone numbers.

This publication brings together the long-awaited results of a study that provides an excellent view of the Vietnam war generation and the effects of post-traumatic stress disorders among them. It is a concise and clear guide for mental health professionals and/or medical professionals requiring further understanding of the trauma of the Vietnam war.

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STRANGERS AT HOME: VIETNAM VETERANS SINCE THE WAR

Edited by Charles R. Figley, PhD, and Seymour Leventman, PhD. 416 pp. New York, Brunner/Mazel Publishers, 1990. $26.95 (paperback)

This book brings together a multidisciplinary collection of 18 essays and studies that provide a remarkable examination of the psychological, social, and economic aspects of Vietnam veterans. First published in 1980 and now back in print, *Strangers at Home* continues to provide observations, findings, and predictions that hold true even today.

In 1980, the third revision of the *Diagnostic and Statistic Manual of Mental Disorders* was published by the American Psychiatric Association. Included for the first time in this manual was a concept entitled "Post-traumatic stress disorder," which was defined as a collection of stress reaction symptoms linked to some extraordinary, highly stressful event, and which was seen most commonly in returning Vietnam veterans. After the first edition of *Strangers at Home* was published, a number of historical events occurred that brought together Vietnam veterans for the first time in the five years since the war had ended. It was the same year that the Veterans Administration made "post-traumatic stress disorder" a compensable medical condition, and additionally opened up a nationwide system of centers to counsel Vietnam veterans. Other developments closely followed, including the passage of federal laws to ensure that Vietnam veterans' rights in critical services were protected and extended.

This book contains a priceless amount of knowledge regarding the war and the Vietnam veteran. It includes, for example, oral history by Clark Smith describing the stress and the day-to-day living conditions of the Vietnam veteran. It also reviews how other war veterans have been treated since the beginning of time in a chapter called "The Victors and the Vanquished" by Willard Waller. It goes on to talk about how veterans become immigrants in their own native land after returning home from fighting in foreign countries. Other excellent chapters include John Wilson's chapter, "Conflict Stress and Growth: The Effects of War on Psychosocial Development Among Vietnam Veterans," which describes the Forgotten Warrier project which was funded by the Disabled American Veterans and started Dr John Wilson on a ten-year research data base on Vietnam veterans and the effects of the war among Vietnam veterans. The last two chapters of this vital and important book were written by Dean Philips, a long-time advocate for veterans' rights, and himself a decorated combat Vietnam veteran. It is ironic, perhaps, that Dean is no longer with us, having passed away in 1985 from a non-Hodgkin's lymphoma which many believe was linked to Agent Orange to which he was exposed in Vietnam. Dean Philips was a major force behind the scenes within the Veterans Administration. His work resulted in several major programs for Vietnam veterans. It seems only fitting that Dr Charles Figley and Dr Seymour Leventman have dedicated this second edition of *Strangers At Home* to Dean K. Philips.

This text is an important and valuable analysis of governmental and institutional responses to the returning veterans and their psychosocial, economic, political, and ideologic perspectives, not only in the war but also, more importantly, when they returned home. It provides a powerful look at the psychological complexity and brutality of the Vietnam war as it is seen through the eyes of its participants.

This text is an absolute must for any health practitioner, social worker, and/or other allied health individuals who may be working with Vietnam veterans, particularly with Vietnam veterans in therapy. It also will allow the practitioner to have a clear understanding of how to deal with history gathering, psychosocial assessment, and understanding the veteran's perspective of what it felt like to live through the Vietnam war. This text has become the classic of the Brunner/Mazel Psychosocial Stress Series and should be thought of as a must for any practitioner dealing with Vietnam veterans.

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HEALING AND RESTORING: HEALTH AND MEDICINE IN THE WORLD'S RELIGIOUS TRADITIONS

Edited by Lawrence E. Sullivan. 468 pp. New York, Macmillan Publishing Co, 1989. $35.00 (hardcover)

Medicine reflects the culture of the people among which it evolves. Traditions of medical care vary with the beliefs and cultural assumptions of the people. Religious customs and beliefs are intimately linked with the evolution of models of health care. With ethnic minorities becoming more prominent in the population demographics of the United States, physicians will certainly encounter more patients with different religious convictions. Dr Sullivan has compiled an excellent text that will aid medical practitioners in learning about the religious aspects of culturally diverse people. His text is also a scholarly resource for students of religion.

Healing and Restoring is a series of 15 essays, each authored by an academic expert, prefaced by an insightful introduction by the editor. Dr Sullivan clearly states that the subject matter cannot be comprehensively covered in any single volume. Instead, the editor builds a mosaic from the contributing authors, linking history, creed, and custom under the theme of health and well-being. Case presentations, including a psychiatric assessment of an anxious Hindu woman, the use of public temples of Japan as a resource for pregnant Shinto women, the interface of depression, meditation, and possession in Sri Lanka, provide clinical relevance. The chapters cover several broad areas: Buddhism as it relates to traditions of life cycle in China and of contemporary health care in Japan; Hinduism and Ayurvedic health care in India and Sri Lanka; the historic and living traditions of Islam; religion and healing in Ghana and South-Central Africa; religious cultures in the islands of Haiti and Hawaii; and Native American traditions in North, Central, and South America. The interface of religion with varieties of life experiences, such as birth, death and life passages, sexuality, pain, illness and suffering, mental health, dignity and self-esteem, is covered in a purposeful style.

This book is a sophisticated text, requiring thoughtful attention and careful reading; it is not for the cursory reader who might be looking for an introductory anthology. The writing is clear and concise, but often technical. Each chapter is extensively referenced, and a concise guide to further reading complements each chapter at the end of the text. In balancing brevity with depth, some omissions are to be expected. The religion and health practices of Sikhs are not covered, nor are the religious health customs of the Jews; Jainism is only mentioned.

This text is suitable for upper level courses in anthropology, religion, medicine, and nursing; health care providers with humanistic concerns for understanding the depths of the human condition will surely benefit from reading it. Overall, it is a ready reference for the religious aspects of the biopsychosocial model in clinical settings. It extends our awareness beyond current medical interventions to an understanding of the positive attributes of well-being and healing, the psycho-spiritual aspects of symbol and faith, and the cosmologic dimensions of belief. This book is well suited for the task of overcoming the ethnocentric and medicocentric bias of health care providers. It is an aid to melding the tenets of science and medicine with the disparate beliefs of diverse people seeking health care. As such, it is a thoughtful, person-oriented guidebook which complements the technologic approach of modern medicine.

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AIDS and cytomegalovirus-related appendicitis

Among patients with AIDS, an estimated 90% will become infected with cytomegalovirus (CMV). AIDS-associated opportunistic infections with CMV have been shown to involve almost every organ in the body. Lin et al describe an AIDS patient who presented with acute, right-sided abdominal pain, which was diagnosed as appendicitis on laparotomy (*Am J Med* 1990; 89:377–379).

Microscopic evaluation of the appendiceal tissue was remarkable for a "striking proliferation of endothelial cells, many of which were enlarged." Intranuclear and intracytoplasmic inclusion bodies characteristic of CMV infection were present; these stained positive for CMV on in situ DNA hybridization using a DNA probe. Although no blood or appendiceal cultures grew CMV, a urine culture was positive. The diagnosis of CMV was further confirmed by the serum CMV titer—1:256 after removal of the appendix, compared to a baseline of 1:8 one month previously.

Number of AIDS patients on Medicaid is increasing

The "Medicaidization of AIDS" is a phrase coined by Green and Arno to describe the trend of shifting payer distribution for patients with AIDS away from private insurance and toward Medicaid (*JAMA* 1990; 264:1261–1266). "The Medicaidization of AIDS implies a shift of financial liability to the public sector at a time when resources are severely strained," they conclude.

The authors evaluated the financing of AIDS-related care in New York, San Francisco, and Los Angeles for the period 1983–1988. There was a significant ($p < 0.001$) increase in the proportion of hospital costs for AIDS patients financed by Medicaid and dramatic reductions in the amounts paid by private insurance. For example, in San Francisco, the proportion of hospital costs for AIDS financed by private insurance in 1987 was nearly half of that paid in 1983. In all three cities, blacks and Hispanics were significantly ($p < 0.001$) more likely to receive Medicaid than whites. Yet the "Medicaidization" trend is equally evident for all racial groups.

These findings raise the question of access to care for AIDS patients. As Medicaid rates to physicians are substantially lower than those of private insurance, private physicians are far less likely to accept the lower rates and thereby may be less willing to include AIDS patients in their practices. Green and Arno found that AIDS patients on Medicaid were more likely to enter a hospital via the emergency room, rather than via admission by a private physician. In addition, Medicaid recipients are more likely to stay in public hospitals. "In fact, the differences [in admission to public versus private hospitals] are so large (fivefold in New York; nearly 60-fold in Los Angeles)," state the authors, "as to suggest nearly distinct systems of care depending on the type of insurance."

Estimates of the lifetime medical care costs of treating patients with AIDS average $40,000–$75,000. Whereas Medicaid pays for nearly 11% of total health care costs in the United States, it pays for an estimated 25% of the costs of caring for AIDS patients. This creates an enormous strain on the Medicaid system.

Increasing Medicaid reimbursements to physicians could help stifle the trend toward a two-tiered system of care in which Medicaid recipients may not have access to the quality of care and the new and experimental treatments for AIDS that would be available through an office-based private physician.

Another contributing factor to the "Medicaidization" problem is that the debilitating effects of AIDS frequently force patients to give up their employment and thus lose access to private insurance. The authors propose that "Policies designed to prevent the loss of employment-based private insurance would slow or reverse the trend to public financing."

WHO embarks on vaccination campaign

At the Children's Summit in New York City in September, the World Health Organization (WHO) introduced the Children's Vaccine Initiative, a program for achieving vaccination of children worldwide against such deadly diseases as polio, measles, and tetanus (*Nature* 1990; 347:218). Joined in its efforts by the United Nations Development Programme and the United Nations Children's Fund, WHO has established a minimum goal of $15 million per year for ten years. The funds would promote vaccine research and development.

The initiative's primary goals include making existing vaccines, such as the polio vaccine, more heat-stable and affordable, thereby making them more readily available to Third World countries. Other goals range from developing combination, single-dose vaccines that would reduce the number of necessary immunizations, to the development of new vaccines against diseases such as diarrhea and acute respiratory infections.

Gene therapy ushers in an era of hope

There are many diseases, such as diabetes, Parkinson's disease, hemophilia, and certain types of cancer, for which gene therapy holds promise as a cure, rather than simply a short term treatment that must be repeated over and over again with uncertain effectiveness. The dream of gene therapy—replacing defective genes with normal replacement genes which can correctly control protein synthesis—is now a reality.

The first patient, a four-year-old girl suffering from adenosine deaminase (ADA) deficiency, underwent gene therapy in late September (*Time*, September 24, 1990, pp 74–76). She received an intravenous infusion of cells containing healthy genes for ADA, the lack of which leaves the body's immune system vulnerable to destruction by toxic compounds in the bloodstream. Children with ADA deficiency, like the famous "boy in the bubble," have virtually no natural defenses against disease, and they usually die in early childhood.

A successful outcome of this first trial with gene therapy could open the door for further testing of this experi-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

mental mode of treatment. Numerous obstacles still face gene therapy researchers, such as how to ensure that the genes are incorporated into the correct cells and into the right sites within the genetic material of those cells, how to control the activity of the replacement genes so they neither overproduce nor underproduce the target protein, and how to prevent the new genes from disrupting other gene functioning and from activating cancer-causing genetic segments. The risks are many, but the rewards could be dramatic. The next proposed application of gene therapy, now awaiting final approval by the US Food and Drug Administration (FDA), is aimed at treating patients with advanced cases of melanoma.

Warning: Rapid strep A tests may not be reliable

Rapid tests for diagnosing group A beta-hemolytic streptococcal pharyngitis, although permitted on the market by the Food and Drug Administration (FDA), may be "unacceptably inaccurate" compared to agar cultures, warn Gerber et al (*Pediatrics* 1990; 86:457–459). Most such tests, which employ methods similar to existing, previously approved technology, require only that the manufacturer notify the FDA prior to commercial distribution. The FDA does not evaluate the performance and accuracy of these tests.

In 1986, these authors evaluated the Strep-A-Fluor (Bio-Spec Inc, Dublin, Calif) rapid diagnostic test for group A beta-hemolytic streptococcal pharyngitis, which incorporated a new enzyme fluorescence procedure. "We found that the Strep-A-Fluor test was unacceptably inaccurate when compared with blood agar cultures, and it was subsequently removed from the market as a test for group A streptococci," the authors noted.

In this current report, Gerber and colleagues compared the accuracy of the recently released Chromagen (Diversified Diagnostic Industries, Moraga, Calif) rapid test with that of blood agar plate cultures. Chromagen is a modified form of the Strep-A-Fluor test, using a colorimetric instead of fluorometric end point.

Two swab samples were taken from the posterior pharnyx and tonsils of each of 111 patients with symptoms suggestive of group A beta-hemolytic streptococcal pharyngitis. One swab was for the Chromagen test, and the second swab was for a blood agar plate culture. The Chromagen test is performed directly on the throat swab.

"The Chromagen test had a sensitivity of 86%, specificity of 27%, positive predictive value of 70%, and negative predictive value of 50% when compared with the blood agar plate culture." The authors found these results to be unacceptable, and they caution physicians that the availability of a rapid strep test does not mean that the test is either FDA approved or accurate.

Balloon angioplasty finds application in gene therapy

Balloon angioplasty, a technique used by cardiologists to open clogged blood vessels, is proving useful in targeting sites for gene therapy (*Science News* 1990; 138:161–176). While some cells containing healthy replacement genes can deliver their therapeutic protein products while circulating in the blood stream, and some cells can migrate on their own to the correct site of action, future gene therapy procedures may require more precise localization of cells containing the replacement genes.

Using balloon angioplasty in pigs, researchers inserted a catheter containing two uninflated balloons into a blood vessel. Inflation of both balloons temporarily blocked off a short section of the vessel, into which the researchers injected many copies of an easily traced gene. The genes entered the cells that line the blood vessels, remained active for as many as 21 weeks, and appeared not to travel to other parts of the body.

One example of this procedure's potential utility would be to introduce genes for anti-cancer compounds into blood vessels that feed tumors. However, there is skepticism that sufficient gene activity is attainable in such a defined region.

Cardiac effects of carbon monoxide exposure

High levels of atmospheric carbon monoxide can produce myocardial ischemia and cardiac dysfunction. Sheps and colleagues have now demonstrated that low levels of carbon monoxide exposure—such as experienced when living in an urban environment, exercising in areas of high automobile emissions, or working in tunnels or high traffic areas—can increase ventricular arrhythmias in patients with coronary artery disease (*Ann Intern Med* 1990; 113:343–351).

This finding, based on data collected from 41 patients with coronary artery disease, mean age of 62.8 years, was derived from measurement of carboxyhemoglobin in venous blood and electrocardiographic recordings. Venous carboxyhemoglobin levels of 4% and 6% were the target levels for study. Signs of myocardial ischemia have been demonstrated in patients with coronary disease at carboxyhemoglobin levels of 2–4%. In an accompanying editorial, Walden and Gottlieb note that pollution levels in New York City exceed Environmental Protection Agency guidelines about 80 days per year (pp 337–338).

Sheps et al found increased frequency and severity of ventricular premature depolarization during exercise after sufficient carbon monoxide exposure to produce 6% carboxyhemoglobin levels. Four percent levels yielded no increases. A more dramatic, nearly three-fold increase in complex arrhythmias was evident. Cardiac arrhythmia can result in sudden death, and the relative risk for sudden death is greatest during exercise. Is there a link between carbon monoxide exposure, exercise-related arrhythmia, and sudden death? There is as yet no clear relationship between ventricular arrhythmias and cardiac mortality. Such a statement, therefore, would require further study.

Luis Biempica, MD, Bronx. Died May 1990; age 65. Dr Biempica was a 1952 graduate of the Facultad de Ciencias Medicina de la Universidad de Buenos Aires, Buenos Aires, Argentina. He was a Diplomate of the American Board of Pathology and a Fellow of the American College of Pathologists. His memberships included the New York Pathological Society, the American Association of Pathologists & Bacteriologists, the Bronx County Medical Society, and the Medical Society of the State of New York.

Mortimer J. Blumenthal, MD, New York, Died August 5, 1990; age 69. Dr Blumenthal was a 1945 graduate of the Boston University School of Medicine, Boston, Massachusetts. He was a Diplomate of the American Board of Psychiatry and Neurology and a Fellow of the American Psychiatric Association. His memberships included the American Academy of Child & Adolescent Psychiatry, the American Psychoanalytic Association, the New York County Medical Society, and the Medical Society of the State of New York.

Laurence Griffin Bodkin, MD, Larchmont. Died July 11, 1990; age 91. Dr Bodkin was a 1924 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Surgery and a Diplomate of the American Board of Colon and Rectal Surgery. He was a Fellow of the American College of Surgeons and a Qualified Fellow of the International College of Surgeons. His memberships included the American Society of Colon and Rectal Surgeons, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Arthur S. Coriale, MD, Utica. Died May 11, 1990; age 80. Dr Coriale was a 1937 graduate of the University of Rochester School of Medicine-Dentistry, Rochester. He was a Qualified Fellow of the International College of Surgeons. His memberships included the New York Academy of Medicine, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Selvan Davison, MD, Bayside. Died August 16, 1990; age 78. Dr Selvan was a 1937 graduate of the Faculty of Medicine University of Edinburgh, Edinburgh, Scotland. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. His memberships included the New York Academy of Medicine, the American Rheumatism Association, the New York County Medical Society, and the Medical Society of the State of New York.

Caesar De Rostaing, MD, Oceanside. Died August 3, 1990; age 79. Dr De Rostaing was a 1943 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a Fellow of the American College of Obstetricians and Gynecologists. His memberships included the New York Academy of Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

William Engler, MD, Miami Beach, Florida. Died May 26, 1990; age 96. Dr Engler was a 1926 graduate of the George Washington University School of Medicine, Washington, DC. His memberships included the American Academy of Family Physicians, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Wilmer Price Fitch, MD, Mamaroneck. Died August 5, 1990; age 85. Dr Fitch was a 1935 recipient of the Registrable Qualification Granted By the English Conjoint Board. He was a Fellow of the American Academy of Family Physicians. His memberships included the New York Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Hans A. Hartmann, MD, New York. Died August 12, 1990; age 80. Dr Hartmann was a 1936 graduate of the Medizinische Fakultaet der Ludwig Maximiliams Universitaet, Muenchen, Bayern, Germany. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Noemi G. Hochman, MD, Rochester. Died July 29, 1990; age 93. Dr Hochman was a 1925 graduate of the Akademia Medyczna Wydzial Lekarski, Warszawa, Poland. She was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Saul P. Lehv, MD, Delray Beach, Florida. Died June 24, 1990; age 82. Dr Lehv was a 1930 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Benjamin Leighton, MD, Phoenix, Arizona. Died April 29, 1990; age 85. Dr Leighton was a 1936 graduate of the Medizinische Fakultaet der Universitaet Basel, Basel, Switzerland. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Benjamin Bernard Levine, MD, New York. Died August 1, 1990; age 86. Dr Levine was a 1928 graduate of the New York Medical College, New York. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the New York County Medical Society, and the Medical Society of the State of New York.

Duncan R. Mc Cuaig, MD, West Palm Beach, Florida. Died July 7, 1990; age 87. Dr Mc Cuaig was a 1926 graduate of the McGill University Faculty of Medicine, Montreal, Quebec, Canada. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Lloyd Calvin Peters, MD, Binghamton. Died July 29, 1990; age 67. Dr Peters was a 1947 graduate of the McGill University Faculty of Medicine, Montreal, Quebec, Canada. He was a Fellow of the American College of Surgeons. His memberships included the American Geriatrics Society, the American Society of Abdominal Surgeons, the Broome County Medical Society, and the Medical Society of the State of New York.

Arte I. Pierce, MD, New York. Died August 7, 1990; age 35. Dr Pierce was a 1980 graduate of the University of Michigan Medical School, Ann Arbor, Michigan. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Norman P. Pleshette, MD, New York. Died July 31, 1990; age 88. Dr Pleshette was a 1928 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a Fellow of the American College of Surgeons and a Fellow of the American College of Obstetricians and Gynecologists. His memberships included the New York Academy of Medicine, the New York Obstetrical Society, the New York County Medical Society, and the Medical Society of the State of New York.

Albert Jay Ritzmann, MD, Brooklyn. Died July 15, 1990; age 89. Dr Ritzmann was a 1930 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Surgeons. His memberships included the American Thyroid Association, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

George Rose, MD, Lake Worth, Florida. Died July 18, 1990; age 85. Dr Rose was a 1931 recipient of the Registrable Qualification Granted by Scottish Conjoint Board. His memberships included the American Geriatrics Society, the American Academy of Family Physicians, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Patrick P. Scarantino, MD, Garden City. Died August 11, 1990; age 51. Dr Scarantino was a 1965 graduate of the State University of New York Health Science Center at Brooklyn. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a Fellow of the American College of Obstetricians and Gynecologists. His memberships included the American Fertility Society, the Nassau County Medical Society, and the Medical Society of the State of New York.

Nathan Scharf, MD, New City. Died August 6, 1990; age 75. Dr Scharf was a 1940 graduate of the Hahnemann Medical College of Philadelphia, Philadelphia, Pennsylvania. He was a member of the Medical Society of the County of Rockland and the Medical Society of the State of New York.

Benjamin Silberg, MD, Lynbrook. Died August 12, 1990; age 90. Dr Silberg was a 1926 graduate of the New York Medical College, New York. He was a Diplomate of the American Board of Pediatrics and a Diplomate of the American Board of Allergy and Immunology. He was a Fellow of the American College of Allergists and a Fellow of the American Academy of Pediatrics. His memberships included the American Academy of Allergy, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

John James Snodgrass, MD, Manhasset. Died August 27, 1990; age 70. Dr Snodgrass was a 1945 graduate of the Cornell University Medical College, New York. He was a Diplomate of the American Board of Radiology and a Fellow of the American College of Radiology. His memberships included the American Roentgen Ray Society, the Radiological Society of North America, Inc, the Nassau County Medical Society, and the Medical Society of the State of New York.

Joseph Maurice Spitzer, MD, Scarsdale. Died August 16, 1990; age 82. Dr Spitzer was a 1937 graduate of the Medizinische Fakultaet Rheinischen, Friedrich Wilhelms Universitaet, Bonn, Nordrhein Westfalen, Germany. He was a Diplomate of the American Board of Internal Medicine. He was a Fellow of the American College of Physicians and a Fellow of the American College of Chest Physicians. His memberships included the New York Academy of Medicine, the New York Pathological Society, the American Thoracic Society, the New York County Medical Society, and the Medical Society of the State of New York.

Samuel Steigbigel, MD, Brooklyn. Died July 24, 1990; age 91. Dr Steigbigel was a 1928 graduate of the State University of New York Health Science Center at Brooklyn. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Francis C. Symonds, MD, New York. Died August 9, 1990; age 64. Dr Symonds was a 1951 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Surgery and a Diplomate of the American Board of Plastic Surgery. He was a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the American Society of Plastic & Reconstructive Surgery, the American Association of Plastic Surgeons, the American Burn Association, the New York County Medical Society, and the Medical Society of the State of New York.

Zsolt Szekely, MD, Dunkirk. Died January 16, 1990; age 46. Dr Szekely was a 1969 graduate of the Albert Einstein College of Medicine-Yeshiva University, Bronx. He was a Diplomate of the American Board of Radiology. He was a member of the Medical Society of the County of Chautauqua and the Medical Society of the State of New York.

Joseph Alan Tagliamonti, MD, Glen Cove. Died July 26, 1990; age 50. Dr Tagliamonti was a 1969 graduate of the Facolta di Medicina e Chirurgia dell-'Universita di Bologna, Bologna, Italy. He was a Diplomate of the American Board of Pathology. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Harvey Burton Taterka, MD, New York. Died July 22, 1990; age 59. Dr Taterka was a 1956 graduate of the New York University School of Medicine, New York. He was a Diplomate of the American Board of Ophthalmology and a Fellow of the American College of Surgeons. His memberships included the American Academy of Ophthalmology and Otolaryngology, the New York County Medical Society, and the Medical Society of the State of New York.

Nathan Thaler, MD, Brooklyn. Died August 12, 1990; age 88. Dr Thaler was a 1928 graduate of the Tufts University School of Medicine, Boston, Massachusetts. He was a Diplomate of the American Board of Otolaryngology and a Qualified Fellow of the International College of Surgeons. His memberships included the American Academy of Otolaryngology, the American Academy of Facial Plastic & Reconstructive Surgery, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

— ERRATUM —

The September 1990 issue of the *Journal* incorrectly reported the death of Henry Greenbaum, MD, of Forest Hills, NY. The *Journal* regrets the error.

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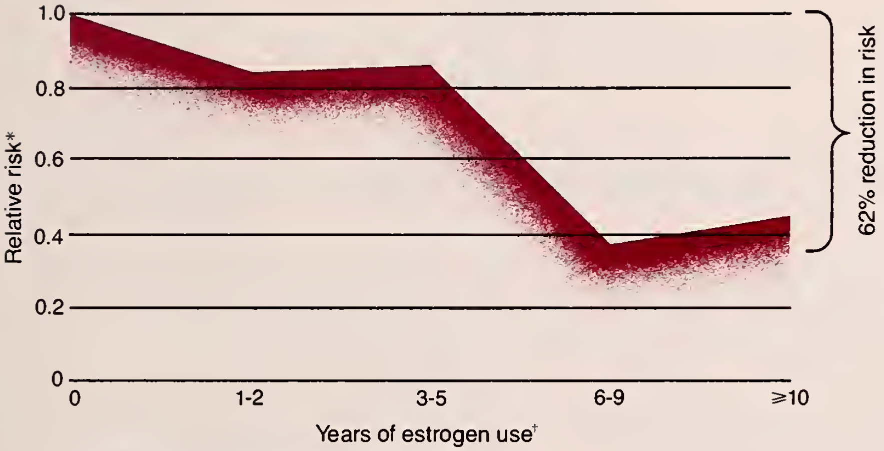

*Standardized for age group (50 to 59, 60 to 69, and 70 to 74 years), history of hysterectomy, and current use versus past use of estrogens
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Adapted from Weiss et al[1]

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Please see brief summary of prescribing information on next page.

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> Estrogen therapy during pregnancy is associated with an increased risk of congenital defects in the reproductive organs of the male and female fetus, an increased risk of vaginal adenosis, squamous-cell dysplasia of the uterine cervix, and vaginal cancer in the female later in life. The 1985 DES Task Force concluded that women who used DES during their pregnancies may subsequently experience an increased risk of breast cancer. However, a causal relationship is still unproven, and the observed level of risk is similar to that for a number of other breast cancer risk factors.
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INDICATIONS AND USAGE: Moderate-to-severe vasomotor symptoms associated with the menopause. (There is no evidence that estrogens are effective for nervous symptoms or depression which might occur during menopause and they should not be used to treat these conditions.) Prevention and management of osteoporosis (abnormally low bone mass). Atrophic vaginitis. Atrophic urethritis. Hypoestrogenism due to hypogonadism, castration or primary ovarian failure.

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae.

PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING).

CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions: 1. Known or suspected pregnancy (see Boxed Warning). 2. Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease. 3. Known or suspected estrogen-dependent neoplasia. 4. Undiagnosed abnormal genital bleeding. 5. Active thrombophlebitis or thromboembolic disorders. 6. Estrogen replacement therapy has not been reported to increase the risk of thrombophlebitis and/or thromboembolic disease. However, there is insufficient information regarding women who have had previous thromboembolic disease.

PREMARIN Tablets and Vaginal Cream should not be used in patients hypersensitive to their ingredients.

WARNINGS: Some studies suggest a possible increased incidence of breast cancer in women taking higher doses of estrogen for prolonged time periods. The majority of studies have not shown an association with usual estrogen replacement doses. Endometrial cancer risk among estrogen users was about 4-fold or greater than in non-users, and appears dependent on treatment duration and estrogen dose. In patients on combined estrogen-progestin therapy, this risk appears to be decreased. (See PRECAUTIONS below.)

Estrogen therapy during pregnancy is associated with an increased risk of fetal congenital reproductive tract disorders.

A 2.5-fold increase in the risk of surgically confirmed gall bladder disease in women receiving postmenopausal estrogens has been reported.

Large doses of estrogen such as those used to treat prostate and breast cancer have been shown to increase the risk of non-fatal myocardial infarction, pulmonary embolism, and thrombophlebitis in men. This cannot necessarily be extrapolated to women. However, to avoid theoretical cardiovascular risk caused by high estrogen doses, the doses for estrogen replacement therapy should not exceed the recommended dose.

Blood pressure should be monitored with estrogen use, especially if high doses are used.

Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases.

PRECAUTIONS: The addition of a progestin for 7 or more days of a cycle of estrogen administration reportedly lowers the incidence of endometrial hyperplasia. Studies of endometrium suggest that 10 to 13 days of progestin are needed to provide maximal endometrial maturation and elimination of hyperplastic changes. Additional risks, such as adverse effects on carbohydrate and lipid metabolism, may be associated with the inclusion of progestin in estrogen replacement regimens. The choice of progestin and dosage may be important in minimizing these adverse effects.

Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear. As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed. Conditions influenced by fluid retention, such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation. Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding and mastodynia. Pre-existing uterine leiomyomata may increase in size during estrogen use. Estrogens should be used with care in patients with impaired liver function, renal insufficiency, or metabolic bone diseases associated with hypercalcemia.

The following drug/laboratory test interactions have been reported, some only with estrogen-progestin combinations (oral contraceptives):

1. Increased prothrombin and factors VII, VIII, IX, and X; decreased antithrombin 3; increased norepinephrine-induced platelet aggregability.
2. Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by T_4 levels determined by column or by radioimmunoassay. Free T_3 resin uptake is decreased, reflecting the elevated TBG; free T_4 concentration is unaltered.
3. Impaired glucose tolerance.
4. Reduced response to metyrapone test.
5. Reduced serum folate concentration.

MUTAGENESIS AND CARCINOGENESIS: Long-term, continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver.

PREGNANCY CATEGORY X: Estrogens should not be used during pregnancy. See **CONTRAINDICATIONS** and Boxed Warning.

NURSING MOTHERS: As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk.

ADVERSE REACTIONS: The following have been reported with estrogenic therapy: changes in vaginal bleeding pattern and abnormal withdrawal bleeding or flow, breakthrough bleeding, spotting, increase in size of uterine fibromyomata, vaginal candidiasis, change in amount of cervical secretion, tenderness or enlargement of breasts; nausea, vomiting, abdominal cramps, bloating, cholestatic jaundice; chloasma or melasma that may persist when drug is discontinued, erythema multiforme, erythema nodosum, hemorrhagic eruption, loss of scalp hair, hirsutism; steepening of corneal curvature, intolerance to contact lenses; headache, migraine, dizziness, mental depression, chorea; increase or decrease in weight, reduced carbohydrate tolerance; aggravation of porphyria; edema, changes in libido.

ACUTE OVERDOSAGE: May cause nausea and vomiting.

DOSAGE AND ADMINISTRATION:

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1. *Given cyclically for short-term use only.* For treatment of moderate-to-severe vasomotor symptoms, atrophic vaginitis, or atrophic urethritis associated with the menopause (0.3 mg to 1.25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible. Administration should be cyclic (eg, three weeks on and one week off). Attempts to discontinue or taper medication should be made at three- to six-month intervals.
2. *Given cyclically:* Hypoestrogenism. Osteoporosis.

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Osteoporosis—0.625 mg daily. Administration should be cyclic (eg, three weeks on and one week off).

PREMARIN® Brand of conjugated estrogens Vaginal Cream

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Attempts to discontinue or taper medication should be made at three- to six-month intervals.

Usual dosage range: 2 g to 4 g daily, intravaginally, depending on the severity of the condition.

Patients with an intact uterus who are treated with either PREMARIN Tablets or Vaginal Cream should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.

Revised August 21, 1989

Reference:

1. Weiss NS, Ure CL, Ballard JH, et al: Decreased risk of fractures of the hip and lower forearm with postmenopausal use of estrogen. *N Engl J Med* 1980;303:1195-1198

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(*continued from p 3A*)

its. New York Hilton. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

Dec 6–7. **Update in Gastroenterology and Hepatology.** College of Physicians & Surgeons of Columbia University. Contact: Office of Continuing Education, College of Physicians & Surgeons, 630 West 168th St, New York, NY 10032. Tel: (212) 305-3682.

Dec 13–15. **Operative Gynecologic Endoscopy.** 16 Cat I Credits. New York. Contact: American College of Obstetricians and Gynecologists, 409 12th St, SW, Washington, DC 20024. Tel: (202) 638-5577.

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Dec 7–9. **Advanced Seminars in Diagnostic Imaging.** 18 Cat I Credits. Ritz-Carlton Resort Hotel, Laguna Niguel. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

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Dec 15–22. **Current Issues in Medicine.** Lionsquare Lodge, Vail. Contact: Professional Seminars, Inc, PO Box 012318, Miami, FL 33101. Tel: (305) 547-6411.

AROUND THE WORLD

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Dec 16–20. **Third International Symposium on Noninvasive Cardiology.** Tel Aviv. Contact: Third International Symposium on Noninvasive Cardiology, PO Box 50006, Tel Aviv 61500, Israel. Tel: (03) 654571.

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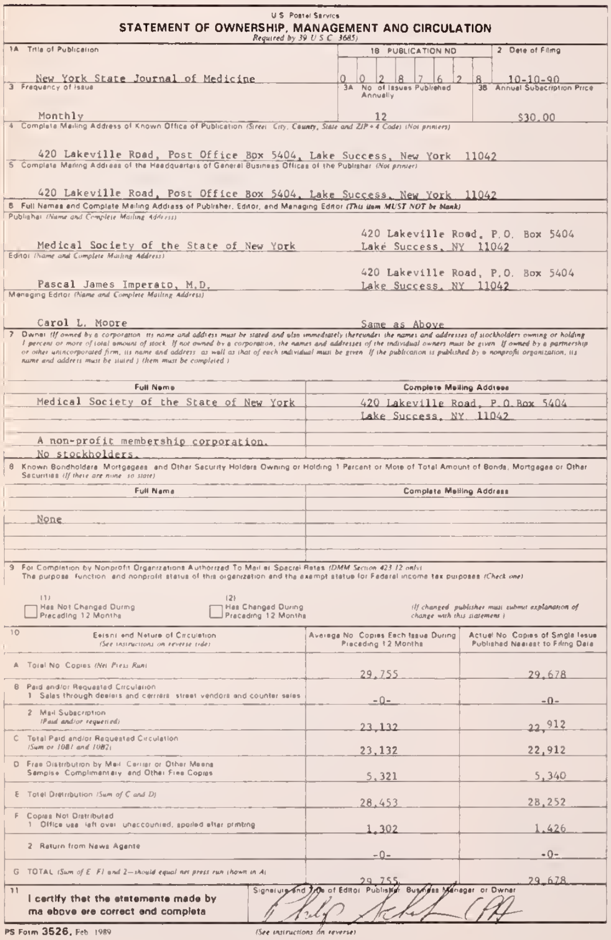

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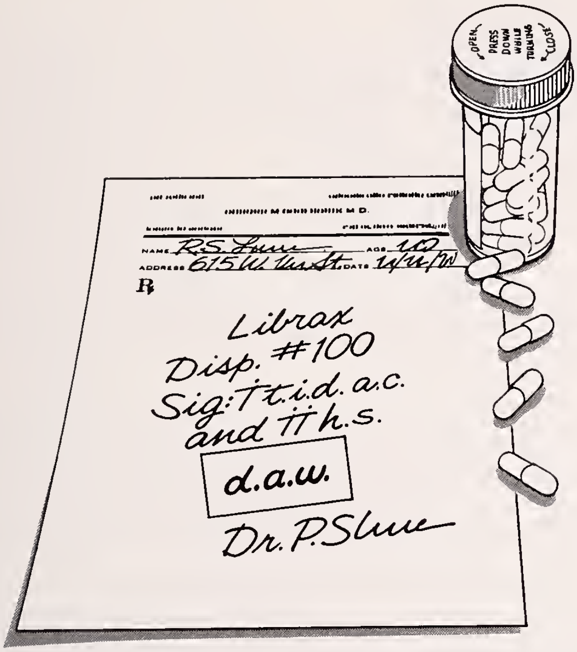

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As with all anticholinergics, inhibition of lactation may occur.

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

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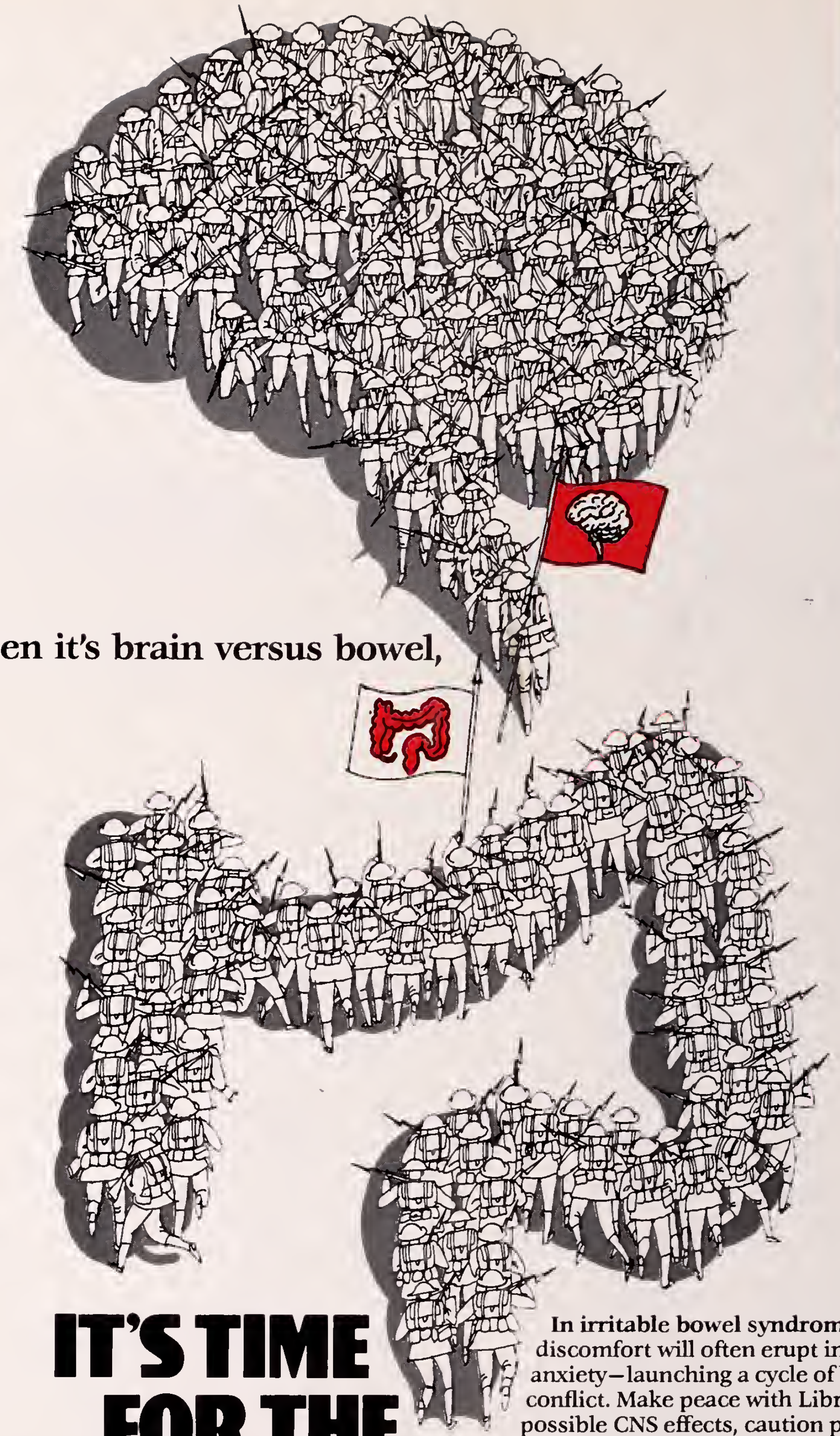

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New York State Journal of Medicine

MEDICAL SOCIETY OF THE STATE OF NEW YORK
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VOL 90
NO 12
DECEMBER
1990

POTT'S DISEASE IN URBAN POPULATIONS

VIOLENT PATIENTS IN A GENERAL HOSPITAL

MULTIPLE ADDICTIONS: CO-SYNCHRONOUS USE OF ALCOHOL AND DRUGS

CENTRAL NERVOUS SYSTEM MALIGNANCIES IN CHILDREN

DISSEMINATED INFECTION CAUSED BY *FUSARIUM SOLANI*

LUNG CARCINOMA PRESENTING WITH SPONTANEOUS PNEUMOTHORAX

UPPER AIRWAY OBSTRUCTION DUE TO KAPOSI'S SARCOMA IN AIDS

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Am Fam Phys 1987;36:133-140

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- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
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- Stevens-Johnson syndrome, toxic epidermal necrolysis, and anaphylaxis have been reported rarely. Anaphylaxis may be more common in patients with a history of penicillin allergy.
- Gastrointestinal (mostly diarrhea): 2.5%
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1% and, rarely, thrombocytopenia and reversible interstitial nephritis.

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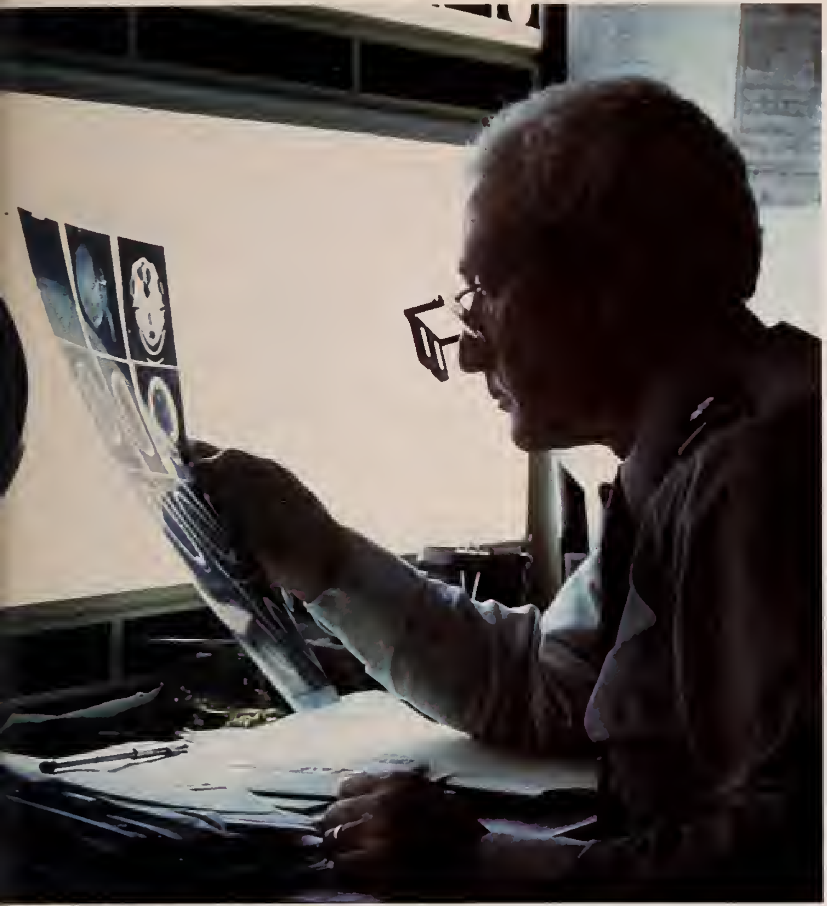

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ARIZONA

Jan 19–21. **Eighth Annual Mohs Micrographic Surgery Conference of The Skin Cancer Foundation.** The Westin LaPaloma, Tucson. Contact: Mitzi Moulds, Executive Director, The Skin Cancer Foundation, 245 Fifth Ave, Suite 2402, New York, NY 10016. Tel: (212) 725-5176.

CALIFORNIA

Jan 12–16. **Second International Symposium on Insulin-like Growth Factors/Somatomedins.** University of California, San Francisco. Contact: Extended Programs in Medical Education, School of Medicine, Room 124-C, University of California, San Francisco, CA 94143. Tel: (415) 476-4251.

Jan 19–21. **Advanced Seminars in Diagnostic Imaging.** 20 Cat I Credits. Ritz-Carlton Resort Hotel, Laguna Niguel. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

COLORADO

Jan 5–10. **Rheumatology for the Practicing Physician.** Keystone Resort. Contact: Cathy Caruso, Office of Continuing Medical Education, Washington University School of Medicine, 660 South Euclid, Box 8063, St Louis, MO 63110. Tel: (800) 325-9862.

Jan 7–9. **How to Write a Winning Workers' Compensation Report.** Vail. Contact: Workers' Comp Advisor, 11772 Sorrento Valley Rd, Suite 160, San Diego, CA 92121. Tel: (619) 792-7809.

FLORIDA

Jan 9–13. **Magnetic Resonance Imaging: Future and Present.** 20 Cat I Credits. Ritz-Carlton Hotel, Naples. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

Jan 12. **Prenatal Care, A Child's Right.** West Palm Beach. Contact: Susan McKnight, Vice President, League of Women Voters, PO Box 2564, West Palm Beach, FL 33402.

Jan 17. **Annual Refresher Course in Practical Pain Management.** *Jan 18–19.* **Seventh Annual Conference: Current Concepts in Pain Medicine.** Clarion Castle Hotel & Resort, Miami Beach. Contact: Peter R. Wilson, MD, Program Director, Mayo Clinic, Rochester, MN 55905. Tel: (507) 284-8312.

Jan 21–23. **14th Annual Gold Coast Pediatric Conference.** 12 Cat I Credits. Good Samaritan Hospital, West Palm Beach. Contact: Laura J. Lehmann,

(*continued on p 12A*)

New York State Journal of Medicine

DECEMBER 1990 / Volume 90, Number 12

MEDICAL SOCIETY OF THE STATE OF NEW YORK ORGANIZED 1807

Contents

COMMENTARIES

585 ***Mycobacterium tuberculosis:* A disease of growing importance in urban population groups**
MATTHEW R. WEIR, MD

586 ***Fusarium* and the immunocompromised host: Liaisons dangereuses**
ELIAS J. ANAISSIE, MD, MICHAEL G. RINALDI, PHD

RESEARCH PAPERS

588 **Pott's disease in urban populations: A report of five cases and a review of the literature**
PANAYIOTIS A. ELLINAS, FRED ROSNER, MD

591 **Prevalence and characteristics of violent patients in a general hospital**
SHELDON TRAVIN, MD; HYUNG KON LEE, MD; HARVEY BLUESTONE, MD

REVIEW ARTICLE

596 **Multiple addictions: Co-synchronous use of alcohol and drugs**
NORMAN S. MILLER, MD; BETH M. BELKIN, MD; MARK S. GOLD, MD

ESSAYS IN PEDIATRICS

601 **Central nervous system malignancies in children**
J. RUSSELL GEYER, MD, MITCHEL BERGER, MD

CASE REPORTS

609 **Disseminated infection caused by *Fusarium solani* in a patient with aplastic anemia**
ARIF S. EL-ANI, PHD

611 **Carcinoma of the lung presenting with spontaneous pneumothorax**
INDERJIT SINGH, MD; RAMESHBHAI P. PATEL, MD; VILAS DESHPANDE, MD; KEDAMBADY P. SHEKA, MD

612 **A large pericardial effusion in a patient with human immunodeficiency virus type 1**
TAK KWAN, MD; ARNOLD EINHORN, MD; ROBERT NACHT, MD

(continued on page 6A)

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New York State Journal of Medicine

CONTENTS, CONT'D

613 **Upper airway obstruction due to Kaposi's sarcoma in the acquired immunodeficiency syndrome**
VIC M. MALABONGA, MD, PETER R. SMITH, MD

LETTERS TO THE EDITOR

615 **New *Journal* cover**
JAMES T. EVANS, MD
PETER ROGATZ, MD

615 **Premenstrual syndrome**
MARGARET L. MOLINE, PHD, SALLY K. SEVERINO, MD

616 **Combining parenthood and practice**
ANNE ELAYNE BERNSTEIN, MD
JOHN P. SHEEHY, MD
ADELAIDE T. SCANLON, MD

618 **Fibrous disease of the breast in juvenile diabetes**
WENDE W. LOGAN-YOUNG, MD, NANCY YANES HOFFMAN, MSC

619 LEADS FROM EPIDEMIOLOGY NOTES

622 BOOK REVIEWS

627 BOOKS RECEIVED

629 NEWS BRIEFS

631 OBITUARIES

633 INDEX TO VOLUME 90

3A MEDICAL MEETINGS AND LECTURES

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1. Brantly ML, Paul LD, Miller BH, et al: Clinical features and history of the destructive lung disease associated with alpha-1-antitrypsin deficiency of adults with pulmonary symptoms. *Am Rev Respir Dis* 1988;138:327-336.
2. Wewers MD, Casolaro MA, Sellers SE, et al: Replacement therapy for alpha$_1$-antitrypsin deficiency associated with emphysema. *N Engl J Med* 1987;316:1055-1062.

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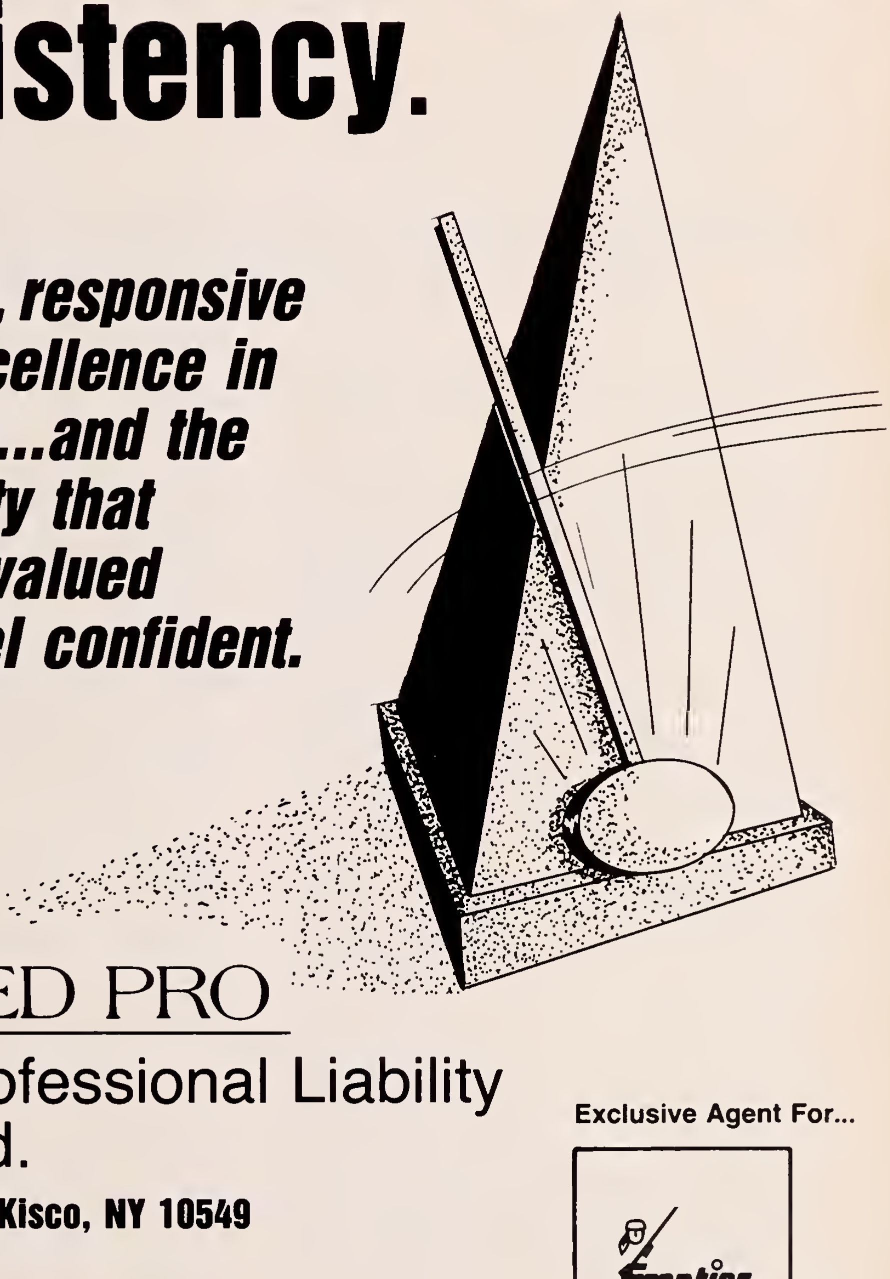

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New York State
Journal of Medicine

COMMENTARIES

Mycobacterium tuberculosis: A disease of growing importance in urban population groups

Over a year ago, I wrote an editorial[1] in the *Journal* about the enigma of extrapulmonary *Mycobacterium tuberculosis* (MTB). My editorial accompanied two reports describing patients with abdominal MTB[2] and mammary MTB,[3] respectively. In this issue of the *Journal* there is a report summarizing the observations of a municipal hospital in the New York City area of MTB infection of the spine.[4]

Prior to the onset of the human immunodeficiency virus (HIV) epidemic, the most important epidemiologic factors assisting physicians in identifying patients at risk for MTB infection included foreign birth, underlying debilitating diseases, and a history of exposure to patients with active MTB.[5] Today, with a growing percentage of HIV infected patients needing medical care, it is important to realize that these patients as well as patients engaged in high risk activity for HIV infection (such as promiscuity and intravenous (IV) drug abuse) are at increased risk for MTB infection. The recent increase in the percentage of MTB infected patients in urban areas may be explained by increased numbers of the gay population, IV drug abusers, and immigrants living in larger cities. Not surprisingly, there are increasing reports of extrapulmonary MTB disease, particularly in urban hospitals.

In a paper in this issue of the *Journal*, Ellinas and Rosner[4] present their observations of five patients with skeletal MTB, all of whom had important epidemiologic risks for MTB: Three had HIV disease and a history of IV drug abuse, the other two patients' risk factors were chronic ethanolism and recent immigration from an endemic region for TB disease, respectively. So their observations correspond with current known epidemiologic risk factors for MTB disease.[4]

Prior descriptions of extrapulmonary organ system involvement with MTB demonstrated that infections of the genitourinary system, lymphatic system, and respiratory system, other than the lung, made up the majority of cases.[5] However, these earlier observations may change with the increased prevalence of the newest epidemiologic risk factor for MTB, HIV infection, which eventually leads to overwhelming immune system dysfunction. Although skeletal MTB is an uncommon form of extrapulmonary disease, it may become a more prominent observation in patients with HIV disease. Although skeletal MTB is more commonly seen in older patients, this may change as younger patients become infected with HIV. The major problem with skeletal MTB is that delays in diagnosis are frequent, and consequently the disease is usually far advanced at the time of diagnosis. Bone involvement is usually by hematogenous spread. However, it may also occur through lymphatic or local extension. Skeletal involvement of the spine is not uncommon because the vertebral bodies and weight bearing joints are most commonly involved. This may be related to the proclivity of MTB bone infections to occur in areas of prior trauma.

Clinically, patients with extrapulmonary MTB may or may not have constitutional symptoms depending upon how early their disease is diagnosed. In the report by Ellinas and Rosner,[4] all of the patients complained of weight loss and weakness, and all had either back pain, leg weakness, or some other form of neurologic dysfunction. The diagnosis in these patients was made by radiographs of the affected area or by biopsy. Chest radiographs and tuberculin testing may not always be helpful in patients with extrapulmonary MTB and this is evident in the Ellinas and Rosner report, where only two of the five patients had a positive PPD, and three of the patients had no chest X-ray abnormalities.[4] Consequently, a high index of suspicion is essential in making the diagnosis. The risk of developing progressive neurologic damage is quite high with vertebral body involvement, especially if the patients are already complaining of neurologic dysfunction.

Thus, the observations of Ellinas and Rosner[4] serve as an important reminder to clinicians practicing in an urban hospital setting where a sizable proportion of patients have a greater epidemiologic risk for tuberculous infection, that any mass lesion or chronic infectious disease process associated with constitutional symptoms or focal neurological findings might be a tuberculous infection. As explained by Ellinas and Rosner,[4] if these symptoms are associated with leg weakness, one should be suspicious that there could be a tuberculous abscess of the spine leading to nerve compression. Because drug resistant MTB is also more common in immigrants and population groups of lower socioeconomic status as well as patients with extrapulmonary MTB, usually two or more anti-tuberculous drugs should be used to treat these patients.[5] Thus, the diagnosis and treatment of extrapulmonary MTB, particularly in urban population groups, will continue to be a problem. The earlier therapy can be started, the greater the likelihood that a satisfactory outcome can be achieved. One future concern about MTB disease in urban populations is that the surge in prevalence of HIV infection may lead to an expansion in the reservoir of latent MTB infection in both non-HIV infected as well as HIV infected men and women.

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Fusarium and the immunocompromised host: Liaisons dangeureuses

Invasive fungal infections present a formidable problem in immunocompromised patients. While most infections are still due to *Candida* species and *Aspergillus* species, newer fungi have now emerged as significant pathogens and are associated with a high mortality.[1] In this issue of the *Journal* El-Ani[2] reports on yet another emerging fungal species, *Fusarium solani*, as a cause of fatal fungal infection.

Infection with *Fusarium* species tends to occur in patients with hematologic disorders, particularly those undergoing intensive chemotherapy and/or bone marrow transplantation. In this patient population, fusarial infection is associated with a poor outcome, with death occurring in nearly all reported cases.[1,3-6]

Fusarium species are known plant pathogens and common soil fungi. During World War II, one million people were poisoned by eating grain infected with *Fusarium* spp. The ensuing illness, alimentary toxic aleukia, is the result of a mycotoxin and presents with gastrointestinal symptoms and weakness and may progress to aplastic anemia and death.[7] Infection with fusaria, as in the case reported in this issue by El-Ani,[2] is not known to be associated with toxin production.

Very little is known about the pathogenesis and management of invasive fusarial hyalohyphomycosis. Two modes of acquisition of the infection seem likely: respiratory, following inhalation of aerosolized conidia as evidenced by the frequent sino-pulmonary involvement in these patients;[3,4] and cutaneous, as in burn patients, in cases of catheter related infections, or following finger or toe cellulitis.[3,8,9] The prevalence of *Fusarium* species in grains and the involvement of the gastrointestinal tract in some cases raise the possibility of a gastrointestinal route of infection.[3]

Based on clinical observations of several patients cared for at our institution and at others, it appears that neutropenia and adrenalcorticosteroid therapy are the two major risk factors for the development of systemic fusarial hyalohyphomycosis.[1-6] In a murine model of systemic infection developed in our laboratory, neutropenic mice and mice treated with corticosteroids were more susceptible to infection with *F solani* than normal untreated mice.[10] The occurrence of fusarial hyalohyphomycosis in a patient with chronic granulomatous disease also highlights the importance of neutrophils in fighting these fungi.[11] The interaction of cellular and humoral immunity with the various morphogenetic forms of the organism is being currently investigated in the laboratory of one of the authors. In addition, the protective role of intact skin is evidenced by the occurrence of serious fusarial infections in patients with extensive burns.[9]

The clinical spectrum of infection in the immunocompromised host has been previously described and consists of limited soft tissue infection, catheter-related fungemia without organ involvement, and disseminated multiorgan disease.[3] The clinical characteristics of patients with disseminated fusarial hyalohyphomycoses are similar in many respects to those with disseminated aspergillosis. The two mycoses share in common the occurrence of nodular erythematous skin lesions with central necrosis, the frequent sino-pulmonary involvement, and the propensity for vascular invasion and thrombosis resulting in tissue infarction.[3-5] However, we made the observation that fusarial infections are associated with a significantly higher incidence of skin and subcutaneous lesions and a much higher yield of the organisms in blood culture specimens than infections with *Aspergillus* species.[3] These observations were subsequently confirmed by others.[4,5] Furthermore, we have now developed a rabbit model of systemic fusarial hyalohyphomycosis in which *F solani* can be regularly isolated from the bloodstream.[12] In a similar animal model of systemic aspergillosis, blood cultures consistently fail to yield the organism (T. Patterson, personal communication, May 1990). The animal model of fusarial hyalohyphomycosis may be of great help in understanding the pathogenesis of angioinvasion with *Fusarium* spp. As with aspergillosis, infection caused by *Fusarium* species is characterized by vascular invasion and infarction of surrounding tissues. The organism appears in tissue as acute branching, septate hyphae. Because of these similarities in the clinical and pathologic features between aspergilli and fusaria, fungal cultures are essential for establishing the diagnosis. Recently, immunohistologic staining has been used successfully for tissue diagnosis and represents a most encouraging trend in diagnostic mycology.[13]

The outcome of disseminated fusarial hyalohyphomycosis has been almost universally fatal. Even when treated with large doses of amphotericin B, most patients have ultimately died from their infection.[3-6] Additionally, prophylaxis with amphotericin B has failed to prevent the infection.[14] On the other hand, induction of remission of the underlying disease, and reversal of chemotherapy-induced marrow suppression have been associated with a positive clinical response.[4] In our model of murine systemic fusarial infection, amphotericin B at a dose of 1 mg/kg per day was unable to prolong survival even when treatment was commenced as early as one hour after inoculation of a clinical isolate of *F solani*. In contrast, newer triazole antifungals SCH 39304 and fluconazole appeared to prolong survival of infected mice. Additional studies with a larger number of strains are needed to confirm these encouraging preliminary findings with the newer triazoles. A clinical trial of SCH 39304 in *Fusarium* infections is currently underway to clarify the role of this agent in patients with this infection.

In summary, *Fusarium* spp have emerged as significant pathogens in immunosuppressed patients. These infections are difficult to manage and frequently fatal. The activity of amphotericin B in human and experimental fusarial hyalohyphomycosis is inconsistent. It is hoped that the advent of newer antifungal agents and biologic response modifiers may result in a significant improvement in the prevention and treatment of these life threatening opportunistic infections.

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Pott's disease in urban populations

A report of five cases and a review of the literature

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ABSTRACT. **Five patients were treated at the Queens Hospital Center between 1987 and 1989 for tuberculosis of the spine. All were members of low socioeconomic status minority groups: four were black and one was Asian. Three were intravenous drug abusers and were positive for human immunodeficiency virus (HIV), one was homeless and suffering from alcoholism, and one had just arrived in the United States from a region in Asia endemic for tuberculosis. All three HIV positive patients had negative tuberculin skin test results, had no radiographic signs of pulmonary involvement, and were anemic on presentation; one of them had a bone marrow biopsy that was positive for acid-fast bacilli. Since there may be a prolonged delay without significant radiographic findings, a high index of suspicion is necessary to make the diagnosis of tuberculosis of the spine.**

(NY State J Med 1990; 90:588–591)

Tuberculosis due to *Mycobacterium tuberculosis* has plagued mankind since ancient times and continues to be a major health problem in most Third World countries. Mycobacteria have been detected in Egyptian mummies.[1] Tuberculosis of the spine was first described in 1779 by Percival Pott.[2] Skeletal involvement is found in only 1% of patients with tuberculosis, of whom about 75% have spinal tuberculosis, with neural involvement occurring in 10–47% of these patients.[3] In the United States, paraplegia may be the first sign of Pott's disease in 7–10% of patients.[4] Approximately 50% of the patients with skeletal tuberculosis have no demonstrable concomitant pulmonary tuberculosis.[3]

The proportion of tuberculosis occurring among nonwhites in the United States increased from 23% in 1953 to 48% in 1985.[5] There is an excess morbidity from tuberculosis in minority populations.[5] Forty-eight percent of all cases of tuberculosis in the United States in 1985 occurred in minorities (blacks, Asians/Pacific Islanders, and American Indians/Alaskan Natives). The 25–44-year age group is particularly involved. Many among minority populations in the United States today are immigrants or refugees from areas with a high prevalence of tuberculosis.[5]

The incidence of tuberculosis in large urban populations such as New York City is increasing in association with a growing homeless population often affected by drug-resistant tubercle bacilli, heroin addiction, alcoholism, and cellular immunosuppression caused by human immunodeficiency virus.[5–7] Tuberculosis alone was the cause of death in 2.1% of addicts in New York City.[8] Tuberculosis and its extrapulmonary manifestations are no longer on the decline in the United States.[9] This report describes five patients with Pott's disease treated at a New York City municipal hospital over a two-year period.

PATIENTS AND METHODS

The records of patients with a diagnosis of tuberculosis of the spine (Pott's disease) treated at the Queens Hospital Center between 1987 and 1989 were reviewed. The following information was obtained from each medical record: age, sex, race, primary diagnosis, risk factors for tuberculosis infection (intravenous drug abuse, human immunodeficiency virus infection, alcoholism, homelessness, and travel to a region endemic for tuberculosis), hospital course, presenting symptoms, chest radiograph, spinal radiograph, and computerized axial tomography (CAT) scan findings, purified protein derivative (PPD) skin test status, histopathology of biopsied tissue, tissue culture and smear results, cerebrospinal fluid (CSF) culture results, and myelogram reports.

Pott's disease was diagnosed on the basis of the patients' clinical presentation and symptomatology, associated risk factors, radiographic and CAT scan findings, magnetic resonance imaging (MRI), and myelogram investigations, and based on the tuberculin skin test status, cultures for acid-fast bacilli (with identification by biochemical characteristics and, where necessary, by genetic probes), and histologic tissue investigations where relevant. A definite diagnosis of Pott's disease was made in the presence of a destructive spine lesion, paravertebral mass, and positive culture evidence of *Mycobacterium tuberculosis*, and improvement following anti-tuberculosis therapy (Cases 2 and 5). A presumptive diagnosis was made in the absence of a positive culture (Case 4) or the absence of a destructive spine lesion (Case 3). One patient (Case 1) was classified as possible Pott's disease because of back pain, positive PPD, and improvement of back pain following anti-tuberculosis therapy.

RESULTS

The patients ranged in age from 35 to 76 years; two were women and three were men. Four of the patients were black and the fifth was Asian. The primary diagnoses, risk factors, and hospital courses are listed in Table I. One of the patients died while in the hospital, and the remainder were discharged and maintained on anti-tuberculous treatment. Three black patients were intravenous (IV) drug abusers and HIV positive, one black patient was homeless and suffering from alcoholism, while the fifth patient was an elderly Tai-

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TABLE I. Clinical Data in Five Patients with Pott's Disease

Patient #	Age (yrs)	Sex	Race*	Primary Diagnosis	Risk Factors	Hospital Course[†]	Outcome
1	38	M	B	AIDS and possible vertebral tuberculosis	IVDA, HIV+, alcoholism[‡]	Improved	Discharged
2	76	F	A	Definite vertebral tuberculosis and urinary tract infection	Immigrant from TB-endemic region	Improved	Discharged
3	35	F	B	AIDS, presumptive vertebral tuberculosis, and bilateral pneumonia	IVDA, HIV+	Deteriorated	Expired
4	46	M	B	Pulmonary and presumptive vertebral tuberculosis	Alcoholism, homeless	Improved	Discharged
5	37	M	B	AIDS and definite vertebral tuberculosis	IVDA, HIV+	Improved	Discharged

* B, black; A, Asian.

[†] After initiation of anti-tuberculosis medication (isoniazid, rifampin, ethambutol).

[‡] IVDA, intravenous drug abuser; HIV+, human immunodeficiency virus positive.

wanese woman who had just emigrated to the United States, after working for years as a health worker in a region of Taiwan endemic for tuberculosis.

The most common symptoms, present in all five patients prior to presentation, were weight loss and lower extremity weakness. Back pain, fever, and night sweats were present in four of the five patients. Patients 2 and 4 presented with associated urinary tract infections, patient 1 had penile herpes, patient 3 had genital syphilis, and patient 5 had urinary incontinence and *herpes zoster* infection. All three patients with acquired immunodeficiency syndrome (AIDS) had associated anemia, oral and esophageal thrush, cough, diarrhea, and head CAT scans that indicated changes consistent with AIDS encephalopathy. Chest and spine radiographs and CAT scan findings for all patients are listed in Table II.

Neurologic manifestations were present in patients 3, 4, and 5. Patient 3 had a Brown-Sequard-like syndrome, patient 4 had a neurogenic bladder, and patient 5 had urinary incontinence. All three AIDS patients had negative tuberculin skin tests, while the other two patients had positive reactions. Tissue biopsy was positive for caseation necrosis and granulomatous inflammation in two of three patients biopsied; the same two patients also had positive cultures for *Mycobacterium tuberculosis.* Cultures of cerebrospinal fluid were done in four patients and were all negative. Myelograms in two of three patients yielded positive findings (see Table III). The sedimentation rate was available only for patient 5 and was elevated. The duration of symptoms varied from one to five months.

The lower thoracic and lumbar vertebrae are most commonly involved by spinal tuberculosis in adults, while sacral involvement is relatively rare. In this study, the destructive focus was clearly identifiable in patients 2 and 5 (L-1,2 and T-7,8 respectively), while on radiographs, "serpiginous calcifications" were visible overlying the lower sacrum of patient 4. A CAT scan on this patient showed paravertebral widening of soft tissues. During a corrective surgical procedure, anterior spinal fusion is achieved by the debridement of the vertebrae above and below the lesion and the insertion of autologous bone graft between these vertebrae to promote bony fusion. Both patients 2 and 5 underwent successful anterior surgical debridement through a left posterolateral thoracotomy.

Four of the five patients improved after two to six months of antituberculous therapy, with significant reduction in back pain and an increase in strength and general feeling of well-being.

DISCUSSION

In a patient with a destructive lesion of the spine, often associated with a paraspinal mass, one must consider neoplasms, myeloproliferative disorders, and an infectious process. Metastatic neoplasm is the most common cause of a destructive vertebral lesion in the older population. In an urban population where there is a high incidence of intravenous drug abuse, as well as HIV infection, one might see tuberculous spondylitis more commonly than would otherwise be seen in immunocompetent individuals. In Third World countries, pulmonary tuberculosis remains a disease of the young, with the majority of cases being diagnosed by the age of ten years.[10] A very high index of suspicion is essential in making the diagnosis of tuberculosis of the spine. One study states that 50% of

TABLE II. Admission Findings of Five Patients with Pott's Disease

Patient #	Presenting Symptom	Chest Film Findings	Spinal Film Findings	CAT Scan Findings	MRI Findings
1	Back pain, weight loss, leg weakness, anemia, night sweats, cough, sputum, SOB,* oral thrush, diarrhea	N[†]	N	N	N
2	Back pain, weight loss, leg weakness, fever, night sweats	N	P[‡]	P[‡]	P[‡]
3	Back pain, weight loss, anemia, fever, night sweats, cough, sputum, oral thrush, pneumonia, diarrhea	Positive for pulmonary TB	N	N	ND[§]
4	Weight loss, leg weakness, SOB, neurogenic bladder	Positive for pulmonary TB	P[‖]	P[‡]	ND
5	Back pain, weight loss, leg weakness, anemia, fever, night sweats, cough, sputum, oral thrush, urinary incontinence, diarrhea	N	P[‡]	P[‡]	ND

* SOB, shortness of breath.

[†] N, negative; P, positive.

[‡] Destructive lesion and paravertebral mass.

[§] ND, not done.

[‖] Serpiginous calcification of uncertain significance.

TABLE III. Diagnostic Procedures in Five Patients with Pott's Disease

Procedure	Patient #1	Patient #2	Patient #3	Patient #4	Patient #5
PPD skin test status	N[*]	P	N	P	N
Histopathology: caseation and/or granulomatous inflammation	P[†]	P[‡]	ND[§]	ND	N[‡]
Culture for *Mycobacterium tuberculosis*	P[†]	P[‡]	P[†]	ND	N
Smear for mycobacteria	N	P	ND	ND	N
Culture of CSF[‖] for mycobacteria tuberculosis	N	N	N	ND	N
Myelogram	ND	P	N	ND	P

[*] N, negative; P, positive.
[†] Bone marrow biopsy.
[‡] Paravertebral abscess biopsy.
[§] ND, not done.
[‖] Cerebrospinal fluid.

patients with skeletal tuberculosis seen in a year were misdiagnosed, with an average delay in diagnosis of 19 months.[11]

Tuberculous spondylitis usually follows primary tuberculous infection either at the time of infection or after a short or long latency period. Skeletal infection also occurs as a distant manifestation of endocarditis or other primary septic process. In the present report, three IV drug abusing patients had all been previously treated for endocarditis, which was not proven to be due to tuberculosis. Extrapulmonary involvement, with or without radiographic evidence of lung damage, can be detected in approximately 15% of patients with tuberculosis. About 20% of patients with extrapulmonary localization have osteoarticular forms involving two or more vertebral bodies and intervertebral disks.[12] Hematogenous or lymphatic seeding of the bones occurs either at the time of primary infection or from the reactivation of a dormant extraosseous focus. The infectious focus is usually in the anterior portion of the vertebral body and thus spreads up and down the spine or anteriorly into the soft tissues.

In about 70% of patients with spinal tuberculosis a paravertebral abscess develops.[13] In this study, only patients 2 and 5 had paraspinal masses identifiable by radiographs and/or CAT scan. The onset of metastatic tuberculosis is gradual, and the patient usually presents with vague, nonspecific symptoms of chronic infection with low grade fevers, chills, night sweats, back pain, weight loss, and malaise. Back pain is the most common presenting symptom in patients with tuberculous spondylitis.[14] In this study, all five patients had significant weight loss and lower extremity weakness prior to presentation, and four of five had back pain, fevers, and night sweats.

The majority of patients have a positive, intermediate strength PPD skin test, but only about half have chest radiographs with signs of new or old pulmonary tuberculosis.[4,14] In this study, only the patients who were not HIV infected had a positive tuberculin skin test. In a study of the cellular immunity in hospitalized intravenous drug abusers without AIDS, Layon et al[15] found that all patients had a positive tuberculin skin test. All three HIV infected patients in this study were anergic to the tuberculin skin test. The prevalence of both pulmonary and disseminated forms of tuberculosis is higher in immunocompromised patients.[6,9,15] In HIV immunocompromised patients in Third World countries such as Haiti and most of Africa, where tuberculosis is very common, tuberculosis is a major opportunistic infection. By contrast, HIV infected patients in the United States have *Mycobacterium avium-intracellulare* (MAI) as the most common mycobacterial infection.[16] Tuberculosis may precede the opportunistic infections characteristic of AIDS simply because *M tuberculosis* is a more virulent organism and, therefore, causes disease at an earlier stage of immunosuppression among tuberculous infected patients who have AIDS. In contrast, disease secondary to atypical mycobacteria (which are less virulent organisms than *M tuberculosis*) occurs at a later stage of immunosuppression. In this study, patient 1 had bronchial washings positive for MAI, while a bone marrow biopsy was positive for mycobacterial tuberculosis bacilli.

One of the presenting signs in all three HIV-infected patients, but in neither of the other two patients, was anemia. The bone marrow biopsy that was positive for tubercle bacilli in patient 1 suggests that granulomas in the marrow may be one of the causes of anemia. In addition, IV drug addiction independent of HIV infection may cause cellular immunosuppression.[15,17] In vitro, opiates impair T-lymphocyte mitogen responsiveness, inhibit migration of polymorphonuclear leukocytes (PMNs) in laboratory animals, and reduce the phagocytic capacity of human PMNs.[18] These findings can be reversed by opiate antagonists such as naloxone or by discontinuation of opiate administration.[17]

The radiographic diagnosis of spinal tuberculosis is often delayed, because the radiographic picture remains unchanged for weeks and the only signs and symptoms may be paraplegia and neural involvement. The radiographic signs of osteomyelitis may not appear for a long time by virtue of the kinetics of bone destruction and repair. Patient 1 underwent six separate radiographic investigations over a three-week period during his hospital stay, all of which were unremarkable and showed no evidence of a pathologic process involving the spine.

With the advent of CAT scanning and MRI, spinal tuberculosis can be detected at a much earlier stage than was previously possible with spinal radiographs. CAT scanning gives a better assessment of the pathologic process and the extent of bony destruction than do spine films.[19] CAT scanning is also very helpful in identifying edema and soft tissue involvement,[4] and for the three-dimensional localization of the abscess for percutaneous needle biopsy and aspiration of culture specimens. Calcifications within a tuberculous abscess are well visualized with CAT scanning but not with MRI. A calcified paraspinal mass was clearly visualized by CAT scanning in patients 2 and 4. In a recent study, fine needle aspiration was shown to be effective in making the diagnosis of Pott's disease and was confirmed by culture of the aspirated material.[20] This technique may make the diagnosis of Pott's disease more rapid and safer by avoiding more invasive procedures such as core biopsies or open biopsies under general anesthesia.[20] An inflammatory mass was evident in patients 2 and 5. In both patients surgical anterior debridement of the diseased vertebrae and biopsy of the inflammatory mass were performed. Cytologic examination of the biopsied mass in patient 2 showed the presence of caseation necrosis, granulomatous inflammation, and the presence of tubercle bacilli, while in patient 5 changes consistent with a chronic inflammatory process was the only finding. A bone marrow biopsy in patient 1 was positive for tubercle bacilli.

In summary, the growing problem of extrapulmonary tuberculosis in urban population groups requires physicians to be aware of the potential for skeletal tuberculosis in any patient who has protean symptomatology of the spine. This high index of suspicion is especially needed if the patient is of low socioeconomic status, has a foreign birthplace, and has associated chronic alcohol or intravenous drug abuse. Individuals who are HIV positive can have *Mycobacterium tuberculosis* as the cause of destructive lesions of the spine despite the fact that these patients more commonly are infected with *Mycobacterium avium intracellulare*. Since there is a prolonged delay without significant radiographic or clinical findings until advanced disease is evident, sophisticated radiographic testing together with a healthy index of suspicion are necessary in order to make the appropriate diagnosis.

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Prevalence and characteristics of violent patients in a general hospital

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ABSTRACT. A survey was conducted in an inner city general hospital to determine the prevalence and characteristics of violent patients. The patients were evaluated by psychiatrists in the psychiatric emergency and psychiatric consultation services during a six-month period, and in the acquired immunodeficiency syndrome (AIDS) consultation service during a one-year period. Of 1,506 patients seen in the psychiatric emergency service, 103 (6.8%) exhibited violent behavior. The majority of these patients were diagnosed as having schizophrenic disorder or substance abuse disorder. Of 390 patients seen in the psychiatric consultation service, 17 (4.4%) were evaluated for violent behavior. The majority of these were diagnosed as having organic mental disorder or personality disorder. Of 256 patients seen by the AIDS consultation service, 11 (4.3%) were evaluated for violent behavior, of which seven patients were diagnosed as having organic mental disorder and four as having adjustment disorder. The authors conclude that violent behavior in their institution was not as prevalent as previously believed.

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Although there is an expanding literature on the problem of violent patients in specialized psychiatric facilities,[1] there is relatively limited information on the nature and extent of violent behavior occurring in a general hospital.[2] Ochitill[2] points out some of the difficulties faced by researchers in collecting such data: hospitals vary in their definitions and documentation of violent incidents, and indeed, individuals define violent behavior differently depending on their psychological and sociocultural backgrounds. Armstrong[3] describes several ways in which

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individuals' beliefs about violence may contribute to difficulties in documenting and even recognizing these problems in general hospitals. He notes, for example, that nurses generally report only the most serious cases of assault, while dismissing most cases of violence as regrettable but not rectifiable. Moreover, violence is not considered to be a medical entity worthy of diagnosis or systematic investigation; physicians usually brand assaultive behavior outside their own professional medical domains, and thus may not ensure that the mangement plans are carried out on their respective services.

Estimates of the rate of violent behavior occurring on the general medical/surgical wards have consistently been low. Bustamente and Ford[4] found that ward management problems were among the more common reasons that psychiatric consultations were requested in the general hospital. In fact, several studies[5-7] list management problems as the second most frequent reason for psychiatric consultations. But the description of these management problems includes a broad range of behavioral disturbances and uncooperative behavior without specifying violence. Hackett and Stern[8] assert that psychiatrists rarely encounter dangerously assaultive patients on general medical or surgical wards. In their two-year study of violent behavior among hospitalized medical and surgical patients, Ochitill and Krieger[9] found that 29 incidents of verbal and physical violence had occurred. Eleven incidents occurred in general medicine, 16 in general surgery, and two in the emergency area. Significantly, of the 28 patients who received a psychiatric diagnosis, 19 abused substances, usually alcohol or opiates. In most instances, altercations with the staff over pain medication or ward regulations preceded the violent incidents. Reid et al,[10] in their major survey of 566 psychiatric and 898 nonpsychiatric beds in 16 hospitals, found that the overall rates of assault were 2.54 assaults per bed per year in psychiatric units and 1.72 assaults per bed per year in nonpsychiatric units. Reid et al[10] concluded that there is a small assault rate in hospitals, mostly by patients against staff or other patients, that very few of the assaults resulted in serious injury, and that there was great variability among hospitals and between units.

In regard to the issue of determining total numbers and descriptions of patients seen for violent behavior in hospital emergency rooms, Skodol and Karasu[11] conducted a two-week study on patients referred for psychiatric evaluations in 1978. By specifically asking questions about violence and utilizing a rating instrument, Skodol and Karasu found that of a sample of 367 patients, 62 patients (17%) were considered to be violent because of "aggressive ideation or behavior in their clinical presentations." Forty-two (11%) of them had acted on these impulses prior to coming to the emergency room. The largest category of violent patients were schizophrenic (N=25), followed by those with various neuroses, character disorders, and situational disturbances (N=18). Skodol and Karasu pointed out that the majority of these violent patients were treatable, either by crisis intervention or short term hospital care. The need for more careful and consistent clinical attention to the circumstances and contexts of violence is underlined by the difference between the numbers cited in this study and those cited by Ochitill and Krieger,[9] who reported only two cases of violent acts actually occurring in the emergency area of a general hospital during the two-year period from 1976 to 1978. In contrast, Taylor et al[12] noted that half of all violent incidents in general hospitals occur in the emergency rooms.

The limited, as well as frequently inconsistent, reports on the prevalence of violence in general hospitals underscore the need for individual hospitals to conduct their own surveys. Based on this information, each hospital would then be able to decide how to allot its resources to meet specific needs. The purpose of this study is to present data obtained on the prevalence of violence in various sections of a 565-bed acute care general hospital in an inner city area.

METHODS

The authors reviewed all cases in which patients had been seen by psychiatrists because of violent behavior on the general medical and surgical wards and in the emergency room. It is the hospital's general policy that every patient who manifests or threatens violence be seen for psychiatric consultation. Also, the Code Blue team, which represents an on-site response to emergency violent situations, must include a psychiatrist. Therefore, patients identified as violent in the psychiatrists' log books on the consultation services and emergency room represent the prevalence rate of violent behavior on these services. The specialized psychiatric unit of the hospital was excluded from this study.

Although the main purpose of this study was to collect data on patients manifesting violent behavior against others, we also presented for comparison some data on the number of patients evaluated for suicidal behavior. Violent behavior was defined as one of three categories: violence to person (physically assaultive), violence to property, or verbally threatening violence to person. Simple agitation or excitement was not considered violent behavior. The data on the psychiatric emergency service were obtained by reviewing the charts of all patients evaluated for the six-month period from October 1, 1988, to March 31, 1989. It should be noted that this hospital does not have an independent psychiatric emergency room but receives direct referrals from the screening nurse, or consultation requests on medical patients with psychiatric problems from the emergency room physicians. Information on Code Blue patients for the six-month period from February 1, 1989, to July 31, 1989, was obtained from the security department of the hospital and then by reviewing the identified patients' charts. The data on violent patients seen in psychiatric consultation among the general medical/surgical inpatient population, including those identified as AIDS patients (January 1, 1989, to June 30, 1989, by the psychiatric consultation service; and January 1, 1988, to December 31, 1988, by the AIDS consultation service), were obtained from detailed discussions with the psychiatrists who had examined these patients as well as by reviewing the charts. The data included the type of violent behavior, demographic characteristics, psychiatric diagnoses, and substance abuse patterns of the violent patients. Psychiatric diagnoses were made according to the criteria established in the *Diagnostic and Statistical Manual of Mental Disorders* (3rd edition-revised) (DSM-III-R).[13]

RESULTS

Of a total of 1,506 patients evaluated in the psychiatric emergency service for a six-month period, the presenting and documented complaints of 45 patients included violence to others. Twelve patients were violent to property, and 46 patients were verbally threatening to persons. Therefore, the total number of patients evaluated for violent behavior was 103 (6.8%) (Table I). During this same period, 209 (13.9%) patients were evaluated for suicidal behavior. Of the patients exhibiting violent behavior, 67 (65%) were males and 36 (35%) were female, while the age group most represented was between 26 and 35 years (Table II). The largest diagnostic category

TABLE I. Total Number of Dangerous Patients Evaluated

	Psychiatric Emergency Service	Psychiatric Consultation Service	AIDS Consultation Service	Total
Period of time	6 mos	6 mos	1 year	
Total number of patients evaluated	1,506 (100%)	390 (100%)	256 (100%)	2,152 (100%)
Total number of patients evaluated for violence	103 (6.8%)	17 (4.4%)	11 (4.3%)	131 (6.1%)
Total number of patients evaluated for suicidal behavior	209 (13.9%)	123 (31.5%)	15 (5.9%)	347 (16.1%)

TABLE II. Age and Sex Distribution of Patients Evaluated for Violent Behavior

	Psychiatric Emergency Service		Psychiatric Consultation Service		AIDS Consultation Service	
Age (yr)	Male	Female	Male	Female	Male	Female
Under 18	1 (0.9%)	1 (0.9%)	—	—	—	—
18–25	18 (17.5%)	7 (6.8%)	1 (5.9%)	—	—	—
26–35	26 (25.2%)	13 (12.6%)	2 (11.8%)	1 (5.9%)	6 (54.5%)	2 (18.2%)
36–45	12 (11.7%)	4 (3.9%)	—	2 (11.8%)	3 (27.3%)	—
46–55	5 (4.9%)	7 (6.8%)	3 (17.6%)	2 (11.8%)	—	—
56–65	2 (1.9%)	3 (2.9%)	—	1 (5.9%)	—	—
Over 65	3 (2.9%)	1 (0.9%)	2 (11.8%)	3 (11.6%)	—	—
Total	67 (65.0%)	36 (35.0%)	8 (47.0%)	9 (53.0%)	9 (81.8%)	2 (18.2%)

TABLE III. Diagnoses of Patients Evaluated for Violent Behavior

Diagnosis	Psychiatric Emergency Service	Psychiatric Consultation Service	AIDS Consultation Service
Schizophrenic disorder	43 (41.7%)	3 (17.6%)	—
Substance abuse disorder	30 (29.1%)	1 (5.9%)	—
Bipolar disorder	6 (5.8%)	—	—
Adjustment disorder	16 (15.5%)	—	4 (36.4%)
Organic mental disorder	3 (2.9%)	8 (47.1%)	7 (63.6%)
Personality disorder	2 (1.9%)	4 (23.5%)	—
Other disorders	3 (2.9%)	1 (5.9%)	—
Total	103 (100%)	17 (100%)	11 (100%)

of these patients was schizophrenic disorder (N=43, 41.7%), followed by substance abuse disorder (N=30, 29.1%), and adjustment disorder (N=16, 15.5%) (Table III). Significantly, 68 (66%) of the total of 103 violent patients were reported to have a history of abuse of alcohol or other substances. The breakdown of reported substances abused is as follows: alcohol, 30 (44.1%); alcohol and cocaine, 11 (16.2%); cocaine, 8 (11.8%), marijuana, 5 (7.4%); others (phencyclidine, opioids, lysergic acid diethylamide), 9 (14.2%); and polysubstances, 5 (7.4%). Of these 103 patients, 61 (59.2%) were admitted to the inpatient unit, and 42 (40.8%) were released from the emergency room. In comparison, of the 209 patients evaluated for suicidal behavior, 99 (47.4%) were admitted to the inpatient unit, and 110 (52.6%) were released from the emergency room.

The results of the study in the psychiatric consultation service were as follows: of a total of 390 patients on the general medical/surgical wards referred for psychiatric consultation for the six-month period, nine patients were evaluated for physical violence against other persons, and eight for verbally threatening violence. Thus, a total of 17 patients (4.4%) were evaluated for violent behavior, in contrast to a total of 123 patients (31.5%) evaluated for suicidal behavior (Table I). The violent patients included eight males and nine females. Almost half of these patients (N=8) were diagnosed as having organic mental disorder, including delirium tremens (1), alcohol dementia (2), organic personality disorder (2), delirious state (2), and senile dementia (1). The next largest diagnostic group comprised various personality disorders (N=4), two of which were severe borderline personality disorders. Of the remainder, three patients were schizophrenic, one a substance abuser, and one a post-traumatic stress disorder patient.

Of these violent patients, only three could not be managed on the medical service and were transferred to the psychiatric unit. All of the violent patients (N=17) were immediately put on constant observation, and all but two were given tranquilizing medications. One patient died of advanced liver disease.

Of a total of 256 patients diagnosed as suffering from AIDS who were admitted to the general medical service and referred for psychiatric consultation during a one-year period, seven were evaluated for violence to persons and four for verbally threatening violence. Thus a total of 11 (4.3%) were evaluated for violent behavior, while a total of 15 (5.8%) were evaluated for suicidal behavior (Table I). The 11 patients evaluated for violence included nine males and two females, all between the ages of 26 and 45 years. Seven were diagnosed as having organic mental disorder (delirium or dementia) and four as having adjustment disorder. Nine of these violent patients had a history of opioid abuse, and two of opioid and other substance abuse. All these patients were initially placed on constant observation, two of them until their deaths. Eight were also medicated, and three were transferred to the psychiatric unit. Similarly, all 15 patients evaluated for suicidal behavior were put on constant observation, but only one was transferred to the psychiatric unit.

Interestingly, patients seen by the psychiatric emergency service were younger (age $\leq 45{:}>45=82{:}21$) than the patients seen by the psychiatric consultation service (age ≤ 45:age$>45=6{:}11$) ($\chi^2=14.67$, df=1, $p<0.001$). Also, the distribution of diagnostic categories shows interesting differences between patients seen by the psychiatric emergency service, psychiatric consultation service, and psychiatric AIDS consultation service (Table III). A majority of patients seen by the psychiatric emergency service had primary diagnoses of schizophrenic disorder or substance abuse disorder, while a majority of patients seen by the psychiatric consultation service had an organic mental disorder or personality disorder, and all patients seen by the AIDS consultation service were diagnosed as having either organic mental disorder or adjustment disorder.

In regard to Code Blue incidents, a total of six such incidents occurred throughout the hospital during the six-month period. There were two violent incidents in the intensive care unit, two in the adolescent medical unit, one on the medical ward, and one in the emergency room.

The case in the emergency room is illustrative of this type of incident. It involved a 32-year-old alcoholic man who, seemingly frustrated at having to wait to see the psychiatrist, started spitting on the floor. When told by the security guard to stop, the patient started punching the guard. A Code Blue was called; the patient was restrained, medicated, and referred to the psychiatric unit.

Discussion

Despite the general public's perception of violent behavior increasing in inner city hospitals because of the high prevalence of substance abuse, particularly of "crack" cocaine, this study confirms earlier reports about the relatively low rate of violence occurring in general hospitals.[8-10] In the psychiatric emergency service, during a six-month period, there were only 103 (6.8%) patients seen who required psychiatric intervention because of complaints of violent behavior. These findings differ from those of Skodol and Karasu's[11] study, which found that 11% of 367 patients acted out their violent urges against others prior to being seen in the emergency room of a city hospital in the Bronx in 1978. We believe that one difference between the results of these two studies reflects a difference in methodology: the Skodol and Karasu study was a prospective one in which clinicians were instructed to use a rating instrument and ask specific questions pertaining to the presence of violent ideation or behavior. On the other hand, our clinicians do not systematically use a rating instrument but are instructed to inquire specifically about dangerous behavior during the course of the psychiatric evaluation. The reason for these explicit instructions is that we agree with Rada's[14] assertion that just as some years ago clinicians were hesitant to inquire about suicide potential, therapists today commonly use denial and tend to avoid direct questioning about violent urges.

An additional explanation for the relatively low rate of violence seen by the psychiatric emergency service has to do with the nature of patients served. Many psychiatric patients who frequent our emergency room are not bona fide emergency cases but are individuals residing in the community who utilize it as a walk-in clinic, especially during off-hours such as evenings and weekends. These patients come to speak to a psychiatrist because they missed their daytime appointment in the outpatient clinic or to request a refill of their medication. Also, patients evaluated for dangerousness and deemed to be in need of emergency hospital care by psychiatrists in the other outpatient sections of this hospital, such as the adult, alcohol, or geriatric services, are often referred directly to the psychiatric unit without being screened by the psychiatric emergency service, and therefore are not reflected in the emergency statistics.

In regard to the number of patients exhibiting violent behavior seen by the psychiatric consultation service on the general medical/surgical wards, the total of 17 (4.4%) out of the 390 patients referred for evaluation is low, and consistent with the previous reports in the literature.[9] It should be pointed out, however, that of the total of 8,456 adults admitted to the hospital, excluding psychiatry and pediatrics during that period, only 4.6% were referred for psychiatric consultation. This figure is also consistent with literature about the underutilization of psychiatric consultation in general hospitals.[4] These 17 violent patients represented only 0.20% of the total number of 8,456 patients admitted to the medical/surgical services during this six-month period. It is also possible that this low prevalence rate is due to a narrow interpretation of violent behavior. Patients exhibiting behavioral or management problems, including agitation and delirium, who were seen by the psychiatric consultant, and who were treated and brought under control, were not included among the violent patients.

Another related issue is that all of the violent patients seen by the psychiatric consultation service were initially placed on constant observation, which means that a staff member is assigned on a one-to-one basis to keep the patient within visual range and within arm's reach at all times. Of the 17 patients seen by the psychiatric consultation service, three were then transferred to the psychiatric unit, and one died. Although there is a hospital procedure on the use of constant observation for patients who may be harmful to themselves, there are none to guide the clinician in cases of violence. Goldberg[15] pointed out that there were significant policy deficits regarding the use of constant observation in general hospitals. He concluded that psychiatry should become more involved in promoting policy in this area, and that more research should be done, including the use of constant observation instructions in cases other than potential suicide.

Among the AIDS inpatients referred for psychiatric consultations, 11 (4.3%) and 15 (5.9%) of the total of 256 new patient admissions were evaluated for violent behavior and suicidal behavior, respectively. Interestingly, seven of the 11 patients evaluated for violence were suffering from an organic mental disorder. Since a total of 968 AIDS patients were admitted to the general medical wards from January 1, 1988, to December 31, 1988, the 11 violent patients represented 1.14% of all the AIDS patients in the hospital.

This article represents an attempt to describe and place numerical values on violent behavior occurring in an inner city general hospital. Because of the great emotional impact that serious violent acts have on everybody in the hospital, there is a natural tendency to form the perception that violence occurs quite frequently. We have examined the existence of violent behavior and found that violent behavior in this general hospital is not as prevalent as previously believed. We nonetheless acknowledge that it is part of our medical responsibilities to evaluate and manage patients exhibiting such behavior. This evaluation should begin in the emergency room, where certain patients present as psychiatric emergencies manifesting violent behavior. On the general medical/surgical wards, the judicious use of psychiatric consultations may help avert the simple agitation of confused or maladaptive patients from escalating into full blown violence.

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Medical Society of the State of New York Sports Medicine Team Physician of the Year Award

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Multiple addictions: Co-synchronous use of alcohol and drugs

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Identifying multiple drug addictions is crucial to the clinical diagnosis, prognosis, and treatment of psychoactive substance dependence, as well as to the formulation of etiologic research models of abuse and addiction. Theories of genetic transmission of alcoholism and drug addiction challenge traditional behavioral theories of addiction. Similarly, the concept of multiple addiction challenges neurobiologic theories of addiction to individual drugs, as it suggests a universal susceptibility to alcohol and drug addictions.

Today's alcoholic usually becomes addicted to alcohol first but goes on to develop other drug addictions at an alarming rate. Consequently, most alcoholics under age 30 are addicted to at least one other drug. Most often, they have multiple addictions with consequences similar to those of their alcohol addiction. Although alcohol is not usually the drug of choice for persons with multiple addictions, it tends to be used in addition to or in lieu of the drug of choice.[1] It is rare to find a drug addict who is addicted only to one drug. For example, one does not typically see a heroin addict who is not or has not also been addicted to marijuana and/or alcohol, or more recently, cocaine. The common practice of adulteration (mixing in the preparation) of one illicit drug with another drug makes it difficult, if not impossible, for the addict to sustain a monodrug addiction, particularly when the drugs are obtained on the street.

The number and variety of drugs sought by the multiply addicted has become increasingly extensive and exotic. In addition, the traditional distinction between illicit drug use and therapeutic use of prescribed medications is not useful. Medical drugs are often used nonmedically by drug addicts.

The effect of multiple addiction on personality development and the evolution of psychiatric symptoms is not well understood. Although the multiply addicted are frequently observed to have greater personality disturbances than monodrug addicts, the impact of alcohol and multiple drugs on personality functioning has not been measured in any meaningful way. The alcoholics and drug addicts of today usually become addicted sometime during adolescence, when the personality is still developing and when no stable or mature identity has been established. The resulting picture is often one of alcoholism and drug addiction combined with an immature personality. It appears that the addict's personality undergoes an arrest of maturation when the onset of addiction occurs.

In addition, drugs have pharmacologic effects on the brain which critically affect behavior and other manifestations of personality functioning. Alcohol, marijuana, cocaine, opiates, sedative/hypnotics, and other drugs produce syndromes of intoxication and withdrawal that significantly disturb mood and cognition. These psychoactive effects are often chronic and cumulative in the multiply addicted with an often marked degree of personality disorganization.[2,3]

TRENDS IN ALCOHOL AND DRUG USE

The natural history of alcoholism is being altered by the phenomenon of multiple addiction. The course of one drug addiction is significantly affected by the addition of another drug addiction. Although not well documented, it is often clinically observed that the course of alcoholism is drastically truncated by an addiction to another drug. Many alcoholics, when giving a history, make the comment that the course of their alcoholism could have continued uninterrupted had they not developed an addiction to cocaine, opiates, or sedatives. Clinical experience strongly supports this notion, as in many instances it was the development of an addiction to another drug that motivated the addict to seek treatment, in spite of an already longstanding addiction to alcohol with significant adverse effects. Furthermore, the younger the addict, the more common the occurrence of multiple addiction.

The National Institute on Drug Abuse (NIDA) has conducted an annual monitoring survey since 1975.[4] This survey has become the most complete and frequently cited reference on alcohol and drug abuse. It consists of interview responses of a nationally representative sample of high school seniors who are usually interviewed over the telephone. Those students who have dropped out of school or are chronically absent (approximately 20%) are not polled, which may lead to sampling bias, as many of these dropout students may have alcohol and drug problems which led to their poor academic outcome.[5] Consequently, the survey may underestimate drug use. In 1985, lifetime use of alcohol by high school seniors was 93%; of marijuana, 59%; of cocaine, 16%; of other stimulants, 16%; and of tranquilizers, 14%. For these same drugs, reported use in

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the last month was 70%, 29%, 5%, 14%, and 2%, respectively.[5] NIDA also sponsors another national survey of households in the United States to assess prevalence of drug use by young and old. Similar figures were obtained for the young as in the high school survey. In addition, this survey also demonstrated the inverse relationship between age and other drug use among alcoholics.

The Drug Abuse Warning Network (DAWN), which records visits to emergency rooms in the United States, has found that alcohol was most frequently used in combination with other drugs, accounting for 24% of all drug-related episodes. Data were not collected for episodes related to alcohol alone.[4]

The National Youth Polydrug Study has found that a sample of 2,750 alcoholic youths regularly used a mean of 4.4 other drugs, most commonly marijuana and alcohol (86% and 80%, respectively). Amphetamines had the third highest prevalence (45%), followed by hashish (42%), barbiturates (40%), hallucinogens (40%), and phencyclidine (PCP) (32%).[6,7]

As far back as 1930, alcoholics have used other drugs in alarming frequency. Freed reviewed 15,447 cases in 46 studies during the years between 1930 and 1970, finding 3,046 alcoholics (20%) who were also addicted to another drug. These other drugs included some of the same ones used by alcoholics today, ie, barbiturates, opiates, benzodiazepines, and marijuana.[8]

There have been only a few studies that have attempted to establish actual prevalence of multiple drug addictions. Until recently, the prevalence of alcoholism was based on estimates of consumption rates of alcohol or of medical consequences of alcohol use. The Epidemiological Catchment Area (ECA) study was the first national assessment of the prevalence of alcoholism according to the standardized diagnostic criteria for alcohol abuse and dependence contained in the DSM-III.[9,10] The ECA study was carried out by a team of researchers assigned to sites in five US cities, namely, St Louis, Los Angeles, Baltimore, New Haven, and Durham. The study was conducted in a standardized fashion with identical methods employed in each site. Unlike other surveys of drug use prevalence, which utilize written, self-administered questionnaires or telephone interviews, these subjects were given a structured personal interview. The findings indicated that alcoholism is a relatively common disorder, with a lifetime prevalence of 13.4% in the total sample population. For men, the lifetime prevalence was even more dramatic, with almost a quarter of the sample (23.8%) meeting criteria for alcoholism. The rate for women was much lower at 4.6%, but that figure appears to be steadily increasing. Interestingly, about half of those who met lifetime criteria for alcoholism had active alcoholism in the past year. Another important finding was that the prevalence of alcoholism is significantly higher in men and women under the age of 45 years. For men, lifetime prevalence for ages 18–29 years was 27%, versus 28% for ages 30–44, and 21% for ages 45–65. For women, lifetime prevalences were 7%, 6%, and 3%, respectively, for these age groups.[9,10]

Alcoholism is increasingly recognized as a youthful disorder. Almost 40% of the cases in the ECA study began abusing alcohol between the ages of 15 and 19, and 80% had begun by the age of 30. Men typically reported an earlier onset of alcoholism than did women: the average age of onset of alcoholism was 22 years in men versus 25 years in women. More than half of the sample (54%) had a duration of alcoholism of five years or less.[9,10]

The prevalence of drug addiction among alcoholics was fairly high in the ECA study: as many as 30% of the alcoholics met criteria for a drug addiction. Marijuana addiction was the most common. The reverse situation—the prevalence of alcoholism among drug addicts—was also quite high, with alcoholism among marijuana addicts being 36%, among barbiturate addicts 71%, among amphetamine addicts 62%, among hallucinogen addicts 64%, among opiate addicts 67%, and among cocaine addicts 84%. Interestingly, less than 1% of the total sample admitted to cocaine addiction at some time in their lives, but 84% of these also had alcoholism. Overall, this study demonstrates that in the general population, alcoholism is a common complicating factor in the majority of cases of drug addiction.[9,10]

There is even greater overlap of alcoholism and drug addiction when a select population is studied, such as those entering a treatment program. In such cases, the occurrence of multiple addiction increases significantly, although it parallels the patterns found in the general population.

For the contemporary drug addict, multiple drug use and addiction that includes alcohol is the rule. The monodrug user and addict is a vanishing species in American culture.[11,12] For example, large scale studies of inpatient populations of adult and adolescent alcoholics and drug addicts in various treatment facilities have found that between 70% and 90% of cocaine addicts are also addicted to alcohol. Similar studies of methadone and heroin addicts show rates of alcohol dependence between 50% and 75%. Approximately 80–90% of cannabis addicts are addicted to alcohol.

Most studies of alcoholics and drug addicts have found that in both groups, alcohol is the first drug used, often addictively. The natural history of alcohol dependence is highly variable and age dependent. Alcoholics who are over the age of 30 typically began drinking in adolescence, progressing to full-blown alcohol dependence in their early 20s. A certain proportion, perhaps 10% to 20%, also began using cannabis in their 20s. Another 10% may have begun using stimulants, including cocaine and amphetamines, while 20% may have used sedative/hypnotics, such as benzodiazepines, barbiturates, and meprobamates. About 50% will continue their alcohol dependence without significant use of any additional drugs.

In contrast, the alcoholic under the age of 30 progresses according to a different timetable, probably in response to the general increase in drug use in our culture. More than 80% of these alcoholics are addicted to at least one other drug; more typically, they are addicted to more than one. A triad of alcohol, marijuana, and cocaine addiction is a regular feature of the young alcoholics currently being admitted to inpatient and outpatient facilities. Typically, the younger alcoholic begins using alcohol in early teenage years, ages 13–15 years, and progresses to addictive use of alcohol by the age of 15 to 16. A year or two after the onset of alcohol use, other drugs are tried and some are then used addictively. The most frequent pattern involves use of marijuana, followed by hallucinogens such as PCP, benzodiazepines, and barbiturates. The typical pattern of cocaine use has changed and continues to change dramatically, especially with regard to consistently earlier age of onset of use. The skillful marketing techniques for the cheaper form of cocaine, "crack," have lured younger individuals to addictive use.[12,13]

Investigations of the utility of the DSM-III-R criteria for substance dependence disorders have confirmed these findings. The dependence syndrome as defined in DSM-III-R is used to diagnose all types of alcohol and drug dependence by utilizing the same criteria for addiction, tolerance, and dependence.[14] The findings of one important study supported the notion of a common dependence syndrome for alcohol and drugs, particularly alcohol, opiates, and cocaine.[15]

Diagnosis of Multiple Addictions

The identification of only alcohol dependence in a given patient is often tenuous and misleading. Because denial is a part of the addictive process, underreporting and underestimation of drug use is to be expected in a clinical interview, especially if only the alcoholic is interviewed. Corroborative sources increase the likelihood of obtaining a more accurate history, but still may not reveal the total pattern and amount of alcohol and drug use. These additional sources may include family members, employers, and legal agencies as well as urine and blood tests for drugs of abuse.[16]

Even in the most obvious cases, it is often difficult to get a reticent multiple addict to answer questions essential to diagnosis. Current criteria for addiction include a preoccupation with, compulsive use of, and relapse to alcohol and drugs. These are often flatly denied by many alcoholics and drug addicts who are actively using these substances. Questions about the development of tolerance and dependence on alcohol and drugs are equally difficult to ascertain adequately. Persistent pursuit of information in subsequent interviews as well as a knowledge of the natural history of alcohol and drug use and addiction, particularly in the multiply addicted, will often yield rewarding results.[17]

Clinical Course

The medical and psychiatric complications of multiple drug addiction are attributable to the effects of each drug singularly as well as of the combined effects of multiple drugs used simultaneously or sequentially. The medical complications are numerous but not nearly as common as the psychiatric complications, with the exception of intravenous (IV) drug use, which is associated with a substantial morbidity and mortality. The proportion of alcoholics who develop severe gastrointestinal, cardiovascular, and neoplastic sequelae is a distinct minority of the total population of alcoholics. Accidental, suicidal, and homicidal deaths remain common, especially among the young, for whom multiple drug use is significantly more predominant.

The IV drug addict is highly prone to develop acquired immunodeficiency syndrome (AIDS). IV drug addicts who have AIDS readily transmit the virus to other drug addicts by engaging in activities that involve an exchange of bodily fluids between donor and recipient, such as sharing syringes and needles, and engaging in sexual contact. Other relatively common sequelae of IV drug use are viral hepatitis, endocarditis, pulmonary infections and allergic reactions, meningoencephalitis, brain abscesses, lethal overdoses, accidents, and trauma.[3]

Drug induced psychiatric syndromes are clinically indistinguishable from the idiopathic psychiatric disorders they mimic. Personality disorders, mania, depression, schizophrenia, anxiety disorders, and eating disorders may be caused by alcohol and drugs, especially addictive use of these substances.[18,19]

One of the most devastating effects of alcohol and drugs involves their impact on the developing personality of the younger addict. The fundamental disturbance in personality due to alcohol and drug addiction seems to occur in the area of personal relationships. This disturbance in interpersonal relationships affects many spheres of the addict's life, including the home, friends, school, employment, and the larger society. There is a characteristic personality which appears to develop after years of chronic use and which may be typified by a narcissistic, antisocial, depressive, dependent, immature, and histrionic core. This core personality is clinically observed to be more pronounced and prominent in the multiply addicted.

A key factor in the development of this core personality appears to be a function of the addictive process itself, ie, the preoccupation with acquiring multiple drugs and using them compulsively with a loss of self-control. Additionally, the toxic effects of alcohol and drugs on the brain produce an organic syndrome that impairs insight and judgment, fosters impulsivity, disturbs mood, and affects memory and cognitive functions. These adverse effects may be expressed in various aspects of personality functioning. They tend to be more evident in multiple addictions because of multiple drug effects as well as the life style required to obtain expensive, illicit drugs.

The existence of a personality that predisposes one to alcoholism and drug addiction or a general addictive process has not been confirmed. No one personality type seems to determine the biological development of addiction. However, some types of personalities appear to promote exposure to alcohol and drugs, especially with regard to the development of multiple drug addiction. The antisocial personality in adults and the conduct disorder in children enhance the potential for exposure to alcohol and drugs, especially at a young age. This early and significant exposure to multiple drugs combined with a particularly vulnerable personality type frequently results in the development of an addictive process.

Another interesting phenomenon that is frequently observed clinically but is poorly documented in research studies is the arrest of personality development and maturation at the time of onset of the addiction. This seems to occur whether the onset of the addiction is in childhood or in adult life. It is not uncommon for the multiply addicted with a previously well-integrated personality to undergo a deterioration in personality functioning characterized by the appearance of narcissistic, antisocial, or depressive characteristics which increase with the progression of the addiction. The developing personality in childhood may not only be delayed, but may be supplanted by the core personality of the multiple addictive process. Arguing whether the personality or the multiple drug use came first is fruitless, as the core personality of the multiply addicted may be integrated into the individual's basic personality, so that it is impossible to distinguish between the two.[2,3,20] The organic personality syndrome in DSM-III-R is analogous to the core personality and may be used to describe the effects of chronic multiple drug use on personality functioning.[14] This phenomenon is present in the adult as well, but here it involves a deterioration in personality rather than a delay in maturation.

A composite personality that illustrates the problems of the multiply addicted may be derived from an examination of individual drug effects on personality. All drugs, including alcohol, have "typical" effects on personality functioning. In the multiply addicted, these are manifested to varying degrees to form a core personality. For instance, the antisocial and violent traits associated with cocaine addiction and the criminal activity associated with heroin addiction may be combined with the narcissistic traits of the alcoholic to produce a perverse distortion of normal personality functioning.

Differential Diagnosis

The differential diagnosis of the multiply addicted is especially troublesome because of the various drug effects on the mind and behavior. The severe disturbance in mood and affect produced by drugs may be associated with mood congruent psychotic delusions, psychomotor retardation, vegetative symptoms, and suicidal actions. For example, alcohol produces significant depression that is indistinguishable from major depression as defined in DSM-III-R. Alcohol withdrawal, with its hyperexcitable and hyperaroused state, and alcohol intoxication, with its episodes of euphoria, poor judgment, and loss of control, have been confused with mania. In addition, the repeated stimulation of the sympathetic nervous system in alcohol intoxication and withdrawal may produce an anxiety syndrome that is indistinguishable from disorders involving generalized anxiety, panic attacks, and phobias. In fact, phobias such as agoraphobia are quite common in alcoholics. Finally, alcohol hallucinosis with auditory and occasionally visual hallucinations needs to be differentiated from schizophrenia.

Cocaine and other stimulants produce effects that fulfill the criteria for mania—a triad of euphoria, hyperactivity, and distorted self-image. The withdrawal from cocaine, particularly in chronic use, is characterized by severe depression with attendant signs and symptoms that fulfill criteria for major depression. Chronic cocaine use may produce delusions, particularly of a paranoid type, and hallucinations, both visual and auditory, which may be difficult to distinguish from schizophrenia. Furthermore, the anxiety generated from the pharmacologic effects of addictive chronic cocaine use is in the form of generalized anxiety, panic attacks, and intense agoraphobia.

Marijuana, phencyclidine, and other hallucinogens produce intense disortions of mood, affect, thinking, and perception with the consequent development of depression, mania, delusions, and hallucinations. The chronic effects of marijuana are similar to those of both alcohol and cocaine, as marijuana appears to have psychopharmacologic effects somewhere between sedation and stimulation. Other hallucinogens such as LSD, methamphetamine, and psilocybin share properties with marijuana.

The task of sorting out and attributing various drug effects to the correct drug is difficult and at times impossible. Similarly, one may be unable to differentiate the effect of acute and chronic drug use and addiction from other psychiatric disorders. An important aspect of differential diagnosis is not being compelled to make a single, final diagnosis too quickly. It is essential to keep in mind that alcohol and drug addiction are primary disorders that produce multiple complex symptoms and syndromes. The treatment of multiple addiction with detoxification and abstinence will frequently be sufficient to establish the definitive diagnosis. However, occasionally the drug effects, particularly in the multiply addicted, will persist for protracted periods so that diagnosis becomes even more problematic.

Pharmacologic Intervention

Complete knowledge of all drugs used in the multiply addicted is critical in treating the patient during the acute detoxification period as well as in sustaining recovery and preventing relapse. Different drugs, including alcohol, may require individualized detoxification regimens that do not always overlap. For example, the physiologic withdrawal from alcohol is treated with benzodiazepines; the delusional and hallucinatory symptoms of PCP withdrawal with a neuroleptic; and opiate withdrawal with clonidine or methadone. Furthermore, the protracted withdrawal from hallucinogens, cannabis, and stimulants may require prolonged pharmacologic intervention with neuroleptics and antidepressants if psychotic and depressive symptoms persist.

Treatment modalities for addiction are affected by other drug use, especially if alcohol addiction is present. The principles of abstinence-based treatment programs such as Alcoholics Anonymous (AA) will work for the alcoholic who has additional drug addictions. However, individualized education and support are indicated for specific drugs like cocaine and opiates. In general, the similarities among the multiple drug and alcohol addictions are greater than the differences, so that recovery in self-help groups such as AA and Narcotics Anonymous (NA) is recommended for the multiple addict. Finally, individual psychotherapy may be directed at the core personality of alcoholism and multiple drug addiction as well as at its effects on the mind and behavior.

The management of overdose or intoxication should involve a thorough history and physical examination, including drug screens of blood and urine. Because of the typical denial that is present in the multiply addicted as well as in those who know and are associated with him or her, a reliable account of type and severity of drug use is difficult if not impossible to obtain. For this reason, it is desirable to assess the patient's clinical condition with an open mind and always to maintain a differential diagnosis regarding the drugs involved in the overdose or intoxication.

In the case of overdose, the stomach should always be emptied by inducing vomiting or using activated charcoal. If the clinical condition does not improve, other drug states should be considered. It is routine in emergency rooms to give naloxone to attempt to reverse opiate overdose. In addition, the respiratory and cardiovascular systems need support in cases of multiple drug overdoses such as alcohol, opiates, sedative/hypnotics, and antidepressants. These drugs have a consistent synergistic depressant effect on respirations and may generate arrhythmias in the cardiovascular system. Psychotropic medications such as antidepressants are frequently involved in overdoses with other drugs, particularly alcohol. Constant monitoring of the vital signs in intensive care units may be required in cases of overdose.

The physician should treat objective signs of intoxication and withdrawal, but should take the self-report of the multiply addicted with skepticism. The drug-seeking behavior of an addict may be confused with signs and symptoms of withdrawal, so that iatrogenic overuse of medications may occur in a population in which the use of drugs needs to be minimized. This drug-seeking behavior in the multiply addicted may be particularly strong because of the high potential interchangeability of drugs of abuse.

Because of the general vulnerability to alcohol and drug addiction in the multiply addicted, the use of any drugs or medications renders a risk of reactivating the addiction. Anticholinergic and antihistaminic drugs often become addictive in the multiply addicted. Even aspirin, acetominophen, or nonsteroidal anti-inflammatory agents, such as ibuprofen, may be used addictively, particularly by a multiple drug addict.

The clinical use of "anticraving" or blocking drugs in the multiply addicted is not a common practice. These drugs are used most often in the acute withdrawal period and are not usually administered on a continuous basis. Abstinence from all drugs should be the ultimate goal in this vulnerable population. However, bromocriptine and antidepressants have been used in treating the craving for cocaine. Bromocriptine is a dopamine agonist, while desipramine enhances dopamine transmission for the purported effect of neutralizing the dopamine depletion which is believed to underlie the cocaine craving. Only modest experimental success has been achieved with these drugs in treating the multiply addicted in the acute withdrawal period.

Antabuse (disulfiram) is a drug that has been used with limited efficacy over the years. Antabuse is a competitive inhibitor of alcohol dehydrogenase, an enzyme which is responsible for degrading acetaldehyde, the breakdown product of the action of the enzyme alcohol dehydrogenase on alcohol. Acetaldehyde is a noxious intermediary that produces an unpleasant syndrome of nausea, vomiting, and lightheadedness. Unfortunately, in severe reactions, it can produce cardiovascular collapse. The more alcohol consumed and the higher the dose of antabuse, the more severe the adverse reaction. A major difficulty in the use of this drug is that the alcoholic must continue to take antabuse daily in order for the reaction to take place when alcohol is imbibed. It only takes a day or two for the antabuse to be washed out and for alcohol to be consumed without significant interactive effect. At times, the alcoholic is able to drink "through" or in spite of the antabuse reaction.

In conclusion, identification of multiple drug addiction is critical in the diagnosis and treatment of today's alcoholics and drug addicts. It is important for the clinician to recognize the various intoxication and withdrawal syndromes attributable to each specific drug as well as the medical and psychiatric complications of combining such drugs. Unless contemporary treatment methods are adapted to fit changing patient characteristics, attempts at rehabilitation may be futile.

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Central nervous system malignancies in children

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Primary tumors of the central nervous system (CNS) constitute the most common type of solid tumor in childhood. A widely held perception, even among health care professionals, is that the prognosis for children with these tumors is bleak. In fact, more than 50% of all children with brain tumors will be alive five years after diagnosis,[1] and progress in the treatment of some highly malignant tumor types, such as the medulloblastoma, has been impressive. Forty years ago, virtually no child survived with this diagnosis, while today more than half of patients with medulloblastoma are long term survivors without recurrence of disease.[1]

Nonetheless, improvement in the treatment of childhood brain tumors has not been as rapid as that seen for many other childhood cancers, such as Wilms' tumor or acute lymphoblastic leukemia. Brain tumors still account for a disproportionately large number of childhood cancer deaths.

There are several reasons for the relative slowness of progress in the therapy of childhood CNS malignancies. While these tumors as a group are a relatively common form of childhood cancer, the number of children with any single tumor type such as medulloblastoma, high grade astrocytoma, and other tumors, is small. This lack of patients prevents the accrual of sufficient numbers of patients to conduct therapeutic trials.[2] Further aggravating this problem has been the self-fulfilling expectation among most physicians that, because of the presumed bleak prognosis, vigorous treatment is not warranted.

In addition, basic biologic problems face the neuro-oncologist. A major factor in the increasing success of treatment of non-CNS tumors in childhood is the ability of chemotherapy to eliminate micrometastases, thus preventing recurrence of tumor at sites distant from the original site. However, control of systemic metastases is not the major therapeutic problem with most CNS tumors. Tumor recurrence at the original site accounts for the majority of treatment failures.

The techniques useful for preventing local failure with non-CNS tumors, such as radical "cancer" surgery with wide margins of excision or wide-field high-dose radiotherapy, are less applicable to tumors of the nervous system. In addition, delivery of effective chemotherapy is, to some extent, limited by the blood-brain barrier.[2] Finally, as with therapy of cancer in any location, aggressiveness of treatment must be balanced against likelihood of damage to normal tissue. The therapeutic window, or risk-benefit ratio, in the treatment of tumors of the CNS is particularly small. Thus, minimizing the impact of therapy on the developing brain is a vital concern.

EPIDEMIOLOGY OF CHILDHOOD BRAIN TUMORS

About 1,500 children receive a diagnosis of brain tumor each year. Duffner et al[1] examined the Surveillance-Epidemiology-End Result (SEER) registry data, a population-based data reporting system covering approximately 10% of the US population, for the interval 1973 to 1980. She found that in children less than 15 years of age at diagnosis, 25% of brain tumors are low grade astrocytomas, 23% medulloblastomas, 12% cerebellar astrocytomas, 11% high grade supratentorial astrocytomas, 9% brain stem gliomas, and 8% ependymomas.[1] In very young children, the distribution of tumor types is somewhat different, with medulloblastomas followed by ependymomas and low grade astrocytomas.[3] Brain stem gliomas and cerebellar astrocytomas are rare before age two years, but low grade astrocytomas of the hypothalamus, optic tracts, and thalamus are relatively more common in these very young children.[4]

A relatively new tumor classification deserves mention, as it frequently appears in recent literature. The term primitive neuroectodermal tumor (PNET) was originally used by Hart and Earle.[5] The term is used to describe a group of cerebral tumors that are highly malignant and are histologically indistinguishable from medulloblastomas and other primitive CNS malignancies, such as ependymoblastomas and pinealoblastomas.[5] There is a common histologic appearance among this group of tumors, and the primitive neuroectodermal tumor shares with these other primitive tumors the propensity to spread throughout the neuraxis. Thus, many authors now use the term primitive neuroectodermal tumor to refer to this entire group of tumors, including medulloblastomas.

The etiology of brain tumors in childhood remains for the most part unknown. Patients with neurocutaneous syndromes, such as neurofibromatosis and tuberous sclerosis, are at increased risk of a CNS tumor.[5] Also, cancers of the CNS or hematopoietic-lymphopoietic system occur more frequently in relatives of children with CNS tumors, as compared to controls and to known incidence rates.[6] This occurrence suggests an inherited predisposition to develop these tumors.

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On the other hand, environmental factors have been implicated in the etiology of brain tumors. A possible relationship to prior CNS irradiation is suggested by a number of reports of the development of malignant glioma years after the administration of radiation to the CNS.[7] Also, Miller et al[8] recently reported that ten out of 468 patients with acute lymphoblastic leukemia treated with cranial irradiation and intrathecal methotrexate developed CNS malignancies. Farwell et al[9] noted an increased incidence of medulloblastoma in the offspring of women who received polio vaccine contaminated with the papovavirus SV40. This finding, plus the fact that the SV40 virus causes CNS neoplasms in animals, suggest that antenatal exposure to this virus may be related to subsequent development of medulloblastoma.

SIGNS AND SYMPTOMS

Compared to children with acute lymphoblastic leukemia or Wilms' tumor, children with brain tumors experience the greatest delay between the onset of symptoms and diagnosis.[10] Symptoms are usually nonspecific, reflecting increased intracranial pressure: thus, headache, vomiting, or both are characteristic findings. These symptoms may occur daily, often in the early morning. Initially, the symptoms may be episodic and can be mistaken for symptoms of a viral infection. However, the persistence and duration of symptoms, or the onset of additional symptoms, such as diplopia, distinguish the patient with brain tumor. Ataxia is the most frequent focal symptom of childhood brain tumor, reflecting the high incidence of posterior fossa lesions, while occurrence of focal convulsive seizures is less common.[5]

It is even more difficult to diagnose brain tumors in very young children. Again, symptoms of increased intracranial pressure, such as unexplained and episodic vomiting, are often found.[4] Since other causes of vomiting are not unusual in this age group, very young patients with brain tumors often receive extensive diagnostic evaluation of organs other than the brain prior to diagnosis. In children less than one year of age, expanding head size is often noted.[4] Another presentation unique to the very young is that of failure to thrive, the so-called diencephalic syndrome, which is most often seen in children with tumors of the hypothalamus or optic chiasm. This latter group of patients frequently also show abnormal eye movements.

DIAGNOSTIC TOOLS

The development of computed tomography (CT) revolutionized brain tumor diagnosis. The specificity and lack of associated morbidity of the technique make it a superb method for initial diagnosis as well as for subsequent follow-up. Most childhood brain tumors are easily visible on CT, particularly with contrast enhancement. The CT scan has proven to be very useful in postoperative studies to determine extent of residual tumor, when the scan is obtained within 72 hours after surgery to avoid post-surgical artifact. Finally, computed tomography allows close follow-up, usually every three months, with the capability of early detection of recurrence. Such follow-up provides more accurate evaluation of tumor response to therapeutic intervention, which may be of prognostic significance.[11] Close follow-up also allows objective assessment of anti-tumor activity of new chemotherapeutic agents and drug combinations (Figs 1, 2). CT has thus largely superseded arteriography and pneumoencephalography in the evaluation of patients with brain tumors.

Certain tumors (such as medulloblastoma, malignant ependymoma, PNET, and dysgerminoma) have a tendency to disseminate throughout the neuraxis. For example, in some studies, as many as 40% of patients with medulloblastoma, although usually exhibiting no symptoms, have evidence of metastatic spread of tumor visible on a myelogram.[12] Thus, it is necessary to examine the spinal subarachnoid space of patients with newly diagnosed tumors. Contrast myelography remains the "gold standard" for this purpose, although magnetic resonance imaging (MRI) with paramagnetic contrast may replace the more invasive myelogram if ongoing comparative trials show that the two methods are equivalent.

Magnetic resonance scanning has proven to be particularly useful in evaluating those areas of the CNS that are difficult to examine by CT, such as the base of the brain and the spinal cord. Extent of tumor may be better visualized by MRI. For example, ependymomas of the posterior fossa not uncommonly extend into the upper cervical cord, and thus are not well seen on CT.[13] Because of the ability to create an image in multiple planes, the three-dimensional relationship of tumor to the surrounding brain can be seen. This capability is useful to both the neurosurgeon and the radiation oncologist. Finally, magnetic resonance imaging appears to be more sensitive than

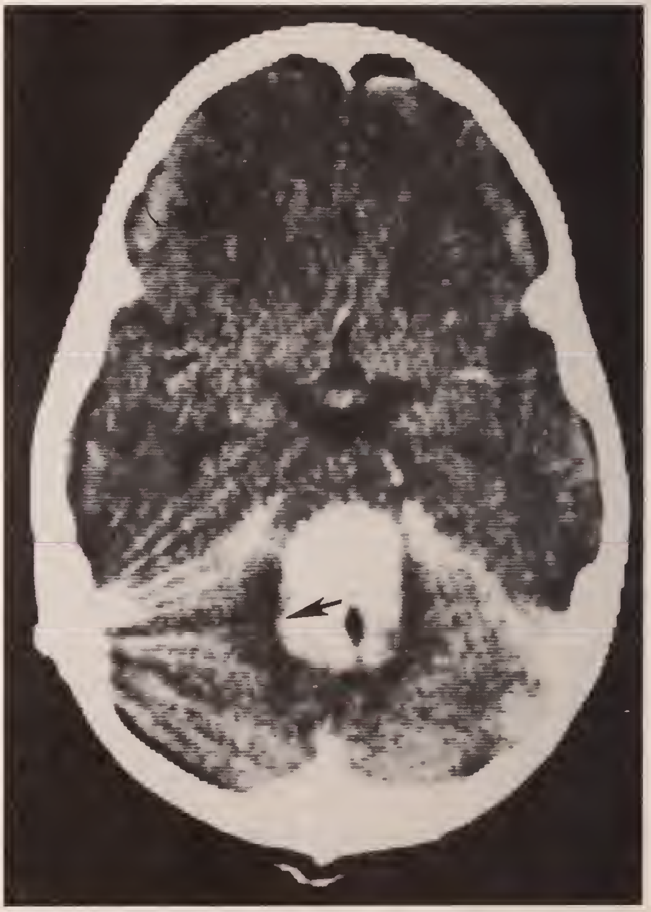

FIGURE 1. Head CT scan of child with recurrence of medulloblastoma (arrow).

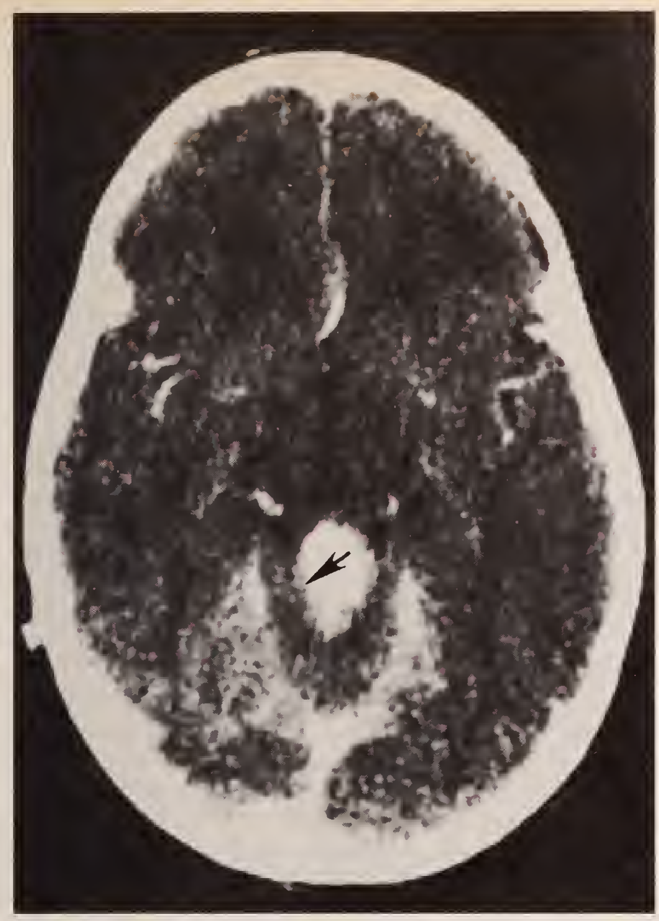

FIGURE 2. Head CT scan of same patient as in Fig 1, showing disappearance of tumor (arrow) after two courses of chemotherapy (according to the "eight drugs in one day" regimen).

computerized tomography in evaluating the effects of treatment on the brain, such as the changes in white matter noted following CNS irradiation,[6] although CT is superior for demonstrating postradiation calcification.

In addition to the use of radiologic examinations for staging of a brain tumor, examination of the cerebrospinal fluid can provide useful information, particularly in those tumor types with a tendency to spread throughout the neuraxis. Except in specimens of spinal fluid obtained shortly after surgery, the presence of malignant cells in the CSF provides direct evidence of tumor dissemination and appears to be of prognostic importance.[2] Levels of alpha-fetoprotein and human chorionic gonadotropin in the serum and cerebrospinal fluid are useful in the diagnosis and follow-up of germ cell tumors.[4] In addition, increased levels of CSF polyamines, particularly putrescine, have been correlated with the recurrence of medulloblastoma in some studies.[14]

NEW TECHNIQUES IN NEUROSURGERY

With few exceptions, the initial therapy for the child with brain tumor is surgery. If complete resection is possible in the patient with a low grade supratentorial astrocytoma or cerebellar pilocytic astrocytoma, long term, recurrence free survival is likely without additional adjuvant therapy. For highly malignant tumors, such as malignant glioma and medulloblastoma, total resection is associated with better survival than that following less complete resection.[15-17] Major improvements in pediatric anesthesia and intensive care have greatly reduced the morbidity and mortality of brain tumor surgery. With the availability of new technical equipment, more aggressive tumor resections are feasible without jeopardizing patient safety. We describe several of these techniques.

It is often difficult for the surgeon to distinguish tumor from normal adjacent brain tissue by direct visual examination alone. Intraoperative ultrasound readily identifies echogenic tumor and normal anatomical structures in contrast to hypoechoic normal brain and edema.[18] Contrast enhancing and non-enhancing lesions depicted on CT scans are routinely identified with sonographic techniques. Current investigations indicate a close (within 5mm to 10 mm) correlation between intraoperative ultrasound findings and the CT scan in defining tumor boundaries, thus enabling greater tumor resection without sacrificing normal adjacent brain tissue.

The use of CT-stereotactic localization techniques coupled with the surgical laser has resulted in a novel computer-assisted system that enables the neurosurgeon precisely to remove deep lesions.[19] This technique allows the resection to correspond with any abnormal area visualized on the CT or MRI scan.[20] More superficial lesions located in critical functional areas can now be treated in the same manner. CT-stereotactic localization and intraoperative physiologic mapping of the brain are used to avoid damage to regions that control language, sensory, and motor functions. Children less than 10 to 12 years of age who cannot tolerate surgery while awake can have language cortex identified prior to tumor resection. This is accomplished by first implanting a subdural electrode array while the patient is under general anesthesia. The child is then tested for speech arrest by subdural electrical stimulation while he or she is awake on the hospital ward. Thus, the extent of surgical resection can be maximized to a greater degree than was previously possible in eloquent brain regions without sacrificing patient safety.

Advanced surgical aids also enable more aggressive surgery of spinal cord tumors. Somato-sensory-evoked potentials may be elicited by stimulation of the median and posterior tibial nerves to enable early detection of spinal cord dysfunction during surgical manipulation.[21] High resolution, near-field (10 megahertz) ultrasound probes allow exact localization of solid tumor and adjacent syrinx cavities before myelotomy. Improved operative techniques afforded by the use of evoked potentials, ultrasound, operative microscopy, and the cavitron ultrasonic surgical aspirator (CUSA) have enabled radical tumor resections throughout the entire length of the spinal cord.

A radical resection is not always possible for many lesions of the thalamus or brain stem. With recent advances in sterotactic surgery, any intracranial region of interest may be safely biopsied with millimeter precision.[22,23] Lesions greater than 1 cm in diameter and located in more superficial areas can also be sampled accurately through a burr hole, with the aid of high resolution ultrasonography.[24] This special ultrasound technique does not require the bulky coupled sterotactic head frames of CT or MRI.

ADJUVANT TREATMENT

Medulloblastoma is the most frequently occurring highly malignant tumor in childhood. In the experience of Harvey Cushing, who first described this tumor, medulloblastomas were nearly always fatal—only one out of 60 patients on whom Cushing operated were alive after three years.[5] Introduction of focal radiation therapy did little to alter this bleak prognosis. Prolonged survival of children with tumor only occurred when the tendency of the tumor to spread throughout the neuraxis was recognized; total neuraxis radiotherapy was introduced in the 1950s. Bloom et al[25] noted a 30% five-year survival in the late 1960s, and the SEER data demonstrated a 45% five-year survival in children treated between 1973 and 1980.[1]

In addition to the need for irradiation of the neuraxis, the importance of delivering a sufficient total dose to the site of the primary tumor has been emphasized by several studies. These studies demonstrate a dose-response relationship for this tumor. Single institutions now report five-year event-free survival rates of greater than 50% in those children given a dose of at least 5,000 centigray (cGy) of radiotherapy to the posterior fossa.[17,26] While there are recurrences after five years, the majority of patients who are free of disease at five years are probably cured.

With the improvement in the overall survival of patients with medulloblastoma, certain features have emerged as being predictors of a poor prognosis. Several studies have demonstrated reduced survival in patients who have large tumors at diagnosis, or who have significant residual tumor remaining after surgery.[27,28] Metastatic spread of the tumor, either in the posterior fossa or throughout the neuraxis, is especially indicative of a high risk of tumor recurrence.[29] Young age (less than two to four years at the time of diagnosis) has been shown in most studies to be a poor risk factor. Very young children often have extensive disease at diagnosis and may receive less aggressive treatment because of concern for the late effects of therapy on the developing brain.[2] In a recent cooperative clinical trial on medulloblastoma (see below), two prognostic factors were statistically significant. These were age of the child and presence of metastases at the time of diagnosis.[30]

Tumor histology may also predict outcome. A recent retrospective study demonstrated a significantly better prognosis in patients whose tumors showed no evidence of differentiation to astrocytic, ependymal, or neuronal cell types. There was a four-year survival rate of 70% in patients with such undifferentiated tumors, compared to patients whose tumors had differentiated to one of these cell types (32% four-year survival), or to more than one cell type (10% four-year survival).[31] Another study, however, noted the opposite effect of histologic differentiation of the tumor on prognosis.[17]

As noted, the major factor in improving survival of patients with medulloblastoma has been the improvement of radiotherapy technique. The concept of giving prophylactic irradiation to the entire neuraxis provided a breakthrough in the treatment of this tumor, and the introduction of megavoltage equipment has allowed further refinement and precision in radiation therapy. It is not likely, however, that further advances in radiotherapy technique will lead to significant further increases in survival.

With the exciting successes of chemotherapy in treating other forms of childhood cancer, a reasonable hope has been that the addition of chemotherapy to surgery and radiation would further improve survival from medulloblastoma. Indeed, many trials of chemotherapeutic agents, given either singly or in combination, have demonstrated the chemosensitivity of recurrent medulloblastoma. This tumor appears to respond to many of the same agents that are effective in other primitive childhood tumors; these drugs include vincristine, cyclophosphamide, cisplatin, methotrexate, and the nitrosoureas (BCNU and CCNU).[5] However, it has not been easy to demonstrate the value of chemotherapy as an adjuvant treatment in the patient with newly diagnosed medulloblastoma. In an attempt to define the role of adjuvant chemotherapy in the treatment of medulloblastoma, the Children's Cancer Study Group (CCSG) in 1975 began a randomized trial of patients with newly diagnosed tumor. In this study, patients were randomly assigned to receive either postoperative radiotherapy alone or chemotherapy (consisting of vincristine, CCNU, and prednisone) plus radiotherapy (Fig 2). The study accrued about 200 patients and closed in 1981.[30] During this same time period, the International Society of Pediatric Oncology (SIOP) conducted a similar trial in Europe.

Overall, there was a 10% to 15% advantage in the rate of event-free survival in favor of chemotherapy in both studies. However, this improvement was statistically significant only in the SIOP study. In both studies, patients with extensive disease at diagnosis clearly benefited from the addition of chemotherapy. In the CCSG study, of those patients who had metastases at the time of diagnosis, 45% of those who had received chemotherapy had not suffered relapses at five years, while none of the patients who had received only radiation were free of disease. On the other hand, patients who had small tumors at diagnosis did not appear to derive any benefit from the addition of chemotherapy to radiation.[2,30] In summary, two large randomized trials of patients with medulloblastoma have demonstrated the value of adjuvant chemotherapy in a subset of patients, namely, those who are at high risk of recurrence. Another subset of patients, those without poor prognostic features, do not appear to benefit from chemotherapy as administered in these trials.

As is true with medulloblastomas, there is virtually no chance of survival in patients with high grade gliomas, such as anaplastic astrocytomas and glioblastoma multiforme, following surgery alone. This bleak outlook exists in spite of the fact that these tumors rarely disseminate widely throughout the neuraxis; local recurrence is certain even after wide resection that appears to remove all the tumor on gross examination. The addition of postoperative radiotherapy has significantly lengthened survival, but the majority of patients with these tumors die of their disease. Age is a strong prognostic factor, and children with high grade gliomas do somewhat better than adults. However, the SEER data (1973 to 1980) for high grade astrocytoma, including both anaplastic astrocytoma and glioblastoma multiforme, indicate a 40% five-year survival.[1] A review of the Connecticut Tumor Registry published in 1977 demonstrated a five-year survival rate of less than 5% following treatment with irradiation alone in children with glioblastoma multiforme.[32]

In 1975, a CCSG trial was begun to investigate the value of adjuvant chemotherapy in the treatment of children with high grade astrocytoma. The study design was identical to that described above for medulloblastoma. Patients were randomly determined to receive radiation and chemotherapy following tumor resection, or to receive radiation alone (Fig 2). In this study, a significant improvement in five-year survival was seen with the addition of chemotherapy; the survival rate was 42% as compared to a rate of 10% when only radiotherapy was given.[2,30] This was the first study to demonstrate unequivocally the value of adjuvant chemotherapy in the treatment of malignant gliomas.

Unfortunately, the outcome for brain stem gliomas has not appreciably changed in the last 20 to 30 years. The SEER data (1973 to 1980) showed a 20% five-year survival, and most series report a median survival of less than one year following radiation. These results from earlier studies are not significantly different from the results of a recently completed CCSG study. This study was virtually identical to the trials for medulloblastoma and high grade astrocytoma described previously (Fig 3), and it demonstrated no difference in outcome between patients receiving chemotherapy and radiation and those receiving radiation alone.[2] Likewise, a limited study in which 5-fluorouracil (5-FU) and CCNU were given prior to irradiation, with hydroxyurea being given during radiation, showed no improvement in outcome.[2] A subset of patients with brain stem gliomas having good prognostic features has been identified. These are patients with no cranial nerve abnormalities, with tumor visualized as non-hypodense on CT scan, and with low grade histologic findings. This subset of patients may do appreciably better following treatment with conventional radiotherapy.[33]

Low grade glial tumors have a much better prognosis than do the more malignant tumors described thus far, particularly if complete resection is possible. Patients with cerebellar astrocytoma have a five-year survival rate of greater than 90%, and those with low grade cerebral astrocytoma have a five-year survival rate of 70% to 80%,[1] though late recurrences may still occur. The usefulness of adjuvant therapy for these tumors is not clear, and no prospective randomized study has yet been undertaken. However, radiotherapy of the partially resected low grade glioma is probably of value.[5,34]

Treatment of gliomas of the optic chiasm continues to provoke considerable controversy. Surgical excision is not possible because of location, and some authors have suggested that even without treatment progression of the tumor does not always occur. In a recent extensive review of the literature, among patients with chiasmatic optic gliomas who had hydrocephalus, 75% of untreated patients and 50% of patients treated with at least 4,500 cGy of irradiation showed progression of their tumors.[35] If hydrocephalus was not present, 50% of patients had disease progression regardless of whether they received irradiation. The authors concluded that radiation therapy was possibly of benefit in gliomas of the optic chiasm.[35]

The benefits of radiotherapy in gliomas of the optic chiasm are uncertain, and there are known neurotoxic hazards following irradiation of very young children, in whom these tumors are often quite extensive. Thus, children less than five years of age with progressive gliomas of the optic chiasm have been treated with chemotherapy alone.[36] The results showed projected 45% disease free survival at five years for these children. Perhaps most importantly, progression of tumor occurred at a median of three years after the initiation of therapy. Therefore, irradiation, which arrested the tumor in all patients treated, was significantly postponed in these young children,[35] which possibly decreased long term morbidity.

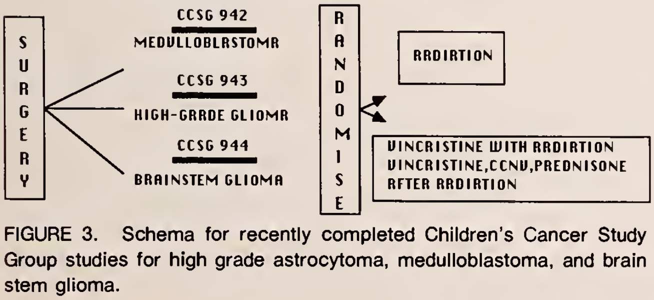

FIGURE 3. Schema for recently completed Children's Cancer Study Group studies for high grade astrocytoma, medulloblastoma, and brain stem glioma.

MORBIDITY OF TREATMENT

Long term survival is no longer rare in children with brain tumors. Thus, attention over the last decade has turned to the quality of life for the survivors. In particular, attention has focused on the long term effects of treatment.

Radiation therapy, with or without concomitant chemotherapy, can damage the CNS. This morbidity, often becoming apparent several years after treatment, ranges from the asymptomatic changes noted on CT (such as microangiopathic calcification) to fatal brain necrosis or leukoencephalopathy.

The most severe consequence of radiation of the CNS, brain necrosis, is rare with the total dose of irradiation and the size of the fractional doses now being used. Brain necrosis is characterized by vascular narrowing and occlusion, with onset usually months to years following radiation.[37] There is no evidence that children are more susceptible than are adults to this potentially devasting sequela, although doses of radiation are routinely decreased for the very young. However, the latent period between radiation treatment and the development of brain necrosis does appear to be shorter in children. Brain necrosis represents the dose-limiting factor for radiotherapy of the CNS.

Leukoencephalopathy is another devasting sequela of CNS-directed cancer therapy. This entity is characterized by global deterioration of the brain, often terminating in death or producing severe morbidity. Leukoencephalopathy is characterized by diffuse destruction of white matter, with little evidence of vascular involvement.[37] The syndrome in general has a shorter latency to onset than does brain necrosis, with neurological or psychological deterioration most often occurring witin one year after treatment begins.

The condition is very rare following either CNS irradiation alone or chemotherapy alone, and it is most commonly seen when whole brain irradiation is followed by administration of intravenous methotrexate.[38] Leukoencephalopathy may also occur following high doses of intravenous methotrexate at frequent intervals when radiation is not given. Other drugs, such as CCNU and cisplatin, have also been implicated, either when administered alone in very high doses intravenously, when given intra-arterially,[39] or when administered in association with radiotherapy.[40] With the development of more effective methods of drug delivery to the CNS, leukoencephalopathy may prove to be the dose-limiting toxicity of brain tumor chemotherapy.

Fortunately, both brain necrosis and leukoencephalopathy are uncommon. However, intellectual dysfunction often occurs following cancer treatment directed at the CNS. Initial reports of intellectual function in children with acute lymphoblastic leukemia following prophylactic treatment of the CNS with radiation and intrathecal chemotherapy with methotrexate were optimistic.[41] Subsequent reports, however, have indicated a significant decrease in children's IQ several years after treatment,[42] although the mean IQ appears to remain within the normal range. In addition, significant learning disabilities, not reflected by standard IQ tests, may be seen.[6] Younger children, and children with a higher IQ at diagnosis, appear to suffer greater intellectual loss.[43]

The long term intellectual status of children with brain tumors has not been as extensively studied as has that of patients with acute lymphoblastic leukemia, in part because there are fewer patients with brain tumors and fewer survivors. Furthermore, there are additional variables besides treatment that may adversely affect the IQ, such as the nature of the intracerebral tumor itself, the presence of hydrocephalus, as well as the type of treatment given, such as focal radiation therapy versus whole brain radiation. So many variables makes it difficult to analyze data in determining the cause of intellectual deficits of children following treatment for brain tumors.

Initial reports of intellectual status in survivors of medulloblastoma used global assessments of function and suggested that the majority of surviving patients were functioning well.[43] Later studies, using a more complete evaluation and including IQ testing, demonstrated a worse outcome. Thus, in one study, 75% of patients with medulloblastoma who received whole-brain radiation had an IQ of less than 80 on follow-up testing.[28] A more recent report, however, showed a mean IQ of 96 when children were tested three years after diagnosis of medulloblastoma.[45]

As in children treated for acute lymphoblastic leukemia, those treated for brain tumors appear to be at risk of developing specific learning disabilities. Impairment of perceptual skills occurs to a greater extent than impairment of verbal ability,[6] and school performance may lag behind that predicted by the IQ score. In a recent prospective evaluation of intellectual function in children with brain tumors, there was significant loss of IQ in children receiving whole brain irradiation, and the severity of this loss varied inversely with the age of the child at the time of treatment. Chemotherapy and focal radiation therapy did not appear to affect cognitive function.[44]

Children who survive brain tumor therapy appear to be at risk of impaired growth. This results both from radiation of the pituitary-hypothalamus and subsequent diminished growth hormone levels, and from radiation of the vertebral bodies if the entire neuraxis is irradiated. Finally, as noted above, children who survive childhood cancer may be at risk, probably less than 2%, of having second tumors develop.[4]

NEW DIRECTIONS FOR RESEARCH

The goals of present research in brain tumor therapy are to increase the number of children who survive free of disease and to minimize the adverse effects of therapy on the quality of life. Current nationwide treatment protocols are addressing both of these goals.

Adjuvant chemotherapy has been shown to be beneficial in the treatment of patients with medulloblastoma who have poor prognostic features and in patients with high grade astrocytomas. New trials are now studying whether intensification of chemotherapy can further increase survival in these patients. Two ongoing CCSG trials, one for medulloblastoma and malignant ependymoma and one for high grade astrocytomas, are randomly dividing patients into two groups. One group is given conventional chemotherapy of vincristine, CCNU, and prednisone; the other group is treated according to an intensive regimen consisting of eight antineoplastic drugs administered in one day (Fig 4). This latter, eight-drug regimen has produced a 50% response rate in patients with recurrent brain tumors as well as in newly diagnosed patients who had been treated with chemotherapy prior to irradiation.[46]

The "eight drugs in one day" regimen is given over a period of 18 hours. The drugs used include vincristine, CCNU, procarbazine, hydroxyurea, cisplatin, cytosine arabinoside, methylprednisolone, and either cyclophosphamide or DTIC (dacarbazine). The regimen was rationally designed based on current concepts of the biology of brain tumors. The selection of drugs was based on known activities of the various drugs against brain tumors, and their anti-tumor effects when given in combination or in sequence.

The goal of reducing toxicity and long term sequelae is also being addressed in group studies. The Children's Cancer Study Group and the Pediatric Oncology Group (POG) are conducting a joint study to determine the effect of lowering the dose of prophylactic neuraxis irradiation on survival and on neuropsychologic outcome. In this study, patients with medulloblastoma who have no poor prognostic features are randomly assigned to receive craniospinal irradiation at a dose of either 2,340 cGy or 3,600 cGy.[30] All patients receive the standard total dose of irradiation to the posterior fossa.

The outcome for patients with brain stem glioma re-

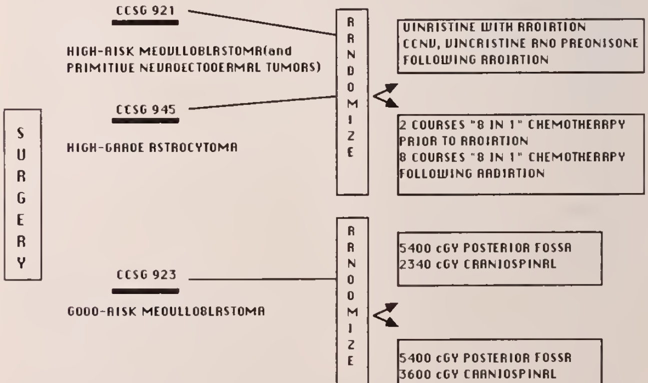
CCSG 921
HIGH-RISK MEDULLOBLASTOMA(and PRIMITIVE NEUROECTODERMAL TUMORS)
CCSG 945
HIGH-GRADE ASTROCYTOMA
SURGERY
CCSG 923
GOOD-RISK MEDULLOBLASTOMA
RANDOMIZE
VINCRISTINE WITH RADIATION
CCNU, VINCRISTINE AND PREDNISONE FOLLOWING RADIATION
2 COURSES "8 IN 1" CHEMOTHERAPY PRIOR TO RADIATION
8 COURSES "8 IN 1" CHEMOTHERAPY FOLLOWING RADIATION
RANDOMIZE
5400 cGY POSTERIOR FOSSA
2340 cGY CRANIOSPINAL
5400 cGY POSTERIOR FOSSA
3600 cGY CRANIOSPINAL

FIGURE 4. Schema of current protocols conducted by the Children's Cancer Study Group for children with brain tumors.

mains bleak. To date, no study has shown that any benefit is gained by the addition of chemotherapy to radiotherapy. Thus, an attempt is being made to increase the dose of radiation delivered to the tumor by using smaller, more frequent fractions of radiation. This radiotherapy technique is called hyperfractionation. This approach is based on the concept that normal tissues recover more quickly than neoplastic tissue from a fraction of radiation, and a total dose of radiation is better tolerated if it is delivered in smaller fraction sizes.

Very young children with malignant brain tumors have a worse prognosis than older children, in terms of both survival and of neuropsychologic outcome. In an attempt to avoid or at least delay irradiation of children less than three years of age, they have been given chemotherapy using the MOPP regimen, which includes nitrogen mustard, vincristine, procarbazine, and prednisone. Radiotherapy was not given unless there was tumor progression. This study has produced promising results.[47]

In a Pediatric Oncology Group study, patients less than three years of age with malignant brain tumors are being treated either with chemotherapy alone until there is tumor recurrence, or until they have completed one or two years of chemotherapy, at which time they receive conventional radiation.[47] In another group study, all children less than 18 months of age who have malignant brain tumors are being initially treated with chemotherapy alone. While these children will ultimately receive irradiation as part of their treatment, it is hoped that the delay will lessen radiation-induced neuropsychologic sequelae.

Continued progress in the treatment of children with brain tumors will require improvement in the delivery of therapeutic agents to the CNS. This improvement may come from new drugs that can more readily penetrate the blood-brain barrier. Another possibility is administration of a hematopoietic growth factor, such as granulocyte-monocyte colony stimulating factor (GM-CSF). This would hasten recovery of the bone marrow following suppression by chemotherapy and would allow the use of much higher levels of systemic chemotherapy.

Targeting tumor tissue with tumor-specific monoclonal antibodies may allow more precise delivery of cytotoxic agents. This approach may be particularly useful in the treatment of subarachnoid micrometastases. In vitro analysis of tumor sensitivity may identify the most cytotoxic agents for a given tumor.

It is clear that even among patients with a single tumor type, there is marked heterogeneity of prognosis and of response to treatment. A better understanding of the biological basis of such heterogeneity may allow more accurate delineation of risk groups. Thus, high risk groups could be identified and given intensified therapy, and lower risk groups would be spared the dangers of such therapy. For example, cytogenetic analysis and analysis of cell cycle phase and ploidy by flow cytometry may provide information relating to assessment of risk.[48]

Definite progress has been made in the treatment of children with brain tumors, and it is likely that this progress will continue. Collaboration between scientists and clinicians, as well as cooperation and interaction among pediatric oncologists, pediatric neurologists, neurosurgeons, and radiotherapists, together with primary care physicians, is essential for the successful management of the child with a brain tumor.

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FROM THE LIBRARY

BRAIN TUMORS IN INFANCY AND CHILDHOOD

A study of brain tumors in infancy and childhood soon discloses that cerebral neoplasms in this age period differ from those of adult life in symptomatology, location, and histologic character. In adult life, headache, mental and personality disturbances, visual impairment, convulsive seizures, speech disturbances, and focal paralytic phenomena among others are outstanding symptoms which lead to a suspicion that the patient may harbor a brain tumor. Occasionally, a focal jacksonian attack or an olfactory or visual hallucination may be the initial symptom of an intracranial expanding lesion. Such symptoms appear infrequently in infancy, although some occur more often in childhood. In infancy, the outstanding symptoms in the presence of an intracranial expanding mass are vomiting and enlargement of the head. In childhood, added to these are headache, failing vision, awkwardness in the use of the upper extremities, and/or unsteadiness of gait.

The variation in symptomatology in infancy and childhood contrasted with adult life can be explained by the differences in the developing skull and brain compared to those of adult life. In infancy the open fontanelles and ununited sutures allow the head to increase in size rapidly to accommodate a growing tumor or enlarging ventricles due to obstruction of cerebrospinal fluid pathways. Signs of increased intracranial pressure are thus often delayed until the lesion has been present for a relatively long period and has assumed an enormous size. For example, in an infant two months of age symptoms were present for only two days. Postmortem done six weeks after the onset of symptoms disclosed a huge pineal tumor.

After the tenth or eleventh year the sutures become more firmly united, and the skull becomes much like a closed box so that new growths very often are manifest early by signs of increased intracranial pressure, in many cases with few or no signs of focal neurologic disturbance. Neurologic examination in infants is more or less restricted to tests which do not require the intelligent cooperation of the patient. The general behavior, size of the head, motor activity, and condition of the pupils and fundi furnish the clues which may lead to a diagnosis of brain tumor. Examination of the visual fields and sensation is necessarily limited. In older children, with care and infinite patience it is frequently possible to carry out detailed visual field and sensory examinations.

Of the laboratory aids in the diagnosis of brain tumors in infancy and childhood, x-ray examinations alone or with the use of contrast media furnish the most valuable information. Enlargement of the head, separation or premature closure of the sutures, and areas of abnormal intracranial calcification are readily recognized. Ventriculography and encephalography provide information regarding the size, shape, and position of the cerebral ventricles, aqueduct of Sylvius, and the subarachnoid cisterns. In the presence of a history or clinical evidence of increased intracranial pressure, ventriculography should always be followed by immediate surgical exploration and removal of the tumor if possible. Ventricular drainage for two or three days following operative intervention has materially reduced postoperative morbidity and mortality.

Cerebral angiography and electroencephalography are employed in the preoperative investigation of many infants and children suspected of having cerebral neoplasms. Both methods often yield significant information, but so far ventriculography remains the most important diagnostic procedure in infancy and childhood.

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(*NY State J Med* 1950; 50:2267–2269)

Disseminated infection caused by *Fusarium solani* in a patient with aplastic anemia

ARIF S. EL-ANI, PHD

Fusarium **species are** common contaminants and ubiquitous soil saprobes that cause worldwide plant diseases. Although long known as causes of keratomycosis, skin wounds, and nail infections, it is only recently that they have emerged as etiologic agents of serious disseminated infections in the compromised host. This report details the clinical course of a patient with aplastic anemia who had a fatal disseminated infection caused by *Fusarium solani*. Therapy for fusariosis is marred by controversial results. For reasons discussed, there is a poor correlation between the results of tests for antifungal susceptibility in vitro and the outcome of clinical therapy.

CASE REPORT

A 46-year-old man who exhibited anorexia, a skin rash, and weight loss, and who had experienced an episode of chest pain and syncope, was admitted to Buffalo General Hospital, where a diagnosis of aplastic anemia was made. He was transferred to Roswell Park Cancer Institute for an allogeneic bone marrow transplant.

On admission to Roswell Park, laboratory tests revealed a white blood cell count of 1,600/mm^3 with 16% blasts, 8% eosinophils, and 76% monocytes; platelets, 27,000/mm^3; and reticulocyte, hemoglobin, and hematocrit levels of 2%, 9.1%, and 26.7%, respectively. The chest film was unremarkable, and blood, urine, and throat cultures were negative. Serum protein electrophoresis was within normal limits, and a bone marrow biopsy showed aplasia.

The patient received high/dose cyclophosphamide and total nodal irradiation before undergoing an allogeneic bone marrow transplant from his brother. He subsequently became febrile and was treated with vancomycin, tobramycin, and carbenicillin. Six days after transplant, erythematous rashes on the face, trunk, and extremities developed. Biopsies of the skin lesions revealed a fungal pathogen that was thought to be *Candida*, and amphotericin B therapy was started. The patient received 15 mg daily for two days, 32 mg daily for seven days, 32 mg every other day for seven days, and finally 55 mg daily for four days. Administration of amphotericin B was stopped because of renal toxicity after a total dosage of 602 mg had been given.

A chest film showed interstitial pneumonia. An open lung biopsy was performed that revealed interstitial fibrosis, but cultures of the biopsy specimens grew no organisms. However, swabs of the skin sites grew *Fusarium*. The patient was given 500 mg ketoconazole for one day through a nasogastric tube. His clinical course following the open lung biopsy was marked by progressive renal and respiratory failure, worsening liver function, and persistent skin lesions. He died one month after receiving the allogeneic bone marrow transplant.

Autopsy Findings. An autopsy revealed disseminated fungal infection involving the lungs, kidneys, spleen, lymph nodes, testes, and skin. Extensive necrosis observed in each of these organs was associated with broad, septate, dichotomously branched hyphae (Fig 1), which are indistinguishable from hyphae produced in vivo by *Aspergillus* species. Postmortem cultures of the lungs, spleen, and kidneys yielded *F solani*. The fungus showed a marked predilection for blood vessels, resulting in widespread thrombosis and tissue necrosis.

Mycology. *F solani* grew rapidly on Difco potato-dextrose agar (PDA) and on Sabouraud's dextrose agar (SDA) at room temperature and 37°C, producing a colony 3.0-cm in diameter in four days. On SDA, the colony was floccose, purple to violet, and the reverse was deep violet. On PDA, the colony was brown and blue and produced a few buff to yellow sporodochia. Mycelial variants without color and sporodochia were often encountered either as mutant sectors in growing colonies or as single conidium cultures isolated from the wild type.

Microscopic examination revealed abundant fusoid, three-septate macroconidia, 23.0 to 40.0 × 5.0 to 7.0 μm, the foot cell of which was marked by a heel-like base. The macroconidiophore was simple or branched; each branch terminated in a single phialide. The microconidia were ellipsoid to allantoid, one- to two-celled, 6.0 to 17.0 × 3.0 to 5.0 μm, formed from simple, elongated lateral phialides or from branched conidiophores bearing terminal phialides. Because the macroconidia in this particular strain were relatively small, and the basal cell often lacked a distinct heel, there was a considerable amount of overlap between macroconidia and microconidia. The chlamydospores were thick-walled, globose, terminal or intercalary, 7.0 to 10.0 μm in diameter, and formed singly or in pairs with condensed protoplasm. Similar chlamydospores were formed by the transformation of one, rarely two, of the macroconidial cells. These chlamydospores were terminal or intercalary and measured 6.0 to 9.0 μm.

Based on the above characteristics, the fungus was identified as *F solani*, according to the taxonomic system described by Booth[1] and the more recent system by Nelson et al.[2]

DISCUSSION

Fusarium species are common soil inhabitants and plant pathogens worldwide. Their isolation from the air has demonstrated the role played by wind and rain in their dissemination.[3,4] Therefore, the epidemiology of fusariosis should be viewed on the basis that *Fusarium* is not only a soil fungus but is

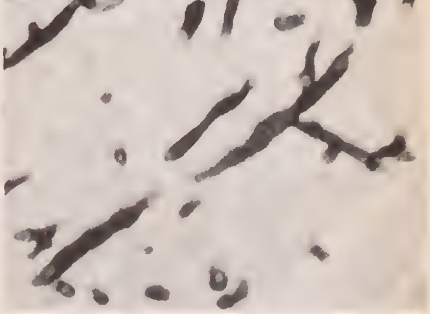

FIGURE 1. Broad, septate branching hyphae in lung (methenamine silver stain; original magnification × 900).

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also an airborne opportunistic human pathogen. The genus *Fusarium* can be readily recognized by its fusoid and septate macroconidia. Each has a notched basal cell. On the other hand, species identification is a difficult task, complicated by great variability[5] and controversial species concepts that have led to a number of diverse taxonomic systems.[1,2]

Of the large, yet indefinite, number of species included in the genus *Fusarium*, only a few have been implicated in a variety of human mycoses, ranging from skin and toenail infections to keratomycosis.[6-8] Disseminated infection is rare and is invariably confined to the immunocompromised host. Species of *Fusarium* that have been isolated from systemic infections are *F solani*,[9,10] *F oxysporum*,[11-14] *F moniliforme*,[15-16] *F chlamydosporum*,[17] *F proliferatum*,[18] and *F anthophilum*.[19]

As pointed out previously, *Fusarium* and *Aspergillus* are indistinguishable in vivo; both produce broad, septate, branched hyphae within the same width range. Both organisms also share an affinity for vascular invasion, causing thrombosis and tissue necrosis.[16,20] However, despite the above similarities, there are striking differences between the two infections. *Fusarium* infections are frequently associated with widespread skin lesions, which are uncommon in most cases of disseminated aspergillosis.[18] Indeed, Anaissie et al[20] reported that 13 of 17 reported patients with disseminated fusarial infection developed skin lesions.

Another important difference is the high rate of isolation of *Fusarium* species from the blood, in contrast to the rare detection of *Aspergillus* species in the bloodstream of patients with disseminated aspergillosis.[18-20] This striking difference between these two opportunistic infections has been the subject of speculative explanations. One explanation proposed by Merz et al[21] is that toxins produced by *Fusarium* species may be responsible for barrier breakdown and thus enhance bloodstream invasion. A different interpretation advanced by Richardson et al[18] stipulates that *Fusarium* species, including *F proliferatum*, are capable of producing abundant dispersive conidia in submerged culture, a characteristic shared by yeasts but not by most pathogenic *Aspergillus* species. Indeed, *Fusarium* is a dimorphic fungus, which under certain conditions grows as a unicellular yeast and produces abundant blastoconidia.[22]

The literature on the treatment of systemic fusariosis abounds with conflicting results.[17] Using a broth microdilution method for determining antifungal susceptibility of 44 clinical isolates of *Fusarium* species, Reuben et al[23] concluded that amphotericin B and natamycin were the most effective agents, and flucytosine had no activity against any of the isolates. However, the correlation between in vitro antifungal susceptibility status and the outcome of therapy has been disappointing.[18] The outcome of any therapy varies with the different underlying diseases, their impacts on the defense system, and the extent of fungal dissemination.[18,20] These factors may explain the failure of treatment with amphotericin B and ketoconazole in the present case.

Acknowledgments. Dr Paul E. Nelson, Department of Plant Pathology, Pennsylvania State University, University Park, Pa, confirmed the identification of *F solani*. Histologic sections were provided by the Department of Pathology at Roswell Park Cancer Institute.

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Carcinoma of the lung presenting with spontaneous pneumothorax

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Spontaneous pneumothorax is usually caused by a benign lesion, such as rupture of a subpleural bleb or an emphysematous bulla. However, it may complicate any underlying lung disease, especially those associated with fibrotic or cystic changes, such as emphysema, tuberculosis, sarcoidosis, and asbestosis.[1] Malignancy is a rare cause of pneumothorax and may be primary or metastatic. In children and adolescents, lung metastases from sarcomas that cause pneumothorax have been well described.[2] Spontaneous pneumothorax as a presenting feature of a bronchogenic carcinoma is extremely rare.[3] We present a report of one such case.

CASE REPORT

A 55-year-old man presented with sudden onset of dyspnea. He had had no previous episodes of a similar nature. His medical history was unremarkable except that he was a chronic smoker. A chest radiograph revealed a right pneumothorax. Laboratory studies, including a complete blood count and SMA-18, were within normal limits.

A size 32F chest tube was placed in the sixth intercostal space in the midaxillary line. The pleurevac was connected to wall suction; despite this, the lung failed to expand (Fig 1). A second chest tube was placed anteriorly in the second intercostal space; again the lung failed to expand.

After about ten days of conservative treatment, a right thoracotomy was performed. Air leakage was observed from the surface of the upper lobe, and an indurated area of approximately 1 × 1 cm was felt around the site of the air leak. A biopsy and frozen section of this lesion was reported as epidermoid carcinoma. A right upper lobectomy was performed. Histologic evaluation confirmed the presence of a well-differentiated squamous cell carcinoma. The regional lymph nodes were not involved. The patient made an uneventful recovery. One year later, the patient was doing well and was free of disease.

DISCUSSION

Although lung cancer is the most common malignancy in the United States, its association with spontaneous pneumothorax is quite rare. Hyde and Hyde[4] were able to find only one out of 3,000 cases of bronchogenic carcinoma presenting with spontaneous pneumothorax. Four of 1,143 patients with lung cancer seen at the Mayo clinic had a coexistent pneumothorax, an incidence of 0.35%.[2] A slightly higher incidence has been reported by others.[5]

Heimlich and Rubin[6] described the first two cases of primary carcinoma presenting as spontaneous pneumothorax. In 50% to 75% of cases in which there is an association of lung cancer and pneumothorax, spontaneous pneumothorax is the presenting symptom, while in the remaining patients, pneumothorax occurs as a late symptom following the diagnosis of carcinoma.[5,7] The condition is more common in males over 40 years of age; only three of 46 cases reported by Steinhauslin and Cuttat[5] were under 40 years. As expected, there is high association with smoking. The right side appears to be more commonly involved than the left.[5]

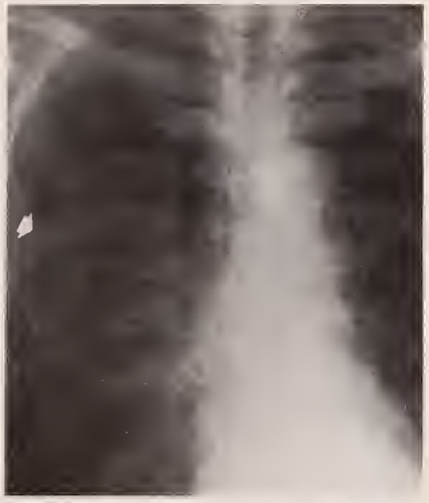

FIGURE 1. Anteroposterior chest roentgenograph following the insertion of the first chest tube. Note persistent pneumothorax. The arrow points to the lung border.

The exact mechanism by which spontaneous pneumothorax arises is not clear, and more than one mechanism may be involved.[5] The most common explanation is erosion of tumor onto the visceral pleura resulting in a bronchopleural fistula. Spontaneous pneumothorax may also result from rupture of dilated alveoli distal to a stenotic bronchial lesion. Lastly, carcinoma may coexist with an underlying lung disorder, and may cause pneumothorax either by directly involving the bulla or by altering the architecture leading to rupture of the bulla.[5]

The initial diagnosis of carcinoma may commonly be missed, especially if there are no other associated symptoms. This is also likely to happen if the lung re-expands after insertion of a chest tube, and the subsequent discharge of the patient from the hospital. Later readmission with recurrence in an advanced stage is inevitable. Squamous cell carcinoma is the pathologic disorder in the majority of patients presenting with this complication.[5,7] Other cell types also occur and include adenocarcinoma, giant cell carcinoma, alveolar cell, and oat cell and undifferentiated carcinoma.

Primary lung cancers were treated in about 70% of reported cases associated with spontaneous pneumothorax[5,7]; treatments included surgery, radiotherapy, and chemotherapy, alone or in combination. The overall survival is poor, with 17% of patients living up to one year. The average survival is 5.2 months.[5] No five-year survivors have been reported. This holds true even for T1 tumors, which suggests that lung cancer presenting as a pneumothorax is at an advanced stage.

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A large pericardial effusion in a patient with human immunodeficiency virus type 1

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Pericardial effusion frequently occurs in patients with infection, uremia, malignancy, trauma, myocardial infarction, collagen vascular disease, and many other conditions.[1] In patients with acquired immunodeficiency syndrome (AIDS), pericardial effusion is a common finding, but the volume of the effusion is usually small.[2] We describe a patient who was seropositive for human immunodeficiency virus (HIV-1) with a 6.4-liter pericardial effusion.

CASE REPORT

A 32-year-old man presented to the emergency room with a two-month history of dyspnea, fever, chills, and productive cough. His medical history was noncontributory except for intravenous drug use. Physical examination revealed a thin young man in moderate respiratory distress. His temperature was 99°F orally; heart rate, 106/min; respiratory rate, 32/min; and blood pressure, 105/80 mm Hg with a paradoxical pulsus of 8 mm Hg. There was jugular venous distention at 45 degrees. Chest examination revealed no rales or rhonchi. The heart sounds were distant without a friction rub, and the abdominal examination was normal. There was no peripheral edema, and the neurologic examination was normal.

The electrocardiogram revealed a sinus tachycardia with generalized low voltage. The 2-D and M-mode echocardiogram revealed a huge pericardial effusion with a "swinging heart" pattern and evidence of diastolic collapse of the right ventricle. A bedside pericardiocentesis was performed, and about 500 mL of straw-colored fluid was removed. An indwelling catheter was inserted and about 5 liters of fluid was drained during the next 12 hours. A total of 6.4 liters of fluid was removed in 48 hours.

Laboratory studies of the pericardial fluid revealed a white blood cell count of $2/mm^3$ and a red blood cell count of $960/mm^3$; protein content, 5.3 g/L; glucose, 82 mg/dL; and lactate dehydrogenase, 108 U/L. Bacteria, fungi, virus, and mycobacteria cultures were all negative. Skin tests for tuberculosis, mumps, and *Candida* were all nonreactive. Serum antinuclear antibodies were negative. An enzyme-linked immunosorbent assay and a Western blot confirmation test for antibodies to HIV-1 were positive.

The patient was well until four weeks later, when a repeat echocardiogram showed recurrence of the pericardial effusion. The patient then underwent a pericardectomy. Histologic examination revealed mild, chronic inflammatory change of the pericardium. A repeat chest film showed marked regression of the heart size (Figs 1,2). The patient was discharged with no further complications.

DISCUSSION

Pericardial effusion involving the heart has been described in HIV-1 infected patients. The causes usually include tuberculosis, Kaposi's sarcoma, congestive cardiomyopathy, or fibrinous pericarditis.[3,4] In the patient described here, there was no evidence of opportunistic infection; all the studies for bacteria, mycobacteria, fungi, or virus were negative. Histologic evaluation revealed only chronic inflammation. The cause of the huge pericardial effusion is unknown.

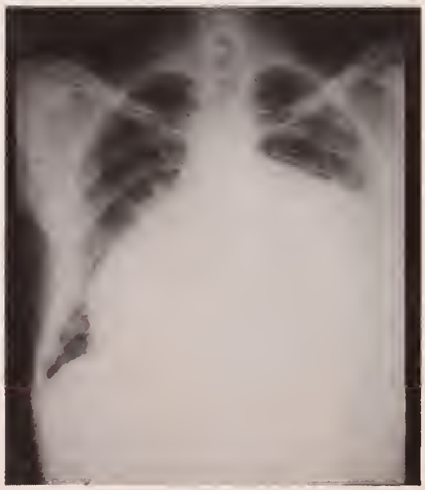
FIGURE 1. Chest film on admission.

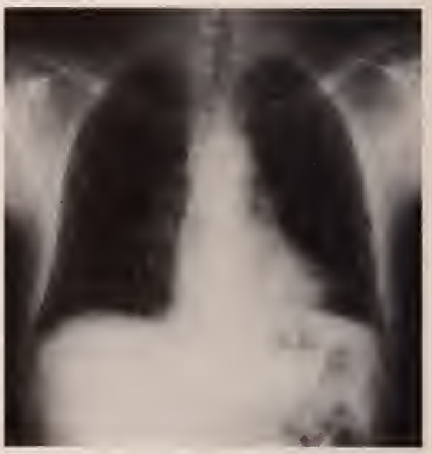
FIGURE 2. Chest film on discharge.

The pathogenesis of development of this huge pericardial effusion could involve an inflammatory process of the pericardium, as suggested by the exudative character of the pericardial fluid. As a consequence, the colloid oncotic pressure in the pericardium would have increased, and more fluid would have accumulated. Also, lymphatic drainage might have been impaired as a result of inflammation involving the lymphatic vessels.[5]

The finding of 6.4 liters of pericardial fluid is surprising. The combination of pericardiocentesis and pericardectomy resulted in complete resolution of the patient's symptoms. It is important that physicians be aware of this potentially treatable problem in HIV-1 infected patients.

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Upper airway obstruction due to Kaposi's sarcoma in the acquired immunodeficiency syndrome

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At the outset of the epidemic of acquired immunodeficiency syndrome (AIDS) in the early 1980s, Kaposi's sarcoma was seen in about 35% of AIDS patients,[1] with clinical pulmonary involvement evident in 20% to 30%.[2] Since then, the reported frequency of Kaposi's sarcoma in AIDS has declined and was about 15% in 1989.[3] The frequency of pulmonary involvement with Kaposi's sarcoma at autopsy is greater than that recognized during clinical evaluation because of difficulty in antemortem diagnosis.[2] Lesions in the upper airways are not uncommon. However, upper airway obstruction has only occasionally been reported.[4,6,7] The patient described here lacked cutaneous involvement, however, acute upper airway obstruction developed due to laryngeal and subglottic lesions of Kaposi's sarcoma.

CASE REPORT

A 28-year-old Haitian-born man with a history of intravenous drug abuse and homosexuality was admitted to the hospital three times over a seven-month period for intermittent fever, cough, dyspnea, and weight loss. Chest radiographs showed progressively increasing, poorly defined, bilateral nodular infiltrates. Diagnostic studies included fiberoptic bronchoscopy, which showed normal airways; bronchoalveolar lavage was negative for pathogens and malignant cells. The patient did not respond to empiric trials of antibiotics including trimethoprim-sulfamethoxazole or to anti-tuberculous chemotherapy.

On his fourth and final admission his symptoms included fever, diarrhea, and severe prostration. The chest radiographs (Fig 1) showed bilateral, patchy, poorly defined nodular infiltrates. In retrospect, the tapering of the tracheal air column at the glottis appeared increased. Sputum and blood cultures grew *Escherichia coli*. The patient was treated with cefotaxime and gentamicin, but spiking fevers continued. Severe respiratory distress developed accompanied by hoarseness and wheezing. The fiberoptic bronchoscope was used to examine the upper airway. Violaceous, fleshy lesions involved the posterior aspects of the true vocal cords, which were fixed in adduction with a glottic aperture of about 5 mm. The subglottic area was narrowed to a slit-like opening. Computerized tomography confirmed severe narrowing at and just below the glottis (Fig 2). The trachea was patent distal to the subglottic area. Urgent tracheostomy was performed with marked improvement in dyspnea.

Two days later, bronchoscopy was done via the transnasal approach. The lesions in the subglottic region were biopsied without incident. The bronchoscope was then introduced via the tracheostomy. The distal trachea and remainder of the bronchial tree showed numerous discrete, raised erythematous lesions. Bronchoalveolar lavage and transbronchial biopsy were done without incident. With the first biopsy of one of the erythematous lesions in the right lower lobe there was copious bleeding, which eventually totalled about 500 mL. The bleeding subsided with vigorous suctioning and instillation of aliquots of epinephrine in a 1:20,000 dilution via the bronchoscope. Coagulation profile, platelet count, and renal function had been normal prior to the procedure.

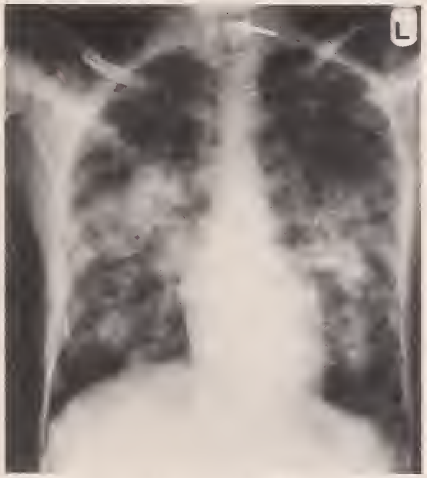

FIGURE 1. Chest radiograph showing poorly defined nodular densities. There appears to be exaggeration of the normal tapering of the tracheal air column at the glottis (arrow).

Pneumocystis carinii infection was diagnosed from the bronchoalveolar lavage. The biopsies from the subglottic area were consistent with Kaposi's sarcoma. The endobronchial and transbronchial biopsies were nondiagnostic. The patient had no mucocutaneous lesions due to Kaposi's sarcoma.

Trimethoprim-sulfamethoxazole therapy was started. Radiotherapy was planned for treatment of the laryngeal and subglottic lesions. However, respiratory failure developed, requiring mechanical ventilation, and the patient died.

At postmortem, extensive involvement with Kaposi's sarcoma was present in the larynx, tracheobronchial tree, and pulmonary parenchyma. Widespread pneumonia due to *Pneumocystis carinii* was also present. Cultures of fluid from both pleural spaces grew *Escherichia coli*, and *Streptococcus faecalis* was cultured from a minimal

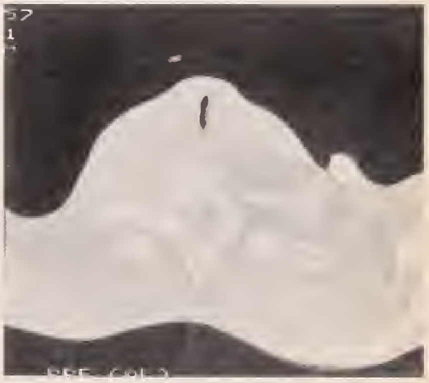

FIGURE 2. Computerized axial tomograph showing narrowing of the airway at the level of the glottis.

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pericardial effusion. Findings in the brain were consistent with AIDS encephalopathy.

Discussion

Upper airway obstruction due to Kaposi's sarcoma is a life threatening but potentially treatable condition. There have been a few previous reports in patients with AIDS. Zibrak et al[6] reported two patients with upper airway obstruction due to laryngeal and tracheal lesions; the patients' conditions improved with radiation and vincristine therapy. In the series of Meduri et al,[4] one of 11 patients with pulmonary Kaposi's sarcoma had stridor from laryngeal and tracheal obstruction. Greenberg et al[7] reported a patient with obstruction at the pharyngeal level. In the patient described here, lesions of Kaposi's sarcoma were present in the larynx and throughout the tracheobronchial tree. Extensive involvement of the vocal cords and in the subglottic region led to marked narrowing of the airway, which was relieved by tracheostomy.

In addition to its morbid effects, upper airway obstruction may also be significant as a clue to the diagnosis of pulmonary involvement with Kaposi's sarcoma.[4] In some patients, hemoptysis, hilar adenopathy, or hemorrhagic pleural effusions suggest the diagnosis of AIDS-associated pulmonary Kaposi's sarcoma. More often, the clinical and radiographic abnormalities are nonspecific.[1,2] In part, this is due to the high incidence of coexisting pulmonary pathology, especially opportunistic infection.[8] Findings of hoarseness or stridor should lead to consideration of this entity even in the absence of skin lesions, as was the case in this patient.

Radiotherapy, which has been a primary form of treatment in classic Kaposi's sarcoma, is probably the most appropriate initial mode of therapy for upper airway obstruction due to Kaposi's sarcoma in AIDS.[9] Cooper et al[10] demonstrated fairly rapid improvement of cutaneous and mucosal lesions in patients with AIDS using radiation doses in the range of 1,800 to 3,000 rads. Success with a short course of radiotherapy was reported by Patow et al[11] in a patient with dysphagia, gagging, and inanition due to pharyngeal obstruction from Kaposi's sarcoma.

Chemotherapeutic agents and interferons have been used for the treatment of Kaposi's sarcoma in patients with AIDS. These modalities are unlikely to work as rapidly as radiation to relieve upper airway obstruction, and chemotherapy carries the risk of myelosuppression. Experience with laser therapy in classic Kaposi's sarcoma suggests that this may be of value for the treatment of upper airway obstruction due to AIDS-associated Kaposi's sarcoma.[12] Local extirpation may also be possible in some cases.[13]

Our experience with this patient is pertinent to another issue concerning pulmonary Kaposi's sarcoma, namely the role of bronchoscopy and bronchoscopic biopsy for diagnosis. Visualization of circular or oval, red or purplish lesions in the airways permits a tentative diagnosis.[2] The utility and safety of biopsy remains controversial. In most reports, endobronchial and transbronchial biopsies have generally been nondiagnostic.[1,2,4,8] The small size of biopsy specimens obtained with the fiberoptic bronchoscope and the lack of specific cytologic markers make the diagnosis difficult. Moreover, crush artifact and hemorrhage may mimic the histologic features of Kaposi's sarcoma.[1] Hamm et al,[14] however, obtained diagnostic endobronchial biopsies in five of seven patients who had characteristic lesions in the airways. In the case presented here, biopsies from the subglottic area were consistent with Kaposi's sarcoma, but the endobronchial and transbronchial biopsies were nondiagnostic.

Because of the vascular nature of the tumor, the safety of bronchoscopic biopsy has been debated. Significant bleeding did not occur in 17 patients reported by Garay et al,[2] or in the seven patients reported by Hamm et al.[14] However, this patient bled profusely, and there have been previous reports of excessive bleeding after both endobronchial[15] and transbronchial biopsy.[4] A fatal hemorrhage occurred during tracheostomy for upper airway obstruction due to Kaposi's sarcoma involving the oropharynx in a patient who also had lesions in the trachea.[7]

Despite the low yield and potential dangers of biopsy, bronchoscopy remains an important diagnostic tool in suspected pulmonary Kaposi's sarcoma. The presence of characteristic lesions in the tracheobronchial tree strongly suggests the diagnosis, and bronchoalveolar lavage is a sensitive method for detection of coexistent pulmonary infection, which is often present,[8] as in this patient.

Acknowledgment. The authors thank Kathy Dwenger for secretarial assistance.

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LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

New *Journal* cover

TO THE EDITOR: I am responding as requested in your commentary.[1] Overall, my response is extremely positive and you and the subcommittee of Drs Bonnani, Cohn, and Muratore are to be congratulated. I especially appreciate the following: the new cover design (may it outlast many of its predecessors), the new table of contents, and the new typeface (Helvetica) for the cover and table of contents. The new pagination combined with footers containing the volume on even pages and month and year on odd pages is very professional and helpful. My only constructive criticism would be to enlarge the size of the seal of the Medical Society of the State of New York—this would be possible without disturbing the "clean look" of the new cover. It is my hope that the new stylistic changes will continue to be combined with the *Journal's* high quality content.[2]

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1. Imperato PJ: A new cover for the *New York State Journal of Medicine. NY State J Med* 1990; 90:345.
2. Evans JT, Nadjari HI, Burchell SA: Quotational and reference accuracy in surgical journals: A continuing peer review problem. *JAMA* 1990; 263:1353–1354.

TO THE EDITOR: I like the new design, format, and related changes in the *Journal*. The changes add aesthetic appeal while also lending increased dignity. Altogether a classy move. Congratulations!

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Premenstrual syndrome

TO THE EDITOR: We are writing with reference to the recent review article by Ellen Yankauskas, MD, entitled "Primary female syndromes: An update."[1] Overall, the review is timely and well-written, yet its section on premenstrual syndrome (PMS) suffers from several serious misconceptions.

First, the subsyndromal classification system promoted by Abraham and colleagues[2,3] has no substantive etiological support, and the nutritional deficiency theories that accompany their classification system have not been supported when carefully examined.[4,5]

Second, a diagnostic evaluation for PMS should consist of the following: a complete physical examination (as is stated in the article), a psychological examination to rule out major psychopathology, and at least two months of *prospective* daily symptom ratings. Women with major affective illness or other Axis I psychopathology should not receive a diagnosis of PMS unless their premenstrual symptoms as shown on their daily symptom ratings are distinct from those of the preexisting psychiatric disorder. The daily ratings are critically important, since retrospective histories alone inflate the incidence of PMS.[6] Further, late luteal phase dysphoric disorder (LLPDD) is not "the appropriate appellation for the psychological manifestations of PMS." Rather, this is a research diagnosis restricted to a small subgroup of women with PMS who have severe and incapacitating affective symptoms.

Third, the description of treatments was inadequate. It should be made clear to women suffering from PMS that the simple dietary changes that were recommended in this review and in the popular press are not supported by empirical data from well-designed studies. In double blind controlled studies, vitamins B_6 and E have not been shown conclusively to be effective.[7,8] The expanded table of treatments did not indicate to the reader which treatment modalities can be recommended on the basis of solid research that was carefully controlled regarding both diagnostic evaluation and the well-described placebo response. The use of progesterone, for example, cannot be supported.[9-11] Many of the drugs listed have never been tested in controlled clinical trials of PMS (eg, pamabrom, pyrilamine, naproxen, ibuprofen, tricyclics, monoamine oxidase inhibitors, propranolol, verapamil, and levothyroxine), and their use should not routinely be advocated.[6] Finally, it should be mentioned that active research is being conducted on antidepressant medications, such as fluoxetine, to treat the affective symptoms of PMS.[12]

Premenstrual syndrome is a serious disorder affecting many women. It is therefore very important to separate the substantive research findings from the majority of weak research in this area, and to educate clinicians accordingly.

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Combining parenthood and practice

TO THE EDITOR: I congratulate Dr Scanlon[1] on her achievement! As a mother of four children who managed with long term household "help" and with a maximum maternity leave of five days, I appreciate what it takes. I have been active in finding better answers for the new generation of women who wish to combine maternity and medicine with less role strain.[2] I have also written about networking problems among female professionals.[3] I suspect that the psychological issues that govern the ability of women to choose supportive women as friends and to maintain relationships with them apply to the selection of long term child care persons, as well as to office nurses, assistants, partners, and colleagues. The ability to relate well, comfortably, noncompetively, and loyally to other women is more dependent on a woman's relationship to her own mother than is her choice of a husband or relationship to male colleagues.

I doubt that Dr Scanlon would disagree with Dr Kaiser[4] that children are our most precious resources. I think that she would agree that Dr Jacob[5] should not have chosen such a demanding specialty, ob-gyn, if she intended to have five children, unless she could afford the life style that Dr Scanlon, I, and many others have given their children.

In the best of all possible lives, a full time medical career and a large family, which is well attended to, requires both tremendous interpersonal skills and vast financial resources. Choices must be made. Yet, I submit that combining maternity and medicine is a viable option.

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1. Scanlon AT: Parenthood and practice. *NY State J Med* 1990; 90:326-327.
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5. Jacob JR: Responses to parenthood and practice. *NY State J Med* 1990; 90:334-335.

TO THE EDITOR: I enjoyed reading my mother's (Dr A. Scanlon) article, "Parenthood and practice," in the June 1990 issue of the *Journal*.[1] I also read with interest the responses it created. Perhaps as an insider I can clarify some of the questions that arose in the letters to the editor by Dr Kaiser[2] and Dr Jacob.[3]

Dr Scanlon's article was her reminiscence of practice and parenthood, not an editorial on child care in today's society. Therefore, I do not feel it is fair to say her remarks did not meet the challenge. The challenge was not the point of the article and therefore not addressed.

Dr Scanlon began her article with, "How lucky can Tom and I be!" She will be the first to admit that she could not have maintained her practice and raised ten children without "Mac." Mac was a valued member of the family until her death in 1971. She was the constant as we went through 12–14 other live-ins and assorted help over 22 years. Mac was with us from 1949 until 1971, and she was my sister Maggie's godmother. I am quite sure my mother could furnish details on how the household was run. In fact we have all encouraged her to write her memoirs, but with the allotted space she could only give a sketch of her reminiscences.

My mother was always available for her practice because her office was at home and she was always at home. Having her office in the house did not deter us from running to her with bumps and cuts during office hours.

Lake Placid was the first and only full family vacation I can recall and it was fun. My parents bought the Vermont home the following winter after agonizing over the cost ($12,000) for some time. The investment paid off because Vermont became a place we could all enjoy during vacation time and school breaks.

Times and economics have changed. My parents were not independently wealthy. In fact they have only recently gotten out from beneath the monumental debt incurred educating ten children.

The continuing medical education (CME) trips did not occur until I was well into high school, which meant everyone was in school at the time and there were extra teenagers at home to help out with the younger children.

My parents rarely took time for themselves. They were either working or spending time with us. Ten children were an economic drain. My mother was lucky she found a good child care giver in Mac. She also luckily married a man like my father and luckily had ten children who only gave her headaches and not heartaches.

In closing, I would be more than happy to give Dr Jacob a description of how I survived early childhood, as I am sure my nine siblings would be happy to give their version.

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1. Scanlon AT: Parenthood and practice. *NY State J Med* 1990; 90:326-327.
2. Kaiser V: Responses to "Parenthood and practice." *NY State J Med* 1990; 90:334.
3. Jacob JR: Responses to "Parenthood and practice." *NY State J Med* 1990; 90:334-335.

TO THE EDITOR: I read with interest the comments of Dr Kaiser[1] and Dr Jacob[2] on my article, "Parenthood and practice."[3] The essential issue for all working mothers is child care. Concern about a stranger having "such influence in raising my children" was hardly an issue as Mrs McCormack worked only part-time while I was in the city for eight months and during this time she knew our son Tom and our wishes on child care.

Dr Kaiser said we need new social structures to care for and protect our children. What we need is commitment to the same values that have endured through the centuries. This includes the commitment to family as the basic unit of society with mother and father in command, nurturing love, strong moral values, and self-discipline in each child, and obtaining a support system when needed that is not in conflict with our ethics regardless of the mores of the day. What difference does it make if we rotate child care with colleagues? This type of rotation would not lend itself to the stability of a constant loving careperson. I feel this was my responsibility and not the responsibility of the medical profession or the community. Certainly a professional woman can afford to find someone and I do know that it takes a lot of looking. I also know that we should try to make "child care" a profession and not treat it as a servant class. It should be in the same category as governesses and teachers.

Dr Jacob would like to have a detailed description on "how it was done." Could one article even begin to touch the subject? I do not deny that some days were hectic and even frantic. But show me a household of only two or three children that does not have days when a mother needs to be three people and also a wife, even when there is no practice with which to contend.

Dr Jacob says that she is greeted by five screaming children and babies. If this is usual, I would reevaluate my child care person. I had the advantage of having my office at home. My husband, if no one was in labor, came home for lunch and so did I. After I had six children I had an au pair from Scotland who joined Mrs McCormack and stayed for seven years. She was married from our home and my husband "played father of the bride."

Economics have changed. We pay our employees more but in turn we charge our patients more. We were not independently wealthy and we re-mortgaged the house and farm to help with education costs. Believe it or not we still have two more years to go until our house is free and clear.

Last but not least, children adjust well in happy situations, where there is structure, love, and approbation, so when stressful times occurred all rallied and met the challenge as they still do today. My joy is seeing children and grandchildren all happy, in productive careers, and those married nurturing their children to follow in their parents' footsteps.

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2. Kaiser V: Responses to "Parenthood and practice." *NY State J Med* 1990; 90:334.
3. Jacob JR: Responses to "Parenthood and practice." *NY State J Med* 1990; 90:334-335.

In reply. The response to my letter is most interesting.[1] I find it more direct and pertinent than Dr Scanlon's first article as it focuses on child care.[2] However, I disagree with her approach to the issue. In terms of Dr Sheehy's remarks, I have few words. As it happens, I too come from a large family of eight siblings. Each of our mothers could tell a few stories, I am sure. But if you are going to talk about parenthood and practice, you cannot avoid the prime concern.

Now to return to Dr Scanlon. There remains no question in my mind that we need new social structures to deal with child care, and this will require group changes at times. For example, when I was a resident, a colleague was pregnant and about to give birth. The program had no way to deal with her absence. They eventually decided to give her leave, but expected her to pay back the calls she missed. This would have meant double calls for three months while caring for an infant. Eventually, the residents independently volunteered and covered her in a team effort without expecting repayment. Could this resident's needs be solved without a change in the system and support of colleagues? Another example is that of two residents who were raising their children alone. Their specific reason for coming to that program was the day care available on campus in the resident housing. A resident cannot afford a care-person. Even after residency, money is hardly the only issue. My idea of rotating colleagues is flawed, as I noted in my original letter. I agree with Dr Scanlon's view of this. However, my greater fear is having another person in my home. Mac worked part-time, but I work 60 hours a week. Even if I cut down, I need someone full-time. Part-time or full-time, any constant person in a child's life would be influential or Dr Scanlon would not object to rotators. Much more influential than the instructions that person is given is his or her personality and values. It often takes years to truly know this about someone. As I wrote before, "Years to decide on the father and a few hours to decide on a major caregiver?" Yes, I do not want a stranger having such an influence raising my children. This is why my husband is going to do the unconventional thing and become the primary caretaker of our home. Were this not the case, I think I would prefer sharing with colleagues I have known for years, despite the limitations of such a plan. At least I would really know who they were. I agree that for those parents who can accept an outsider in the home, the caregiver should be well trained and well paid. This would attract higher caliber individuals. This is one of the social structure changes I feel we need. Contrary to Dr Scanlon's remarks, I believe that our problem as doctor/parents has nothing to do with commitment to family, moral values, love, or self-discipline in children. This makes it sound like doctors have misdirected values rather than valid concerns. Our problem is trying to balance active careers with the full-time requirements of family life. Traditional values mentioned above were accompanied by a full-time mother. If she is removed from the home, a new social system must replace her. An active search for new social solutions includes day care, on-site day care, professional caretakers, new family roles, shared child care with trusted others, and many others, I am sure. A debate such as this may be helpful in finding and refining such attempts. Some solutions require a community effort.

Dr Scanlon enters into this debate with her ideas, which are at times quite cogent. But there is no continuity in her remarks. Dr Scanlon believes we do not need new social structures, but we do need "support systems." Is there a difference? She does not feel Mac had an excess influence on her children, but fears what would happen without a "constant, loving careperson," so Mac must have had a strong influence. Dr Scanlon describes the need for a mother and father to be "in command," but later she frames child care as something sought by "professional women" instead of couples. I feel society can no longer afford such distinctions. Dr Scanlon felt the child care responsibilities were hers and not the community's, but then suggests society should help by fostering a child care profession. Since child care workers are currently seen as part of a servant class, finding a competent, psychologically healthy person with similar values takes a lot more than looking. It takes a large measure of luck. I guess Dr Scanlon was lucky.

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2. Scanlon AT: Parenthood and practice. *NY State J Med* 1990; 90:326-327.

In reply. It was a pleasure reading Dr Sheehy's honest addendum to Dr Scanlon's "Parenthood and practice."[1] I suppose it is always anxiety provoking to read about the harmony of someone else's household when one's own home reverberates constantly with the normal sounds of five children who play and fight by turns. This anxiety manifests itself in a defensiveness when one reads a fond memoir which omits the sordid details of day to day life and focuses on the best aspects.

Actually Dr Scanlon's memoirs are heartening and encouraging, and I only pray that in 20 years my children will also recount with such pride and joy their upbringing. I, too, have no regrets about the life I chose . . . and I am expecting my sixth source of joy in April! The companionship my children provide each other when I am gone and the gaiety of group play when it does occur are frequent reminders that I did the right thing for my family.

I do agree entirely with Dr Scanlon's astute comments that childcare is primarily the parents' responsibility and that no hired help can or should be expected to provide the guardianship a child needs from day to day. Fortunately, though, I am an obstetrician/gynecologist in private practice. I work in a group where five of us rotate calls, so I am home most evenings to make dinner and do homework, and home most mornings to make corn muffins and prepare lunch.

If I ever do have a crisis maybe I will call Dr Sheehy or Dr Scanlon for some advice!

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Fibrous disease of the breast in juvenile diabetes

TO THE EDITOR: We are pleased to read Gump and McDermott's article, "Fibrous disease of the breast in juvenile diabetes,"[1] because it constitutes the fourth documentation of the link between long-standing insulin-dependent diabetes mellitus (IDDM) and benign breast masses that clinically resemble cancer.

Gump and McDermott's report on their 11 patients with this entity mentions Soler and Khardouri's work in 1984[2] and that of Byrd and his group in 1987,[3] but omits our recent discussion in *Radiology*[4] of 36 patients, from 20 to 54 years of age (median age, 37 years), with this little-known condition that we called diabetic fibrous breast disease (DFBD).

As we noted in our article, the patient must fulfill stringent criteria for the radiologist to consider a possible diagnosis of DFBD. These include a prolonged history of insulin-dependent diabetes mellitus (IDDM); mammographically dense glandular tissue; one or more exceedingly hard, irregular, freely movable, discrete, painless, clinically palpable breast masses with neither skin change nor distortion of the breast contour. Sonographically, these masses reveal strong acoustical shadowing and are firmly resistant to the to-and-fro movement of the needle used for fine-needle-aspiration cytology (FNAC).

By monitoring the patients who meet all these criteria with serial FNACs, we have found that we could spare certain women with IDDM the ordeal of undergoing multiple unnecessary biopsies. During an average of seven years of monitoring these 36 women, not one has developed breast cancer in a mass fulfilling all our requirements for diabetic fibrous breast disease.

As we observed in our article,[2] diabetes confers no immunity from breast cancer. Nevertheless, if radiologists, surgeons, and internists are aware of the constellation of findings produced by diabetic fibrous breast disease, those women with IDDM who meet all our requirements may be able to avoid the physical, psychological, and financial costs of unwarranted biopsies.

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2. Soler NG, Khardori R: Fibrous disease of the breast, thyroiditis, and cheiroarthropathy in type I diabetes mellitus. *Lancet* 1984; 1:193-195.
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In reply. We are pleased to find additional recognition of the association between juvenile diabetes and benign breast masses. Our failure to mention the authors' contribution, however, simply reflects the fact that it appeared after our paper was accepted for publication in the *New York State Journal of Medicine.*

We share the authors' view that these patients can be followed by careful clinical examination and repeated fine needle aspiration cytology. Like them, we believe that increased awareness of the association between type I diabetes and hard breast masses will reduce the need for breast biopsies in these young women.

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ERRATUM

The Special Report published in the August 1990 issue of the *Journal* erroneously included *Nation's Business* among the list of magazines that do not accept tobacco advertising.

LEADS FROM EPIDEMIOLOGY NOTES

Reprinted from the August 1990 issue of Epidemiology Notes (*Vol. 5, No. 8*), *published by the Division of Epidemiology, New York State Department of Health, Albany, NY.*

Birth outcomes in New York State

This article updates the information contained in the August and September 1989 issues of Epidemiology Notes (*Vol. 4, Nos. 8 and 9*).

Measures of infant morbidity and mortality are used by health professionals as indicators of the health of society as a whole. Traditionally, the public health sector is at the forefront in the effort to promote desirable birth outcomes. This article is intended to provide maternal and child health service providers and governmental agencies information that will be useful in monitoring and evaluating existing programs and planning for efficient and effective application of available resources. It contains information on patterns of adverse birth outcome in New York State.

Birth outcomes were examined over time and included: fetal, neonatal, postneonatal and infant mortality; low birthweight and prematurity; congenital defects; frequency of miscarriages and abortions; and selected neonatal diagnosis related groups (DRGs). In addition, sociodemographic variables associated with birth outcomes such as maternal age, race/ethnicity, education and use of prenatal care were also reviewed. These analyses are descriptive in nature and cannot fully explain the interrelationships between the sociodemographic factors and birth outcomes.

There were 279,976 births to New York State residents in 1988. Approximately 46% of births occurred in New York City; 27% of births were to nonwhite residents of New York State. In addition, there were 136,012 induced abortions and 2,316 spontaneous fetal deaths (20+ weeks gestation) reported among New York State residents in 1988. An estimated 4,326,015 women aged 15–44 in New York State in 1988 provided a fertility rate (live births/women 15–44) of 64.4/1,000.

Fetal Mortality

The rate of spontaneous fetal deaths (20+ weeks gestation) has decreased within the last decade. The 20.4% reduction from 10.3 per 1,000 live births plus fetal deaths in 1978 to 8.2 per 1,000 in 1988 was evident in both the New York State and New York City populations, with New York City consistently having a higher fetal mortality rate. The nonwhite spontaneous fetal death rate (11.2 per 1,000) was 41.1% higher than the white rate (6.6 per 1,000) in 1988. The statewide rate dropped between 1987 and 1988 from 8.9 to 8.2 per 1,000.

Infant Mortality

The 1988 New York State infant mortality rate of 10.7 per 1,000 live births was unchanged from 1987 and indicates a leveling off of infant mortality after a near decade of continuous decline. This rate was somewhat higher than the provisional national rate of 9.9 per 1,000. New York City residents had an infant mortality rate (13.2 per 1,000) 51.7% higher than residents in the rest of the state (8.7 per 1,000) (Fig 1).

The gap between white and nonwhite mortality has not substantially changed in the past decade. Nonwhite residents had a 1988 infant mortality rate (14.7 per 1,000) 58% higher than their white counterparts (9.3 per 1,000). Hispanics had the lowest infant mortality rate in 1988 (7.4 per 1,000) compared to non-Hispanic white (8.2 per 1,000) and non-Hispanic black (18.6 per 1,000) births.

The general reduction in infant mortality experienced over the past ten years is primarily due to a drop in the neonatal mortality rate (deaths within less than 28 days of birth). The postneonatal mortality (deaths 28 days to one year) decreased only 15.4% from 3.9 per 1,000 in 1978 to 3.3 per 1,000 in 1988.

Cause of Death

In 1988, there were 3,007 infant deaths to New York State residents. Of these deaths, 69.1% (2,078) occurred during the neonatal period; 30.9% (929) were postneonatal deaths. Of the neonatal deaths, 20.6% were due to congenital anomalies (ICD 740–759), 20.3% due to short gestation-low birthweight (ICD 769) and 36.8% to other conditions originating in the perinatal period (ICD 760–764, 766–768, 770–779). The leading cause of postneonatal deaths was sudden infant death syndrome (ICD 798) comprising 23.4% of the total, followed by congenital anomalies (20.0%) and other conditions originating in the perinatal period (6.6%).

Low Birthweight

Changes in the rates of low birthweight births in the last decade have not paralleled changes in infant mortality during the same time period. The rate of births less than

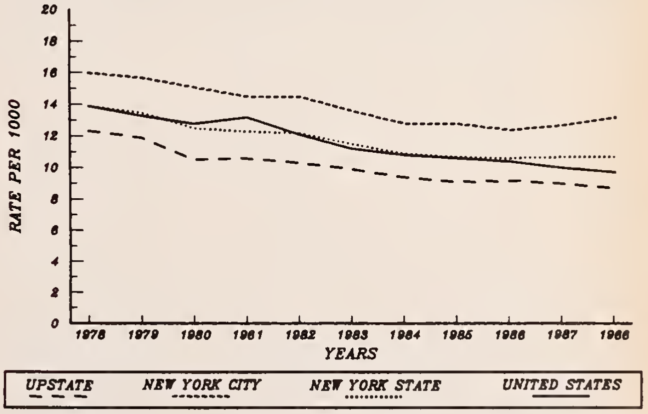

FIGURE 1. Infant mortality rates, New York State, Upstate, and New York City, 1978–1988.

2,500 grams has increased slightly from 7.7 of all live births in 1978 to 7.8% of all live births in 1988 (Fig 2). There was an 11.1% increase in the occurrence of births less than 2,000 grams in the same decade, from 2.7 to 3.0%.

As with infant mortality, the white/nonwhite differential is also evident when comparing the percent of low birthweight infants. The rate for nonwhite residents was 12.7%, more than double the white rate of 5.9%. Non-Hispanic whites had the lowest percentage of low birthweight infants in 1988 (5.3%) compared to Hispanic (8.3%) and non-Hispanic black (14.6%) births.

The premature birth (less than 37 weeks' gestation) rate has remained fairly static over the last decade. The 1988 rate of 10.6%, however, is higher than the 1987 rate of 9.9%. The rate of small-for-gestational-age infants, defined as births with 37 or more weeks' gestation and weighing less than 2,500 grams, increased slightly from 3.4% in 1987 to 3.5% in 1988.

Congenital Malformations

The Congenital Malformations Registry collects clinical and demographic information on every child with a congenital malformation that is diagnosed by the time the child is two years old. The registry now has data on the cohort of children born in 1985. There were 16,744 children born in 1985 reported as having a congenital malformation. This represented 6.5% of all live births in that year. More than 80% of these children were reported as having one malformation. The rate of malformation was highest among black children (7.6%) followed by white (6.1%) and other nonwhite (6.1%).

A major malformation is defined as one having an adverse effect on the individual's health, functioning and social acceptability. Of those born in 1985, 9,636 were reported as having one or more major malformations (3.7% of all births). The race-specific rate was 3.8% among white births, 3.8% among black births and 3.0% among other racial groups. A review of 13,077 major malformation reports revealed the organ systems most frequently affected were the musculoskeletal (29%), followed by cardiovascular (24%) and genitourinary (13%) systems (Fig 3).

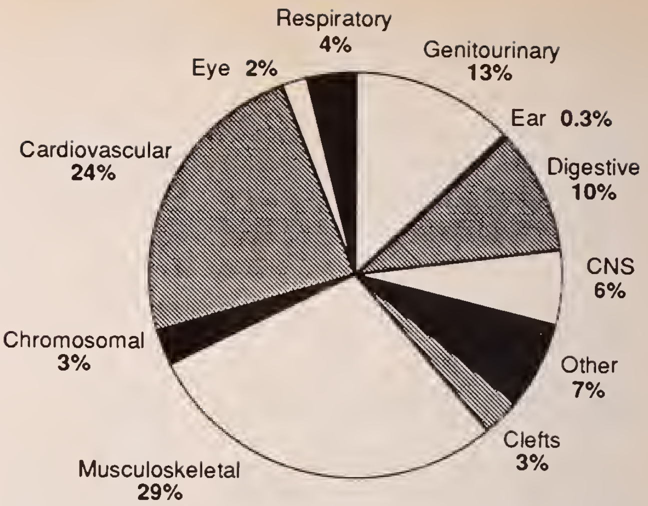

FIGURE 3. Major congenital malformations reported among children born in 1985, New York State.

Live Births/Pregnancy Ratio

During 1988, 66.9% of pregnancies reported by New York State residents resulted in live births. This was higher than the 1987 ratio of 64.8%. The ratio is higher for residents outside of New York City (73.9%) than for women residing in New York City (60.2%). There is a trend toward higher birth to pregnancy ratios since 1984 for all residents of New York State.

Maternal Age

The live birth to pregnancy ratio in women 19 years of age and younger in New York State had decreased from 46.4% in 1978 to 44.3% in 1987. However, the ratio increased to 47.0% in 1988.

Between 1978 and 1988, the pregnancy rate among teenagers (<19 years of age) increased from 40.5 per 1,000 to 48.8 per 1,000. This increase occurred statewide.

During 1988, almost 13% of the births to adolescents 17 years of age or younger in New York State were to females who had given birth previously and 22% to females who had previously been pregnant. Among births to teens 18–19 years of age, 25% of the women had previously given birth and 39% had previously been pregnant (Fig 4).

Maternal age and race have been identified with low

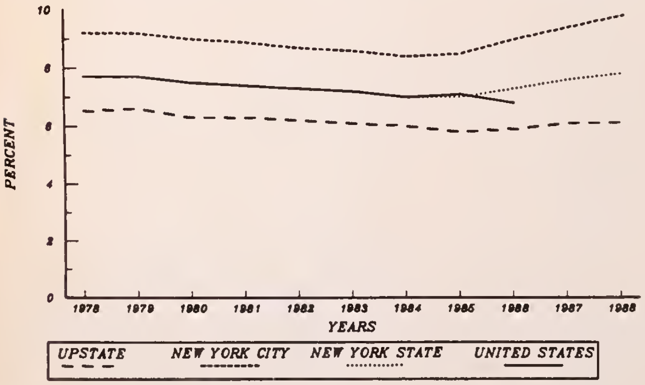

FIGURE 2. Percent of births <2500 grams, New York State, Upstate, and New York City, 1978–1988.

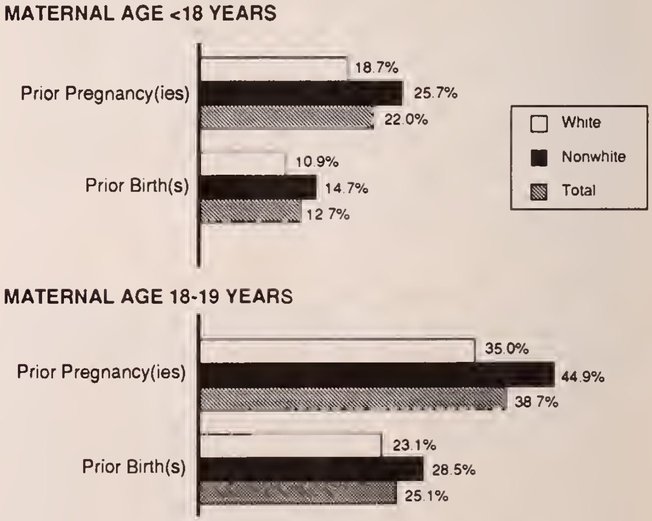

FIGURE 4. Percentage of births to adolescent mothers who had previously been pregnant or given birth, New York State, 1988.

birthweight infants. In 1988, the low birthweight rate was 10.4% for births to mothers 19 years of age and under, compared to 7.8% for all ages. Nonwhite mothers under age 20 delivered 13.6% of their babies weighing under 2,500 grams.

Prenatal Care

Many New York State women continue to receive inadequate prenatal care. Data for the period 1978 to 1986 showed only a 1.6% increase (81.4% to 82.7%) in the number of pregnant women receiving first trimester prenatal care. However, the percentage had increased from 1986 to 83.6% in 1988. From 1978 to 1987 there was an increase in the percentage of women who did not begin prenatal care until the third trimester or who received no prenatal care at all (11.1% to 11.4%). However, in 1988 there was a marked decrease in late or no prenatal care to 7.9%. This arises primarily from a decrease for New York City residents from 21.4% in 1987 to 13.9% in 1988. This trend may partially be an artifact of a change in the reporting of prenatal care on the NYC birth certificate between 1987 and 1988.

This article was adapted from the report entitled *Birth Outcomes of New York State March 1990*. Single copies of the report may be obtained from New York State Department of Health, Office of Public Health Information Group, Corning Tower, Room 742, Empire State Plaza, Albany, NY 12237, or by calling (518) 474-2543.

Pesticide poisoning a reportable disease in New York

To try to better understand the health effects of pesticides on New York residents, Pesticide Poisonings have recently been added to the list of reportable medical conditions in New York (Sections 22.11 and 22.12 of Title 10 {Health} of the Official Compilation of Codes, Rules, and Regulations of the State of New York). The following regulations, which were enacted by the New York State Public Health Council, became effective August 29, 1990:

> **22.11 REPORTING OF PESTICIDE POISONING.** Every physician, health facility, and clinical laboratory in attendance on a person with confirmed or suspected pesticide poisoning or with any of the clinical laboratory results as described in section 22.12 of this Part, shall report such occurrence to the State Commissioner of Health within 48 hours . . . .
>
> **22.12 REPORTABLE LABORATORY TESTS FOR PESTICIDE POISONING.** For purposes of section 22.11 of this Part the following laboratory tests are reportable to the State Commissioner of Health:
>
> (a) Blood cholinesterase levels which are below the normal range established by the clinical laboratory performing the test . . . .
>
> (b) Levels of pesticides in human tissue samples which exceed the normal range established by the clinical laboratory performing the test . . . .

All reports of confirmed or suspected pesticide poisonings in New York will be investigated by epidemiologists working with the newly formed pesticide registry within the Division of Environmental Epidemiology and Occupational Health, New York State Department of Health. The goals of the registry are to (1) promptly investigate and intervene in any situations where the risk of pesticide poisonings is continuing, (2) develop and implement interventions to reduce the risks of pesticide poisoning, (3) monitor both the acute and chronic effects of pesticide exposures and (4) increase the awareness of pesticide poisonings by members of the medical community.

Pesticides, which include insecticides, herbicides, fungicides, rodenticides and fumigants, are widely used in the United States. Agriculture accounts for approximately 75% of total pesticide use; the remaining 25% is used by industry, the public sector and homeowners. Because of the ubiquity of pesticides in our society, the potential for exposure is high in both occupational and nonoccupational settings. Pesticide poisonings have resulted from the following exposures:

Nonoccupational:

- accidental ingestions by children;
- intentional ingestions (suicide or homicide);
- ingestion of pesticide contaminated food or water;
- exposure to pesticide treated buildings, lawns, gardens, fields, and/or animals;
- exposure to pesticides released into the environment either during production or through dumping of waste products;

Occupational:

- worker exposure during the production, formulation and mixing of pesticides;
- worker exposure during the application of pesticides (exposure by either the applicator or by workers in the vicinity of the application area);
- worker exposure to pesticide residues in buildings, fields, and/or on plants;

Medical and laboratory personnel should report all suspected pesticide poisonings directly to the NYS Pesticide Poisonings Registry at 1-800-322-6850. Further information regarding the registry can be obtained by contacting the Bureau of Occupational Health at 518-458-6228. Advice regarding medical treatment can be obtained, toll free, from the National Pesticide Telecommunications Network at 1-800-858-7378 or from the local poison control center:

Western New York (Buffalo)
1-800-888-7655

Finger Lakes (Rochester)
1-800-333-0542

Central New York (Syracuse)
1-800-252-5655

Hudson Valley (Nyack)
1-800-336-6997

New York City
1-212-POISONS

Long Island (East Meadow)
1-516-542-2323

PREVENTING AIDS: THE DESIGN OF EFFECTIVE PROGRAMS

By Ronald O. Valdiserri, MD. 304 pp, illustrated. New Brunswick, NJ, Rutgers University Press, 1989. $15.00 (softcover), $38.00 (hardcover)

The onset of the epidemic of the human immunodeficiency virus (HIV) has necessitated the development of a programmatic response to prevent further transmission of the virus. These responses must occur within the larger framework of society's response to the disease. Nonetheless, as opposed to treatment and research programs in which the intervention is well defined, HIV infection prevention must integrate multiple biologic and social concepts. As educational and social interventions become defined, many programs aimed at reducing the spread of HIV are being developed. This text is intended as a guide for those developing such AIDS prevention programs.

The author is a clinical pathologist who has been involved in the HIV epidemic since its initial recognition. He has published numerous articles describing health education efforts for gay men, and played a major role in the highly successful recruitment efforts of one of the first studies to describe the natural history of HIV infection in this population. He currently serves as the Director of the Division of Laboratory Systems at the Centers for Disease Control in Atlanta. By virtue of both his strong background in the laboratory and clinical sciences as well as his work in AIDS education, he appears to be well-qualified to write such a book. Additionally, he has sought the assistance of two other authors to write the chapters on the theoretical foundation of AIDS prevention and the role of HIV testing in AIDS prevention.

The book is divided into eight chapters. The first is a basic primer in the epidemiology of the infection, and provides the link between epidemiology and the implications for prevention programs. The second chapter places AIDS prevention programs within the historical context of other STD control programs. There is a particularly insightful comparison of the responses to syphilis and HIV. The third chapter, written by a former colleague at the University of Pittsburgh, discusses the theoretical foundation of AIDS prevention. This chapter examines theories of health related behavior so that the reader might avoid the mistakes of many of the previous attempts of health education. In the fourth chapter, the author links the development of specific health promotion messages to specific target groups. This is a critical chapter, since the health education message will need to be tailored for specific age groups (eg, adolescents) or cultural groups (eg, immigrants). Chapter 5 draws on the experience of many different AIDS prevention programs using a case study approach, while the sixth chapter discusses the role of HIV testing in AIDS prevention. In addition to its use as a tool in preventing further spread of HIV, the use of HIV testing is becoming far more important as specific interventions, such as prophylaxis of opportunistic infection and the use of zidovudine, become standard practice in individuals who are not yet symptomatic. The last two chapters discuss the evaluation of AIDS prevention programs and further define the barriers to AIDS prevention programs.

This book is a practical and accessible guide in the development of HIV prevention programs. Until its publication there was a critical gap in this literature. It will be useful to those building programs designed to reach many groups—whether they be adolescents, minorities, or intravenous drug users. In particular, as AIDS prevention programs expand and bring in individuals trained in other fields, this book will serve to emphasize the commonality of AIDS prevention with other health promotion issues.

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YOUR BABY AND CHILD: FROM BIRTH TO AGE FIVE

By Penelope Leach, PhD. 553 pp, illustrated. New York, Alfred A. Knopf, Publisher, 1989. $16.95 (paperback)

Dr Leach, a British-trained child psychologist, skillfully describes the stages of normal child development in this revised volume. Her aim is to present parents with an understanding of their child's growth and development from the "child's point of view." These concepts are beautifully depicted in photographs by Camilla Jessel.

The book is divided into chapters dealing with the newborn, the baby under six months, the infant six months to one year, the toddler, and the preschool child. Each chapter addresses basic age-appropriate issues such as sleeping, feeding, health, play, and emotional and cognitive development. Although Dr Leach states that there are no rules in child care, at times she tends to present her opinions as dogma. A new parent might interpret these generalizations as facts, such as the statement that a six- to 12-month-old baby eats three to four times a day. Certainly, many six-month-olds eat more than four times in a 24-hour period. Pediatricians also might take exception to some of her recommendations such as adding sugar to baby cereal because "most babies will refuse it" otherwise.

Many inserts, drawings, and tables present concise illustration of the text. The book serves as a useful reference for parents, with a complete index and appendices on illness and medical emergencies. Here, again, a flaw is noted. The instructions on how to handle a choking emergency do not comply with the recommendations of the American Red Cross. Other appendices include suggestions on simple yet creative playthings for the infant and older child.

Overall, *Your Baby and Child* is an excellent text for parents, sensitively narrating the phenomenon of the development of a human being from birth to age five. It also provides a virtual encyclopedia of day-to-day child care. It will be received and recommended with the same enthusiasm as was the 1977 edition.

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THE COMPLETE BOOK OF PREGNANCY AND CHILDBIRTH

By Sheila Kitzinger. 399 pp, illustrated. New York, Alfred A. Knopf, Publisher, 1989. $18.95 (hardcover)

This book is a comprehensive and accurate guide for the general public. Ms

Kitzinger, a social anthropologist and childbirth educator, has centered her research on women's experience of pregnancy, childbirth, and motherhood. This is the second edition of her book, which was originally published in 1980.

The book is organized into chapters on early pregnancy, physiologic and emotional aspects of pregnancy, late pregnancy, childbirth, and the newborn period. There is also an important section on abortion, miscarriage, and stillbirth. The logical sequence breaks down at points (such as the discussion of positions for labor in both the chapter on late pregnancy/preparation for labor and again in the chapter on labor and delivery). However, the excellent index, glossary, and appendices and the very readable format (with tables, diagrams, and inserts) more than compensate for the occasional lapses in organization.

The information on physiology of pregnancy and childbirth is more complete than most books on the subject, and the 1989 revised edition includes new information, where appropriate, such as brief discussions of AIDS and pregnancy and chorionic villus sampling. However, it should be noted that Ms Kitzinger does not follow the current American College of Obstetricians and Gynecologists recommendations for pregnant women in that she recommends exercises in which the woman lies flat on her back.

Ms Kitzinger focuses on psychoprophylaxis and the emotional aspects of the childbirth experience. Obstetricians and professionals in the field should be aware that the author is a major proponent of what might be considered alternative birthing experiences, such as home births and water deliveries. She is so opinionated in her discussion of conventional obstetrical care that she leads the reader to expect major difficulties and even confrontation during the first visit with the obstetrician. She outlines the need for arbitration techniques in discussing the anticipated labor/birth experience. Furthermore, the author is so emphatic about the importance of the immediate postpartum period in the bonding experience that she neglects to reassure parents that parent-child contact immediately after birth is not required for adequate bonding. This important and professionally validated fact is not included. Mothers of babies requiring immediate intensive care or mothers requiring general anesthesia may feel unnecessary grief at not being able to hold their babies right away.

There are a multitude of books for the lay public on pregnancy and childbirth. The obstetric practitioner may feel confident that *The Complete Book of Pregnancy and Childbirth* provides thorough, medically up-to-date information in the context of viewing women as positive and active participants in their own medical care.

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THE BIRTH OF THE FAMILY: AN EMPIRICAL INQUIRY

By Jerry M. Lewis, MD. 224 pp. New York, Brunner/Mazel Publishers, 1989. $26.95 (hardcover)

Family systems medicine, a merger of the fields of family medicine and family therapy, is still in its infancy. While the field now boasts a journal devoted to the publication of original research and offers several fellowship training programs, many of the concepts in the field lack an empirical basis.

The concept of the family life cycle is central to an understanding of families in family medicine. The concept is analogous to Erickson's individual developmental stages in that families pass through periods of transition during which developmental tasks must be faced. These stages, which include marriage, pregnancy, launching of children, retirement, and death, represent opportunities for family growth as well as sources of family stress. The construct offers clinical utility in that it alerts the physician to periods during which the family may need support or even therapy. Unfortunately, few studies have examined the validity of the concept.

The Birth of the Family is a longitudinal study of family development that examines the relationship between family competence and successful transition to parenthood. Such studies are rare both because the field is young and because of the complex methodology involved.

Lewis, a senior research psychiatrist at Timberlawn Psychiatric Research Foundation, brings 25 years of family research to his ambitious project. He lays the groundwork in the opening chapter with a discussion of "the continuum of family competence," a construct central to his study. Lewis argues that family function can be assessed on a continuum ranging from highly competent to severely dysfunctional. Moreover, he argues that family function predicts a family's ability to weather such developmental tasks as the incorporation of a first child into the family.

The study was conducted by assessing the marital competence of 38 white, middle class, married, childless couples from obstetric practices at Baylor University Medical Center. Families were assessed twice prenatally, at the birth of the child, and at three months and one year postpartum. Family competence was assessed utilizing a variety of modalities including videotapes, audiotape transcripts, and individually administered inventories.

The results are generally supportive of the hypothesis that more competent families better navigate the passage to parenthood and tend to remain functioning at a better level.

Unfortunately, the study is plagued by a number of methodologic problems not the least of which is the small sample size of family subsets. Furthermore, Lewis tends to over-interpret his data and draws post hoc conclusions based on tenuous statistical correlations. Nevertheless, while Lewis may not have fully succeeded in his ambitious project, he clearly outlines a framework for future work in the field.

The book should be of interest to both clinicians and researchers in the field of family systems medicine. The concept of family competence has potential clinical applications while the research protocols reproduced in the appendix might prove useful to scholars in the field.

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TRAUMA, TRANSFORMATION, AND HEALING: AN INTEGRATIVE APPROACH TO THEORY, RESEARCH AND POST-TRAUMATIC THERAPY

By John T. Wilson, PHD. 368 pp. New York, Brunner/Mazel Publishers, 1989. $40.00 (hardcover)

Fifteen years ago Dr Wilson pioneered a landmark research project regarding post-traumatic stress disorders in Vietnam era veterans with his "Forgotten Warrior" project. Since that time he has published numerous articles regarding the psychological impact of post-traumatic stress. This text is a long-awaited, well-thought-out, and well-written publication that brings together a wide range of theoretical concepts, current research, and comprehensive clinical understanding, providing the most up-to-date and detailed account available of traumatic stress and its consequences. Therefore, for a clinician who is dealing with victims of post-traumatic stress, this text will act as a comprehensive guide to the latest research concerning the psychological, biological, and cultural concepts that one encounters when dealing with post-traumatic stress disorders.

The author moves through the contents of this publication, incorporating theory, empirical support, and clinical applications, and providing a number of important appendices such as the Vietnam era stress inventory and the Pearl Harbor research questionnaire. These two pieces of research methodology alone are well worth the price of this book. As Wilson moves through his text, painting an interwoven matrix of theoretical frameworks from Freud to Selye, examining the interaction between trauma, the person, and the post-traumatic environment, he allows us to understand the interaction of psychological and biological processes in reaction to stress and the adaptations that may develop following stress.

Additionally, this text also addresses issues such as the impact of cultural beliefs with regard to the healing process following a traumatic stressor. An extremely interesting chapter on culture and trauma focuses on cultural rituals for healing, growth, and psychic integration. This chapter offers an in-depth view of the trauma healing process that has been performed for centuries by the Native American shaman. It also carefully reviews a Native American tribal methodology for dealing with the complex social, psychological, and religious processes used to reintegrate warriors within the matrix of their society. This description of returning warriors processed through a cleansing ritual ceremony is perhaps the best of its kind in current literature. As pointed out in the chapter, "These rituals have a common purpose of building inner strength and reaffirming individual identity in a sense of connectedness and continuity within a meaningful community." This chapter also explores the idea of a neurophysiologic link between certain rituals and positive therapeutic consequences which have been reported before in literature examining the adaptive function of a human spirit to emotional distress.

The strength of this book is its ability to examine the transformation that individuals go through even when confronted by insurmountable adversity. An excellent example of this is the thorough study provided on the psychiatric sequelae of the survivors of the Japanese attack at Pearl Harbor in 1941. This study examined 250 members of the Pearl Harbor Survivors Association and offers the same kind of thoroughness and precise methodology for which Wilson is famous in his past research projects with Vietnam veterans.

The clinician who deals with victims of extremely stressful life events certainly needs to be aware of the chapter covering intervention and principles of treatment. It carefully examines the challenging dynamics and progressions that these patients have related to stressors they have encountered. It also carefully examines post-traumatic stress disorder as the expected human pattern of adaptation to extremely stressful life events, and how in the course of treatment both the patient and the therapist should be united and allied in facilitating a safe and supportive environment for healing.

In summary, there is a great need for a comprehensive text describing the therapeutic treatment of post-traumatic stress patients. This text allows the clinician to offer the patient the ability to transform the trauma and restore psychological cohesiveness and continuity in personal identity. *Trauma, Transformation, and Healing* provides a clear, concise understanding of the treatment necessary for therapists to deal with their patients' traumatic events.

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THE HEART FACTOR FOOD PLAN: A COMPREHENSIVE DIETARY PLAN FOR A HEALTHIER HEART

By Mary Joan Oexmann, MS. 191 pp. New York, William Morrow & Co, Inc, 1990. $16.95 (clothcover)

Interest in nutrition has produced so many books for the layman that the bookstore shelves are bulging with them. Most of them have a special gimmick to make them saleable. This one is written by a nutritionist whose experience is concentrated on hypertension patients in a clinic setting.

The author's goal is to motivate her readers with some elementary knowledge to avoid the pitfalls of hypertension and obesity. Generally the book provides good advice, and some good tables and recipes for cutting down calories. In some areas the author goes a little too far in stressing some points that are not generally accepted, such as the therapy of hypertension with potassium. We know that sodium has a deleterious effect on volume, and thus on hypertension. But more potassium is not the answer. Also, not everyone needs to lower sodium intake. This is a genetic sensitivity, and some people experience adverse effects from too much salt restriction. My worry would be that readers would follow this advice without medical supervision. Also, I do not understand what the author means by "nutrient to the heart"; I am not aware that there is a special nutrient to the heart.

While *The Heart Factor* sounds impressive for a title, the formula (meq.Na − meq.K) would lead readers to believe that only sodium and potassium are essential factors with respect to cardiac disease and hypertension. There can be no quarrel, however, with the fact that the author urges the reader to make a commitment to identify the sodium, potassium, and percentage of fat content in their daily food intake, which is certainly good advice. The book is easy to read, concise, and has good tables on calorie counts and percentage of fat content and other minerals. The advice that "calories do count" and to "either cut down on your intake or spend more calories with exercise" is valuable. The author's suggestions on how to deal with the "hungries" and with the common excuses for not making good food choices are sound and effective. The chapter on risk factors is good and makes it easy for the reader to identify with the issues.

The problem of hyperlipidemia in this country is as important as, if not more so than, that of the electrolytes. Yet the chapter on cholesterol is very sketchy, and the importance of the difference between saturated and unsaturated fats is not discussed.

The book is not intended for the medical profession, but rather for the general public. Thus, it does not add to the medical literature. There are many food plans and recipe books, all helpful to the layman. On balance, the book gives a good introduction to nutrition for the layman, and offers solid advice on the risk factors. It is easy to read, practical, and not outrageous. I hope it will reach some people and help a lot of them.

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LEGAL ASPECTS OF MEDICINE

Edited by James R. Vevaina, MD, Roger C. Bone, MD, and the Honorable Edwin Kassoff. 340 pp. New York, Springer-Verlag, 1989. $73.00 (hardcover)

Cookbooks are useful tools to have in one's life. They can make the unknown not only comprehensible but also enjoyable. They can cover a wide subject matter or a limited menu. In any event, they are all divided into chapters which discuss a single topic, with one topic unrelated to the next. That is, you do not have to read the chapter on peach pies to understand the chapter on angel food cake.

This "cookbook" approach of *Legal Aspects of Medicine* has the same advantages and disadvantages. There really is no overall theme, but particular chapters can be interesting and instructive.

The book is divided into two parts: the first, "Law in Medicine: An Overview"; and the second, "Legal Dimensions of Cardiology, Pulmonary Medicine, and Critical Care Medicine." Two of the editors specialize in pulmonary care, and presumably that is the reason for the book's emphasis on pulmonary medicine. Most of the chapters discuss very narrow subjects of pulmonary care, and would probably be very interesting to a specialist. Only a small percentage of this part of the book is devoted to legal issues and they are generally well written.

Chapter 23, "Withholding and Withdrawing Life Support," presents some genuine guidelines, not only for a physician in the intensive care unit, but for any physician taking care of critically ill patients. This chapter should be read in conjunction with chapter 22, "Brain Death and the Law." The combination gives a thoughtful analysis of problems that must be faced by physicians every day.

Part I, "Law in Medicine," gets off to a very shaky start in the first chapter, authored by Melvin Belli. There is an "editorial comment" after Mr Belli's chapter attempting to emphasize his credentials. Whether Mr Belli represented Mae West, however, does not seem particularly relevant to the issues of medical malpractice. Nothing in the chapter is relevant, beginning with his allegation of "the development of the well-documented 'conspiracy of silence.'" Mr Belli also relates how he sued and was victorious against some physicians. He goes on to say, "Doctors who have not committed any wrongdoing will be surprised to learn that their candid cooperation with the plaintiff's attorney during the preliminary investigation often will lead to a dropping of the suit before it is filed." What Mr Belli has omitted to add is that cooperating with the plaintiff's lawyer may very well be a breach of the doctor's professional liability insurance contract.

Unfortunately, Mr Belli is not the only plaintiff's attorney who is given an opportunity to write a chapter in this book. It is a mystery why members of the medical profession turn to the plaintiff's bar to seek advice. Would a chicken farmer turn to the fox for guidance?

Chapter 3, "The Defense of a Malpractice Case," provides some solid information about what is involved in a lawsuit. The concept conveyed that many trials "are won or lost at deposition" is an accurate and critical comment. Unfortunately, the subject is treated in just a few lines whereas it probably deserved its own chapter.

Chapter 5 should be mandatory reading for every physician. This chapter, "The Regulatory Law as It Affects Physicians," discusses legal processes that may be more damaging to physicians than malpractice litigation. The subject is investigations of physicians by government agencies. There is an excellent description of the often devious operation of such agencies, along with a very clear list of things that physicians should do to protect themselves once they become aware of such an investigation. The reason why physicians must be on guard with the first hint of an administrative investigation is summarized by the author when he states that "physicians under investigation by state agencies have fewer rights than common criminals as they are not entitled to nor normally granted the right of due process." In a medical malpractice case, the issue is money (usually from an insurance company), and the doctor is protected by basic due process. With administrative hearings, the issue may be one's livelihood (one's license), and the physician is often denied due process.

Chapter 8, "Risk Management for Physicians," is a very readable description of what a risk management office in a hospital is attempting to do. In recent years, certainly in New York, there has been an avalanche of laws regulating hospitals and the practice of medicine. This chapter is useful in understanding why an office of risk management is crucial in this highly regulated world.

On the other hand, the chapter, "Improving and Refocusing the Medical-Legal System," is mysterious. The author seems to be saying that the physician has too much of an advantage in a medical malpractice case. Whether the author realizes it or not, what he is suggesting is that a jury, not physicians, should decide what was appropriate medical practice in each case. Such an approach would not only be devastating to the defense of a malpractice case; but also, it would be devastating to the practice of medicine.

The many chapters on pulmonary care, although very interesting, shed little light on legal issues. The chapters that discuss legal issues attempt to summarize some very basic knowledge with mixed results. It is not "the blending of great minds in law and medicine" that the editors had hoped to achieve.

Unfortunately, there is no simple recipe to make a lawsuit "a piece of cake."

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THE STATE OF HEALTH IN GREENLAND

By Jens Misfeldt, MD, Anne Rancke-Madsen, Soren Nordby Holck, and Marianne Pedersen. 116 pp. Nuuk, Greenland, Landslaegeembedet, 1989. (softcover)

This volume represents the annual report of the Chief Medical Officer of Greenland for the years 1987 and 1988 and will be of considerable interest to those concerned with health and health care in the Arctic. However, its general observations and conclusions will also interest a wide range of physicians.

Greenland has an indigenous population of 45,000 and a Danish population of 10,000. During the past few decades, the traditional hunting and fishing subsistence life style of Greenlanders has been dramatically altered by industrialization and settlement in towns and hamlets, many along the southwest coast. Because of a thriving fishing industry, per capita income is around $10,000 per year. Despite many advances in health services and living standards, life expectancy is still ten years below that of Denmark, mainly due to a high infant mortality rate. Greenland has one of the highest suicide rates in the world and one of the highest per capita consumption rates for alcohol. The abortion rate is also very high, in large measure because of the provision of free abortions prior to the 12th week of gestation. Sexual abuse of children is a major problem and is the current focus of much public attention. Violence associated with alcohol abuse is a significant problem. Also related to alcohol abuse is the fact that Greenland has the highest rate of jaw fractures in the world. Most are the result of direct injury inflicted by intoxicated men.

Cancer rates are similar to those seen among the indigenous populations of Alaska and Canada. As in those areas, the rates of esophageal, pharyngeal, and salivary gland cancer are higher than in Europe and the United States. However, skin and prostate cancer are relatively rare. Lung and cervical cancer have recently shown dramatic increases, the former being due to a high rate of cigarette smoking. Cancer currently accounts for 18% of all deaths, infectious disease for 4%, and cardiac and cerebrovascular disease for 24.8%. Sexually transmitted diseases are a major problem, even though the incidence of gonorrhea fell between 1984 and 1988.

The first cases of human immunodeficiency virus (HIV) infection were diagnosed in 1985. In 1987, four more cases were diagnosed and in 1988, six. There was one death recorded from AIDS related pneumonia in 1988. The Chief Medical Officer launched an HIV serologic surveillance program in 1987. HIV testing is now routinely offered to all pregnant women, patients at sexually transmitted disease clinics, and those who may have been exposed to infection. All blood donors are also tested. In 1987, 9,266 people were tested of whom only 14 were positive. The Chief Medical Officer has an ongoing health education program for the prevention of AIDS in all districts of the country.

This volume attests to the great strides made in public health by the medical authorities in Greenland. It also demonstrates the key role played by social, environmental, and economic determinants in disease causation. In Greenland these have resulted in morbidity and mortality patterns distinct in many ways from those seen in Europe and the United States. A detailed English narrative is given at the beginning of the volume and bilingual Danish/English legends are presented with most tables. Copies may be obtained from the Chief Medical Officer of Greenland, PO Box 120, DK-3900, Nuuk/Godthab, Greenland.

PASCAL JAMES IMPERATO, MD
Editor

PATENTING THE SUN: POLIO AND THE SALK VACCINE

By Jane S. Smith. 413 pp, illustrated. New York, William Morrow & Co, 1990. $22.95 (hardcover)

The principal focus of this book is on the development of the Salk vaccine. As part of this story, the author, Jane Smith, describes the efforts of many excellent investigators and the part they played in the search for a polio vaccine. Many readers will note that her narrative recalls famous investigators such as Carrel, Frances, Rivers, Enders, and Basil O'Connor. Among those who were stricken by poliomyelitis, the most renowned was President Franklin Delano Roosevelt.

The author spins an interesting and enjoyable tale which details the search for the virus, the testing of the vaccine, and the near eradication of the disease in the United States. Also covered are the rivalry between Sabin and Salk, the live versus the killed vaccine, setbacks, adverse reactions, and the ultimate success. Vaccine development required the use of monkeys, human embryonic nervous tissue, and placental tissue.

Animal models were extremely critical in this research. Those opposing such research should read this volume and also familiarize themselves with Pasteur's work in developing anthrax and rabies vaccines which saved countless numbers of animals.

The author includes interesting anecdotes. Among them is Salk's answer to the journalist Edward R. Murrow's question, "Who owns the patent on this vaccine?" Salk replied, "Well, the people. I would say there is no patent. Could you patent the sun?"

Well done, Jane Smith!

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ORGANIZING VILLAGERS FOR SELF-RELIANCE: A STUDY OF GONOSHASTHYA KENDRA IN BANGLADESH

By Jayanta Kumar Ray. 104 pp. Calcutta, India, Orient Longman Ltd, 1986. NP (paperback)

Gonoshasthya Kendra are community based centers that deal with health, nutrition, family planning, education, and agriculture and which emphasize self-reliance. The focus of this book is on one such center in Bangladesh. There are similar centers located in India as well. The author, Jayanta Kumar Ray, is a distinguished professor who has written on a wide range of topics.

The text outlines the various programs undertaken by the center and describes how it acts as an instrument of reform in the community. It is significant that government employees do not participate in the center's programs. Special coverage is given to how the center teaches self-reliance to children. The emancipation of women and special issues relative to the health and mental health of women are dealt with in some detail.

Unfortunately, the author does not provide data to substantiate a number of his conclusions. Still, the book achieves its objective of providing guidelines for those who wish to undertake similar initiatives.

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BOOKS RECEIVED

AIDS

AIDS Carriers In My Practice. By Medical Video Productions, 1 videotape, 28 minutes, $89.00, rental $45.00/2 weeks. Decatur, Ga, Medical Video Productions, 1989.

AIDS In Your Life. By Medical Video Productions, 1 videotape, 20 minutes, $49.00, rental $35.00/2 weeks. Decatur, Ga, Medical Video Productions, 1990.

The Myth of Heterosexual AIDS: How a Tragedy Has Been Distorted by the Media and Partisan Politics. By Michael Fumento. 411 pp. New York, Basic Books, 1990. $22.95 (hardcover)

ANESTHESIA

Anesthesia for Cardiac Surgery. By James A. DiNardo, MD, and Michael J. Schwartz, MD. 325 pp, illustrated. East Norwalk, Conn, 1990. $55.00 (hardcover)

CARDIOLOGY

Pearls & Pitfalls in Electrocardiography. By Henry J. L. Marriott, MD. 158 pp, illustrated. Philadelphia, Lea & Febiger, 1990. $17.50 (paperback)

ENDOCRINOLOGY

Endocrinology of Thermal Trauma: Pathophysiologic Mechanisms and Clinical Interpretation. Edited By Rajko Dolecek, MD, Loredana Brizio-Molteni, MD, Agostino Molteni, MD, and Daniel Traber, PHD. 469 pp, illustrated. Malvern, Pa, Lea & Febiger, 1990. $78.50 (hardcover)

GASTROENTEROLOGY

Flexible Sigmoidoscopy: Techniques and Utilization. Edited by Melvin Schapiro, MD, and Glen A. Lehman, MD. 227 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1990. $45.00 (hardcover)

GERIATRICS

Geriatric Nutrition: A Comprehensive Review. Edited by John E. Morley, MD, Zvi Glick, PHD, and Laurence Z. Rubenstein, MD. 521 pp, illustrated. New York, Raven Press, 1990. $89.00 (hardcover)

HEALTH CARE ADMINISTRATION

HCPCS 1990: Medicare Level 2 National Codes. By Professional Medical Management Corporation. 214 pp. Willowbrook, Ill, Professional Medical Management Corp, 1990. $28.00 (spiralbound)

The Crisis in Health Care: Ethical Issues. Edited by Nancy F. McKenzie, PHD. 640 pp. New York, Penguin Books, 1990. $12.95 (paperback)

HISTORY OF MEDICINE

Patenting the Sun: Polio and the Salk Vaccine. By Jane S. Smith. 413 pp, illustrated. New York, William Morrow & Co, Inc, 1990. $22.95 (hardcover)

INTERNAL MEDICINE

Risk and Prevention of Arterial Lipidoses. By Peter Schwandt. 183 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1990. $22.50 (paperback)

MEDICAL ETHICS

Ethics in Medicine: Individual Integrity Versus Demands of Society (Karolinaka Institute Nobel Conference Series). Edited by Peter Allebeck, MD, and Bengt Jansson, MD. 240 pp. New York, Raven Press, 1990. $99.00 (hardcover)

MISCELLANEOUS

Quality of Life Assessments in Clinical Trials. Edited by Bert Spilker, MD. 492 pp, illustrated. New York, Raven Press, 1990. $85.00 (hardcover)

Neurosciences: The Basics. By Brian A. Curtis, PHD. 172 pp, illustrated. Malvern, Pa, Lea & Febiger, 1990. $19.75 (paperback)

Strangers at Home: Vietnam Veterans Since the War. Edited by Charles R. Figley, PHD, and Seymour Leventman, PHD. 416 pp. New York, Brunner/Mazel Publishers, 1990. $26.95 (paperback)

Males With Eating Disorders. Edited by Arnold E. Andersen, MD. 288 pp, illustrated. New York, Brunner/Mazel Publishers, 1990. $29.95 (hardcover)

Advances in Pain Research and Therapy. Volume 16. Second International Congress on Cancer Pain. Edited by Kathleen M. Foley, MD, John J. Bonica, MD, and Vittorio Ventafridda, MD. 559 pp, illustrated. New York, Raven Press, 1990. $110.00 (hardcover)

Dinosaurs Alive and Well: A Guide to Good Health. By Laurie Krasny Brown and Marc Brown. 32 pp, illustrated. Boston, Mass, Little, Brown & Co, 1990. $14.95 (hardcover)

Medical Abbreviations: 7000 Conveniences at the Expense of Communications and Safety. Fifth Edition. By Neil M. Davis. 158 pp. Huntingdon Valley, Pa, Neil M. Davis Associates, 1990. $9.95 (paperback)

Advances in Pain Research and Therapy: Myofascial Pain and Fibromyalgia. Volume 17. Edited by James R. Fricton, DDS, and Essam A. Awad, MD. 376 pp, illustrated. New York, Raven Press, 1990. $105.00 (hardcover)

A Wise Birth: Bringing Together the Best of Natural Birth with Modern Medicine. By Penny Armstrong, CNM, and Sheryl Feldman, MA. 288 pp. New York, William Morrow and Co, Inc, 1990. $18.95 (clothcover)

Prisoners of Ritual: An Odyssey Into Female Genital Circumcision in Africa. By Hanny Lightfoot-Klein. 306 pp, illustrated. Binghamton, NY, The Haworth Press, Inc, 1989. $14.95 (paperback)

Down Syndrome: The Facts. By Mark Selikowitz. 205 pp, illustrated. Cary, North Carolina, Oxford University Press, 1990. $18.95 (hardcover)

The Work and Play of Winnicott. By Simon Grolnick, MD. 232 pp. Northvale, NJ, Jason Aronson, Inc, 1990. $25.00 (hardcover)

NEUROLOGY

Epilepsy: Current Approaches to Diagnosis and Treatment. Edited by Dennis B. Smith, MD. 288 pp, illustrated. New York, Raven Press, 1990. $85.00 (hardcover)

Neurologic Emergencies: Recognition and Management. Second Edition. Edited by Michael Salcman, MD. 462 pp, illustrated. New York, Raven Press, 1990. $80.00 (hardcover)

The Practical Management of Spasticity in Children and Adults. Edited by Mel B. Glenn, MD, and John Whyte, MD. 325 pp, illustrated. Malvern, Pa, Lea & Febiger, 1990. $39.50 (hardcover)

Headache and Facial Pain: Diagnosis and Management. Edited by Alan L. Jacobson, MD, and William C. Donlon, MD. 332 pp, illustrated. New York, Raven Press, 1990. $88.00 (hardcover)

NUCLEAR MEDICINE

Nuclear Medicine Annual 1990. Edited by Leonard M. Freeman, MD. 256 pp, illustrated. New York, Raven Press, 1990. $98.00 (hardcover)

NUTRITION

The Heart Factor Food Plan: A Comprehensive Dietary Plan for a Healthier Heart. By Mary Joan Oexmann, MS. 191 pp. New York, William Morrow & Co, Inc, 1990. $16.95 (clothcover)

OBSTETRICS AND GYNECOLOGY

Unexplained Infertility: Basic and Clinical Aspects. (Serono Symposia Publications from Raven Press, Volume 62). Edited by G. Spera, and L. Gnessi. 315 pp, illustrated. New York, Raven Press, 1989. $78.00 (hardcover)

ORTHOPEDIC SURGERY

Knee Ligaments: Structure, Function, Injury, and Repair. Edited by Dale M. Daniel, MD, Wayne H. Akeson, MD, and John J. O'Connor, BE. 576 pp, illustrated. New York, Raven Press,

1990. $110.00 (hardcover)

PEDIATRICS

Bone Marrow Transplantation in Children. Edited by F. Leonard Johnson, MD, and Carl Pochedly, MD. 529 pp, illustrated. New York, Raven Press, 1990. $125.00 (hardcover)

The Malnourished Child. Edited by Robert M. Suskind, MD, and Leslie Lewinter-Suskind, MSS. 432 pp, illustrated. New York, Raven Press, 1990. $48.00 (hardcover)

PSYCHIATRY

Systematic Treatment Selection: Toward Targeted Therapeutic Interventions. By Larry E. Beutler, PHD, and John F. Clarkin, PHD. 384 pp, illustrated. New York, Brunner/Mazel Publishers, 1990. $40.00 (hardcover)

Resolving Resistances in Psychotherapy. By Herbert S. Strean, DSW. 312 pp. New York, Brunner/Mazel Publishers, 1990. $22.95 (paperback)

Stress Management: An Integrated Approach to Therapy. By Dorothy H. G. Cotton, PHD. 288 pp, illustrated. New York, Brunner/Mazel Publishers, 1990. $30.00 (hardcover)

Work Without Stress: A Practical Guide to Emotional and Physical Well-Being on the Job. By Samuel H. Klarreich, PHD. 176 pp. New York, Brunner/Mazel Publishers, 1990. $17.95 (clothcover)

A DSM-III-R Casebook of Treatment Selection. By Samuel Perry, MD, Allen Frances, MD, and John Clarkin, PHD. 395 pp. New York, Brunner/Mazel Publishers, 1990. $42.50 (hardcover)

The Broader Implications of Ericksonian Therapy. Ericksonian Monographs Number 7. Edited by Stephen R. Lankton. 102 pp, illustrated. New York, Brunner/Mazel Publishers, 1990. $25.00 (hardcover)

Brief Therapy: Myths, Methods, and Metaphors. Edited by Jeffrey K. Zeig, PHD, and Stephen G. Gilligan, PHD. 492 pp, illustrated. New York, Brunner/Mazel Publishers, 1990. $42.50 (hardcover)

SURGERY

General Surgery: Current Status and Future Trends. (Serono Symposia Publications from Raven Press, Volume 60) Edited by T. E. Starzl, and W. Montorsi. 502 pp. New York, Raven Press, 1989. $105.00 (hardcover)

Dry blood typing method yields instant results

A rapid blood typing method that requires no laboratory equipment, little training, and no refrigeration of reagents would be very useful as a bedside check of a patient's blood type prior to a transfusion, as well as in battlefield situations and in developing countries where there is limited access to refrigeration. Blakeley et al recently described a monoclonal antibody-based dry instant blood typing plate (*Lancet* 1990; 336:854–855).

Monoclonal antibodies for detecting A, B, and Rh blood group antigens can be dried in the presence of trehalose, a disaccharide, and will maintain full agglutinating activity for more than two years. Blood typing with the monoclonal antibody/trehalose plate involves rehydration with two drops of distilled water, addition of one drop of blood, and agitation of the plate for five minutes. A positive result will be evident within one minute at room temperature.

The authors tested the accuracy of this dry blood typing plate on 1,000 consecutive blood samples that were first typed by the standard semiautomated procedure. The two techniques were in agreement in 997 of 1,000 typings, for an accuracy rate comparable to the standard method of blood group typing.

Breakfast cereal produces anaphylactic reaction

Heartwise, a cereal introduced by the Kellogg Company in August 1989, is the first food product to contain psyllium, a grain used primarily in India and the Mediterranean region. Psyllium is a common ingredient in over-the-counter laxatives, and recent studies have suggested that as a form of soluble fiber, psyllium may lower serum cholesterol levels.

Nurses and other health care workers, as well as workers in the pharmaceutical industry, who are exposed to psyllium dust may develop a sensitivity to psyllium. Kaplan reported on a nurse who had an anaphylactic reaction shortly after eating Heartwise for the first time (*N Engl J Med* 1990; 323:1072–1073). The patient had the following symptoms: itching; swelling of the face, eyes, mouth, and tongue; coughing; vomiting; and syncope. Her treatment in the emergency room included epinephrine and diphenhydramine, and she was discharged on oral medications after four hours. Subsequent skin testing with psyllium yielded a strongly positive reaction.

In response to this report, the Kellogg Company described its efforts to warn of potential allergic reactions to psyllium. These include a notification statement distributed to hospitals and nursing homes, a prominent declaration on the front of the cereal box that Heartwise contains psyllium, and a newly added warning on the box aimed at persons who may have been occupationally exposed to psyllium. The Kellogg Company reported that the majority of documented allergic reactions to Heartwise occurred among nurses who had handled psyllium-containing laxatives.

Ivermectin shown effective against river blindness

Onchocerciasis, or river blindness, is caused by the parasite *Onchocerca volvulus*, whose microfilaria travel through the skin and damage the eye. Blackflies, which breed in rivers in tropical regions, transmit the parasite. The antiparasitic drug ivermectin can kill the microfilarial form of this parasitic worm, thereby blocking progression of the disease and reducing the spread of the disease by lessening the likelihood that a person bitten by a blackfly will be a host for the parasite (*Science* 1990; 250:116–118).

The parasite affects approximately 18 million people living in tropical regions. In one community of 14,000 people on a rubber plantation in Liberia, 90% of the adults harbored the parasite in 1987, at the start of a three-year study. A once-a-year dose of ivermectin administered to the vast majority of this population during the study period yielded an 84% reduction in the average number of microfilaria in skin snip samples. Among all the five-year-old children in this community, the use of ivermectin produced a 21% drop in the rate of new infection.

Experimental anticancer drug may prevent baldness caused by chemotherapy

As yet, the experimental anticancer drug ImuVert has only demonstrated its potential for preventing hair loss caused by chemotherapeutic drugs in rats. However, if this finding is substantiated and further clarified, the prospect of preventing hair loss in human cancer patients would be emotionally uplifting for patients facing the weakening and demoralizing effects of chemotherapy (*Science* 1990; 249:1564-1566).

ImuVert has been under study as an adjunct to standard chemotherapeutic agents. Researchers believe that ImuVert stimulates the immune system in such a way as to enhance the ability of anticancer drugs to destroy malignant cells. In one study of leukemic rats, two of 23 rats receiving the chemotherapeutic drug cytosine arabinoside fought the disease, whereas 18 of 20 rats receiving cytosine arabinoside and ImuVert overcame the leukemia.

The surprising, accidental finding was that while all of the 23 rats in the first treatment group lost some or all of their hair, none of the 20 rats on ImuVert experienced hair loss. Subsequent studies of ImuVert in combination with doxorubicin or cyclophosphamide in leukemic rats duplicated the protection against hair loss with the former drug, but was unsuccessful in preventing hair loss with cyclophosphamide.

Long term study of AIDS focuses on disease progression and therapeutic alternatives

The federally funded National Institute of Allergy and Infectious Disease together with the privately funded American Foundation for AIDS Research will sponsor a long term study of the AIDS epidemic in North America, compiling comprehensive information on HIV infected persons (*Nature* 1990; 347:416). The study will focus on disease progression among various patient populations, in-

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NEWS BRIEFS are compiled and written by Vicki Glaser.

cluding homosexual and bisexual men, women, pregnant women, and intravenous drug abusers, tracking the incidence of the opportunistic infections that plague persons infected with the human immunodeficiency virus.

Another goal of the study will be to assess the effectiveness of approved drugs such as AZT, experimental drugs including ddI, ddC, interleukin-2, alpha- and beta-interferon, and soluble CD4, alternative treatments such as acupuncture and herbal remedies, and "miracle" drugs such as Compound Q that receive media exposure as potential cures, with no scientific evidence to support such claims.

Breast cancer risk related to long term hormone replacement therapy

There is a continuous dialogue and controversy concerning the carcinogenic effects of hormone treatment in women, whether it is for contraceptive purposes or postmenopausal hormone replacement therapy (HRT). Recent studies in the US and Europe suggest an association between breast cancer risk and the duration of HRT, although studies from various countries differ with respect to relative risk and "safe" time periods for HRT (*CA* 1990; 40:289–295).

In the United States prior to 1976, HRT was essentially equivalent to estrogen replacement therapy. Following the publication of studies demonstrating that estrogen replacement therapy greatly increased the risk of endometrial cancer, it became accepted practice to include progestins in all or part of the HRT regimen for women with intact uteri. Progestins counteract the estrogens' proliferative effect on endometrial tissue.

Since combined estrogen and progestin treatment only became accepted practice in the late 1970s, data on the long term effects of combined HRT are just emerging. There is evidence that the addition of progestins promotes an adverse effect with respect to breast cancer risk.

Virtually no support for an association between HRT and breast cancer emanated from studies performed in the 1970s. Evidence that longer periods of HRT may increase breast cancer risk began to appear in the early 1980s. Recent studies of large populations tend to support this association for postmenopausal women with and without uteri and/or ovaries. Recent studies in the US have suggested that the risk estimate for breast cancer increased to 1.5 only after at least 15 years of HRT. Studies conducted in Sweden and Denmark demonstrated an even greater risk estimate after six to nine years of HRT. These differences could be due to the use of different forms of estrogen in various countries.

Asthma mortality increased in 1980s, especially among children

Of the 9.9 million persons affected by asthma in the US, children, blacks, and the poor represent a disproportionate share of this population. In a recent issue of *JAMA* (1990; 264:1683–1687), Weiss and Wagener came to an unsettling conclusion: "Both the prevalence and the severity of asthma seem to be increasing." Asthma mortality in the US among persons 5 to 34 years old steadily declined between 1968 and 1977, with a yearly drop of 7.8% (+1.0%) during the period 1970–1977. The mortality rate began to rise in 1978 and increased at an estimated rate of 6.2% (+1.2%) per year through 1987. Among children aged 5 to 14 years, asthma mortality rose at a significantly increased rate compared to that of persons aged 15 to 34 years. Males and females experienced a similar increase, and the rate was only slightly higher among whites compared to nonwhites.

There is no known cause for the increase in asthma mortality, and the authors ruled out changes in International Classification of Diseases coding and improved recognition of asthma—based on a review of autopsy findings—as possible explanations.

Alzheimer's disease is not a homogeneous genetic disorder

Although the cause of Alzheimer's disease is not known, the disorder has been considered to be a single entity with a genetic basis. Recent genetic linkage studies have suggested a genetic anomaly involving chromosome 21 as the cause of familial Alzheimer's disease, while other studies have failed to substantiate these findings.

A new study of members of 48 pedigrees used five DNA markers that constitute three independent loci on chromosome 21 to test for linkage (*Nature* 1990; 347:194–197). The authors concluded that there are multiple causes of Alzheimer's disease. Whereas a defect on chromosome 21 can cause the disorder, Alzheimer's disease could also be due to "mono- or polygenic genetic defects, from nongenetic agents, or from mixed genetic and environmental factors."

Medicinal leeches are making a comeback

Mention of leeches in relation to medical treatment conjures up the practice of blood-letting, a common treatment for a variety of illnesses prior to the 20th century. However, leeches can aid in healing, particularly following plastic and reconstructive surgery, and their use is documented in current medical journals. Wade, Brabham, and Allen review five cases in which they successfully used leeches to treat venous congestion following replantation or free tissue transfer, primarily involving crush injuries (*South Med J* 1990; 83:1168–1173).

The salivary glands of the leech secrete a local anesthetic, making the bite painless, as well as hirudin, a protein that has vasodilating and anticoagulant properties. The leech may secrete another protein, which acts as a thrombolysin. The combined effect of these proteins is to "prevent the collapse of host capillaries. This allows the parasite to suck uncoagulated blood."

The beneficial effects of hirudin on healing following microsurgery have contributed to the renewed acceptance of leeches in clinical practice. "With replantation as well as free tissue transfer, the most common vascular complications relate to venous outflow . . . ." The leech inhibits venous congestion and prevents coagulation until healing allows for restored venous flow. At the site of attachment, hirudin's effects are long lasting, with a continual oozing of blood from the wound for as long as 24 hours.

Jesse Murray Brown, MD, Jackson Heights. Died September 14, 1990; age 79. Dr Brown was a 1934 graduate of the New York Medical College, New York. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Anthony M. De Angelis, MD, Yonkers. Died August 1990; age 79. Dr De Angelis was a 1934 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Qualified Fellow of the International College of Surgeons. His memberships included the New York Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Alan Charles Geller, MD, Merrick. Died September 13, 1990; age 53. Dr Geller was a 1962 graduate of the Chicago Medical School, Chicago, Illinois. He was a Diplomate of the American Board of Internal Medicine. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

William B. George, MD, Rochester. Died August 28, 1990; age 72. Dr George was a 1943 graduate of the University of Rochester School of Medicine-Dentistry, Rochester, New York. He was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Earle Harold Harris, MD, Forest Hills. Died September 9, 1990; age 84. Dr Harris was a 1933 graduate of the University of Maryland School of Medicine, Baltimore, Maryland. His memberships included the American Public Health Association, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

Marie H. Heller, MD, Buffalo. Died September 4, 1990; age 64. Dr Heller was a 1950 graduate of the State University of New York at Buffalo School of Medicine, Buffalo, New York. Her memberships included the New York Academy of Medicine, the American Medical Women's Association, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Karl Herz, MD, Bronx. Died September 5, 1990; age 84. Dr Herz was a 1932 graduate of the Medizinische Fakultaet der Universitaet Koln, Koln, Nordrhein-Westfalen, Germany. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Stefanie Lichtenstein, MD, New York. Died August 15, 1990; age 77. Dr Lichtenstein was a 1938 graduate of the Deutsche Univerzita Medizinische Fakulta, Praha, Czechoslovakia. She was a Fellow of the American College of Anesthesiologists. Her memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the New York County Medical Society, and the Medical Society of the State of New York.

Samuel L. Lieberman, MD, Buffalo. Died August 23, 1990; age 76. Dr Lieberman was a 1938 graduate of the State University of New York at Buffalo School of Medicine, Buffalo, New York. He was a Diplomate of the American Board of Anesthesiology. He was a Fellow of the American College of Anesthesiologists. His memberships included the American Society of Anesthesiologists, Inc, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Louis Joseph Mautone, MD, St James. Died September 3, 1990; age 80. Dr Mautone was a 1940 graduate of the Facolta di Medicina e Chirurgia dell' Universita di Bologna, Bologna, Italy. He was a Fellow of the American Academy of Family Physicians. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Bernard E. Mishuris, MD, Fort Lauderdale, Florida. Died July 1990; age 82. Dr Mishuris was a 1935 graduate of the Faculte de Medecine de l'Universite de Lausanne, Lausanne, Switzerland. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Eugene Moskowitz, MD, Salisbury, Connecticut. Died September 1, 1990; age 78. Dr Moskowitz was a 1937 graduate of the Medizinische Fakultaet der Universitaet Basel, Basel, Switzerland. He was a Diplomate of the American Board of Physical Medicine and Rehabilitation. His memberships included the American Academy of Physical Medicine and Rehabilitation, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Benjamin F. Norris, MD, Guilderland. Died April 3, 1990; age 69. Dr Norris was a 1950 graduate of the Albany Medical College of Union University, Albany, New York. He was a Diplomate of the American Board of Pathology. He was a Fellow of the American College of Pathologists. His memberships included the American Society of Clinical Pathologists, the Medical Society of the County of Albany, and the Medical Society of the State of New York.

Oscar W. Oberlander Jr, MD, Syracuse. Died September 4, 1990; age 68. Dr Oberlander was a 1946 graduate of the State University of New York Upstate College of Medicine, Syracuse, New York. He was a Diplomate of the American Board of Ophthalmology. His memberships included the American Academy of Ophthalmology and Otolaryngology, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Norbert Joseph Roberts, MD, New York. Died September 1, 1990; age 74. Dr Roberts was a 1940 graduate of the State University of New York at Buffalo School of Medicine, Buffalo, New York. He was a Diplomate of the American Board of Internal Medicine and the American Board of Preventive Medicine, Inc. He was a Fellow of the American College of Physicians, the American College of Preventive Medicine, and the American College of Occupational Medicine. His memberships included the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Philip Sawyer, MD, Schroon Lake. Died August 17, 1990; age 81. Dr Sawyer was a 1933 graduate of the Charles Univerzita Fakulta of Ped-Gen Medicine, Praha, Czechoslovakia. He was a Fellow of the American Academy of Family Physicians. His memberships included the New York Academy of

Medicine, the American Geriatrics Society, the Medical Society of the County of Essex, and the Medical Society of the State of New York.

Edward Schall, MD, Miami Beach, Florida. Died September 8, 1990; age 99. Dr Schall was a 1917 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Joseph Schwartzman, MD, Brooklyn. Died September 19, 1990; age 85. Dr Schwartzman was a 1930 graduate of the New York Medical College, New York. He was a Diplomate of the American Board of Pediatrics. He was a Fellow of the American Academy of Pediatrics. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Simon Shulman, MD, Lawrence. Died September 7, 1990; age 86. Dr Shulman was a 1927 graduate of Tufts University School of Medicine, Boston, Massachusetts. He was a Diplomate of the American Board of Radiology. He was a Fellow of the American College of Radiology. His memberships included the Radiological Society of North America, Inc, the Nassau County Medical Society, and the Medical Society of the State of New York.

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INDEX

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ISSUE	NUMBER	PAGES
January	1	1-50
February	2	51-114
March	3	115-170
April	4	171-230
May	5	231-282
June	6	283-344
July	7	345-392
August	8	393-438
September	9	439-484
October	10	485-530
November	11	531-584
December	12	585-646

CLASSIFICATION CODES

CC = Clinical Conference
CPC = Clinicopathologic Conference
CR = Case Report
ED = Editorial Commentary
EP = Essay in Pediatrics
ER = Essay Review
HIST = History of Medicine
LE = Letter to the Editor
LEN = Leads from *Epidemiology Notes*
NF = News Feature

SUBJECT INDEX

Abdominal aortic aneurysmectomy
- Abdominal aortic aneurysmectomy without preoperative angiography: A prospective study, 176

Abdominal trauma
- Small bowel injuries following blunt abdominal trauma: Early recognition and management, 446

Acquired immunodeficiency syndrome
- Acquired immunodeficiency syndrome: The agenda for the 1990s (ED), 115
- AIDS and cytomegalovirus-related appendicitis (NF), 581
- AIDS behind bars: Epidemiology of New York State prison inmate cases, 1980–1988, 133
- AIDS education: Evaluation of school and worksite based presentations, 129
- AIDS is affecting women at increased rates (NF), 480
- AIDS legislation needed (LE), 163
- AIDS morbidity and mortality (LEN), 572
- Assessment of AIDS knowledge in selected New York State sexually transmitted disease clinics, 126
- Changes in AIDS risk behavior among intravenous drug abusers in New York City, 123
- Controversy grows over parallel track for experimental AIDS drug (NF), 169
- Deep venous thrombosis in patients with AIDS, 159
- Disseminated histoplasmosis in AIDS: Clinicopathologic features in seven patients from a non-endemic area, 234
- Disseminated *Pneumocystis carinii* infection in the acquired immunodeficiency syndrome, 155
- Dissemination of *Pneumocystis* (ED), 121
- Erosive arthritis, atypical psoriasis, and *Pneumocystis carinii* pneumonia in an HIV-positive man, 157
- Expanding the focus of human immunodeficiency virus prevention in the 1990s (ED), 116
- Extent of underreporting of AIDS documented in South Carolina (NF), 168
- Federal funds allocated for clinical care of AIDS patients (NF), 526
- Growing role of bacterial infections in AIDS-related morbidity and mortality (NF), 168
- HIV infected hospital patients in New York State: The development of longitudinal information from a hospital discharge data system, 238
- Increasing role of primary care in the management of HIV-infected patients (ED), 119
- Large pericardial effusion in a patient with human immunodeficiency virus type 1 (CR), 612
- Lessons from New York City on HIV/AIDS in adolescents, 143
- Long term study of AIDS focuses on disease progression and therapeutic alternatives (NF), 629
- Medical student's dilemma, 154
- Number of AIDS patients on Medicaid is increasing (NF), 581
- Perinatally acquired HIV infection: AIDS' youngest victims (NF), 169
- Physicians needed to work with AIDS patients (LE), 163
- Position paper of the Associated Medical Schools of New York on HIV testing and access to treatment, 496
- Residential and home health care for people with AIDS and HIV-related illnesses (LEN), 43
- Seroprevalence survey for human immunodeficiency virus antibody in mentally retarded adults, 139
- Soluble CD4: A new antiviral treatment for AIDS (NF), 280
- Synthetic protein binds AIDS virus and prevents its spread (NF), 480
- Thoracic lymphadenopathy and disseminated *Pneumocystis carinii* in AIDS (CPC), 146
- Upper airway obstruction due to Kaposi's sarcoma in the acquired immunodeficiency syndrome (CR), 613

Adenocarcinoma
- Adenocarcinoma of the cervix (ED), 535
- Primary signet ring cell carcinoma of the uterine cervix (CR), 559

Adolescent health
- Intervention to reduce the sale of cigarettes to minors, 54
- Lessons from New York City on HIV/AIDS in adolescents, 143
- Risk factors for teenage suicide (NF), 46
- Student participation in mixed gender sports competition (LE), 76

Adrenocorticotrophic hormone deficiency
- Isolated adrenocorticotrophic hormone deficiency presenting as chronic hyponatremia (CR), 210

Aeromedical emergency services
- Aeromedical emergency trauma services and mortality reduction in rural areas, 358

Aging
- Growth hormone therapy may reverse some age-related bodily changes (NF), 481
- New Merck manual addresses geriatrics (NF), 481

Alaska
- Alaskan community health aide scheme: A successful rural health program, 61

Alcohol
- Alcohol and public health: Implications for New York State (LE), 41
- Behavioral risks: Mother knows best (ED), 346
- Death takes a ride: Alcohol-associated single vehicle fatalities revisited, 349
- Effects of prenatal alcohol exposure on child development (NF), 388
- Multiple addictions: Co-synchronous use of alcohol and drugs, 596

Allergic reactions
- Breakfast cereal produces anaphylactic reaction (NF), 629
- Study refutes symptom-provocation method for determining food allergies (NF), 527

Alzheimer's disease
- Alzheimer's disease is not a homogeneous genetic disorder (NF), 630
- Diagnosing Alzheimer's disease with a biochemical assay (NF), 432
- Hydergine shown ineffective in Alzheimer's disease (NF), 526

Amputation
- Open supramalleolar amputation of the infected foot: An underutilized, two-stage procedure (LE), 77

Anesthesia
- Fetal heart rate changes during general anesthesia (LE), 272

Angina
- Cervical angina, 8

Angiography
- Abdominal aortic aneurysmectomy without preoperative angiography: A prospective study, 176

- **Animal experimentation**
  - Animal experimentation for medical research (LE), 214
- **Antibiotics**
  - Antibiotics and other drugs may inhibit effectiveness of oral contraceptives (NF), 341
- **Aortobronchial fistula**
  - Infected aortitis masquerading as bronchogenic carcinoma (CR), 415
- **Aplastic anemia**
  - Disseminated infection caused by *Fusarium solani* in a patient with aplastic anemia (CR), 609
- **Appendicitis**
  - AIDS and cytomegalovirus-related appendicitis (NF), 581
  - Gallstone appendicitis (LE), 77
- **Arthritis**
  - Erosive arthritis, atypical psoriasis, and *Pneumocystis carinii* pneumonia in an HIV-positive man, 157
  - Lofgren's syndrome (acute sarcoidosis) sine erythema nodosum mimicking acute rheumatoid arthritis (CR), 463
  - Low-dose cyclosporin reduces joint inflammation in rheumatoid arthritis (NF), 481
  - Promising new arthritis drug awaits testing in humans (NF), 281
  - Salmonella arthritis (CR), 266
- **Arthropathy**
  - Manual labor metacarpophalangeal arthropathy in a baker (CR), 268
- **Associated Medical Schools of New York**
  - Position paper of the Associated Medical Schools of New York on HIV testing and access to treatment, 496
- **Asthma**
  - Asthma mortality increased in 1980s, especially among children (NF), 630
  - Crack abuse and asthma: A fatal combination (CR), 511
  - Metaproterenol inhaler taken off market (NF), 46
- **Avicenna**
  - Avicenna, problem solving, and creativity during sleep (LE), 378
- **AZT**
  - **(see Zidovudine)**
- **Bacteremia**
  - Growing role of bacterial infections in AIDS-related morbidity and mortality (NF), 168
- **Baldness**
  - Experimental anti-cancer drug may prevent baldness caused by chemotherapy (NF), 629
- **Balloon angioplasty**
  - Balloon angioplasty finds application in gene therapy (NF), 582
- **Baltimore Homeless Study**
  - Health problems of the homeless (NF), 226
- **"Bartleby the Scrivener"**
  - Melville's "Bartleby the Scrivener" (LE), 422
- **Benzodiazepines**
  - Benzodiazepines: Additional effects of the triplicate program (LEN), 273
  - Benzodiazepines: Prescribing declines under triplicate program (LEN), 218
- **Beta blockers**
  - Side effects of beta blockers in patients with migraines (NF), 388
- **Bezoars**
  - Intestinal obstruction secondary to bezoars (CR), 561
- **Bicycle injuries**
  - Bicycle injuries (LEN), 426
  - Promoting bicycle helmet use in children (NF), 47
- **Bile duct carcinoma**
  - Bile duct carcinoma (ED), 53
  - Bile duct carcinoma simulating choledocholithiasis (CR), 69
- **Birth outcomes**
  - Birth outcomes in New York State (LEN), 619
  - Birth outcomes in New York State (part II) (LEN), 42
- **Blindness**
  - Ivermectin shown effective against river blindness (NF), 629
- **Blood**
  - Blood donors will now be tested for hepatitis C (NF), 432
- **Blood typing**
  - Dry blood typing method yields instant results (NF), 629
- **Books received, annotations of**, 48, 343, 627
- **Books reviewed**
  - *Anorectal, Presacral and Sacral Tumors: Anatomy, Physiology, Pathogenesis and Management* (Localio, Eng, and Coppa), 276
  - *Ask Sir James: Sir James Reid, Personal Physician to Queen Victoria and Physician-in-Ordinary to Three Monarchs* (Reid), 430
  - *Baby Time: A Grownup's Handbook to Use with Baby* (Brown and Brown), 477
  - *Biliary Tract Surgery: Tactics and Techniques* (Glassman), 385
  - *Birth of the Family, The: An Empirical Inquiry* (Lewis), 623
  - *Black Families in Crisis: The Middle Class* (Coner-Edwards and Spurlock, eds.), 224
  - *Blood, Sweat, and Cheers* (Fraser), 386
  - *Brief Therapy Approaches to Treating Anxiety and Depression* (Yapko), 576
  - *Bronchopulmonary Dysplasia* (Bancalari and Stocker, eds.), 89
  - *Child in Our Times, The: Studies in the Development of Resiliency* (Dugan and Coles, eds.), 477
  - *Child Psychiatry: A Primer for Those Who Work Closely with Children* (Coddington), 576
  - *Clinical Implications of Abnormal Digestive Tract Radiographs* (Morowitz), 523
  - *Complete Book of Pregnancy and Childbirth, The* (Kitzinger), 622
  - *Computer Applications in Psychiatry and Psychology* (Baskin, ed.), 577
  - *Day Hospital Group Treatment Program for Anorexia Nervosa and Bulimia Nervosa* (Piran and Laplan, eds.), 523
  - *Drugs and the Athlete* (Wadler and Hainline), 222
  - *Dry All Night: The Picture Book Technique that Stops Bedwetting* (Mack), 478
  - *Encyclopedia of Phobias, Fears, and Anxieties* (Doctor and Kahn), 525
  - *Essays on the Rise and Decline of Bedside Medicine* (Altschule), 479
  - *Evoked Potentials in Clinical Medicine* (Chiappa, ed.), 430
  - *Exercise and Sport Sciences Reviews* (Pandolf, ed.), 222
  - *Family Fitness Handbook, The* (Glover and Shepherd), 278
  - *"Feeling Great!" Wellness Program for Older Adults* (Weis), 429
  - *Forever Young: 20 Years Younger in 20 Weeks. Dr. Berger's Step-By-Step Rejuvenating Program* (Berger), 279
  - *Guide to Clinical Preventive Services: An Assessment of the Effectiveness of 169 Interventions* (Fisher, ed.), 79
  - *Gynecologic Surgery* (Sanz, ed.), 90
  - *Handbook of Psychiatric Nursing, A* (Wells, ed.), 223
  - *Healing and Restoring: Health and Medicine in the World's Religious Traditions* (Sullivan, ed.), 580
  - *Heart Disease in Infants, Children, and Adolescents* (Adams, Emmanouilides, and Riemenschneider, eds.), 385
  - *Heart Factor Food Plan, The: A Comprehensive Dietary Plan for a Healthier Heart* (Oexmann), 624
  - *House Calls: Memoirs of a Country Doctor* (Brown), 472
  - *Illustrated Guide to the Central Nervous System* (Sugiura, Robinson, and Stuart), 221
  - *Jury of My Peers: A Surgeon's Encounter with the Malpractice Crisis* (Snider), 278
  - *Legal Aspects of Medicine* (Vevaina, Bone, and Kassoff, eds.), 224, 625
  - *Letting Blood* (Platt and Platt), 525
  - *Make Sure You Do Not Have Breast Cancer* (Strax), 477
  - *M.D.: One Doctor's Adventures Among the Famous and Infamous from the Jungles of Panama to a Park Avenue Practice* (Kean and Dahlby), 221
  - *Medicare's New Hospital Payment System: Is It Working?* (Russell), 428
  - *Mental Health Care for Allied Health and Nursing Professionals* (Moffic, Ruiz, Adams, eds.), 476
  - *Merritt's Textbook of Neurology* (Rowland, ed.), 384
  - *Modern Perspectives in the Psychiatry of the Neuroses* (Howells, ed.), 223
  - *Morphine, Ice Cream, Tears: Tales of a City Hospital* (Sacco), 429
  - *Old Men of the Bowery: Strategies for Survival Among the Homeless* (Cohen and Sokolovsky), 476
  - *Organizing Villagers for Self-Reliance: A Study of Gonoshasthya Kendra in Bangladesh* (Ray), 626
  - *Otolaryngology: Problems in Primary Care* (Upchurch, ed.), 276
  - *Patenting the Sun: Polio and the Salk Vaccine* (Smith), 626
  - *Pediatric Neurosurgical Patient, The: A Cooperative Approach* (Ivan, ed.), 88
  - *Pediatric Pulmonary Disease* (Stocker, ed.), 88
  - *Perspectives on Animal Research* (Kaufman and Todd, eds.), 524
  - *Practice Made Perfect: The Physician's Guide to Communication and Marketing* (Kaplan), 524
  - *Preventing AIDS: The Design of Effective Programs* (Valdiserri), 622
  - *Rights of Patients, The* (Annas), 386
  - *State of Health in Greenland, The* (Misfeldt, Rancke-Madsen, Holck, and Pedersen), 625
  - *Strangers at Home: Vietnam Veterans Since the War* (Figley and Leventman, eds.), 579
  - *Stress Management: An Integrated Approach to Therapy* (Cotton), 578

Theories of Carcinogenesis (Iversen, ed.), 277
Toward a National Policy on Drug and AIDS Testing (Falco and Cikins, eds.), 276
Toxicologic and Pharmacologic Principles in Pediatrics (Kacew and Lock, eds.), 89
Trauma and the Vietnam War Generation: Report of Findings from the National Vietnam Veterans Readjustment Study (Kulka, Schlenger, Fairbank, Hough, Jordan, Marmar, and Weiss), 579
Trauma, Transformation, and Healing: An Integrative Approach to Theory, Research and Post-Traumatic Therapy (Wilson), 623
Treatment Planning and Dose Calculation in Radiation Oncology (Bentel, Nelson, and Noell), 429
Truth About Herpes, The (Sacks), 384
Unnatural Causes: The Three Leading Killer Diseases in America (Maulitz, ed.), 568
Work and Play of Winnicott, The (Grolnick), 576
Yankee Surgeon: The Life and Times of Usher Parsons 1788–1868 (Goldowsky), 277
Your Baby and Child: From Birth to Age Five (Leach), 622

Brain death
Defining brain death (LE), 517

Brain tumors
Central nervous system malignancies in children (EP), 601

Breast cancer
Breast cancer detection and counseling program (LEN), 85
Breast cancer risk related to long term hormone replacement therapy (NF), 630
Breast cancer screening: Changing physician practices and specialty variation, 288
Endometrial carcinoma in five patients with breast cancer on tamoxifen therapy (CR), 207
Is lightscanning a viable alternative to mammography for detecting breast cancer? (NF), 341
Mammography attitudes and awareness (LEN), 339
Sarcoidosis of the breast coexisting with mammary carcinoma (CR), 331
Screening for breast cancer: Are physicians doing enough? (ED), 285
Time trends in breast cancer incidence (LEN), 473

Breast disease
Fibrous disease of the breast in juvenile diabetes, 356, (LE), 617
Granulomatous mastitis (ED), 287
Sarcoidosis of the breast coexisting with mammary carcinoma (CR), 331

Campylobacter pyloris
Evaluation of methods for identification of *Campylobacter pyloris* infection, 4

Cancer
Adenocarcinoma of the cervix (ED), 535
Bile duct carcinoma (ED), 53
Bile duct carcinoma simulating choledocholithiasis (CR), 69
Breast cancer detection and counseling program (LEN), 85
Breast cancer risk related to long term hormone replacement therapy (NF), 630
Breast cancer screening: Changing physician practices and specialty variation, 288
Cancer incidence among NYC Hispanics 1982–1985 (LEN), 44
Carcinoma of the colon associated with systemic sclerosis (CR), 73
Carcinoma of the lung presenting with spontaneous pneumothorax (CR), 611
Cervical cancer in New York State (LEN), 521
Endometrial carcinoma in five patients with breast cancer on tamoxifen therapy (CR), 207
Experimental anti-cancer drug may prevent baldness caused by chemotherapy (NF), 629
Hypercalcemia and carcinoma of the esophagus (NF), 387
Is lightscanning a viable alternative to mammography for detecting breast cancer? (NF), 341
Lung cancer rates decreasing for young people in US (NF), 46
Ovarian small cell carcinoma (ED), 171
Para-endocrine hypercalcemia and ovarian small cell carcinoma (CR), 206
Primary signet ring cell carcinoma of the uterine cervix (CR), 559
Screening for breast cancer: Are physicians doing enough? (ED), 285
Skin cancer prevention (LEN), 520
Small cell carcinoma of the ovary: A therapeutic challenge (ED), 172
Time trends in breast cancer incidence (LEN), 473
Trabecular (neuroendocrine) carcinoma of the skin: Report of four cases and review of the literature (CR), 35

Capgras' syndrome
Capgras' syndrome (LE), 470

Carbon monoxide exposure
Cardiac effects of carbon monoxide exposure (NF), 582

Cardiac foreign bodies
Hypodermic needle embolization to the heart (CR), 368
Uninvited guests: Cardiovascular foreign bodies (ED), 347

Cardiovascular disease
Cholesterol levels and ten-year mortality among men with and without preexisting cardiovascular disease (NF), 433

Central nervous system malignancies
Central nervous system malignancies in children (EP), 601

Cerebral edema
Reversible hypoxic cerebral edema in severe hemolytic crisis (LE), 518

Cervical angina
Cervical angina, 8

Cervical cancer
Adenocarcinoma of the cervix (ED), 535
Cervical cancer in New York State (LEN), 521
Primary signet ring cell carcinoma of the uterine cervix (CR), 559

Cervical tuberculosis
Cervical tuberculosis (LE), 270

Charles Bonnet syndrome
Visual hallucinations as grief reaction without the Charles Bonnet syndrome (LE), 216

Chemical warfare
Gas warfare: Military-medical responsiveness of the Allies in the Great War, 1914–1918 (HIST), 499

Chemotherapy
Experimental anti-cancer drug may prevent baldness caused by chemotherapy (NF), 629

Children's health
Asthma mortality increased in 1980s, especially among children (NF), 630
Breakfast cereal produces anaphylactic reaction (NF), 629
Central nervous system malignancies in children (EP), 601
Children of substance abusers in New York State, 179
Effects of prenatal alcohol exposure on child development (NF), 388
Intervention to reduce the sale of cigarettes to minors, 54
Promoting bicycle helmet use in children (NF), 47
Student participation in mixed gender sports competition (LE), 76
Toy safety (LEN), 166

Choledocholithiasis
Bile duct carcinoma simulating choledocholithiasis (CR), 69

Cholesterol
Cholesterol levels and ten-year mortality among men with and without preexisting cardiovascular disease (NF), 433

Choriocarcinoma
Spontaneous renal hemorrhage secondary to choriocarcinoma (CR), 418

Chronic illness
Maintaining serenity in chronic illness, 366

Churg-Strauss syndrome
Churg-Strauss syndrome (CPC), 190

Cigarettes
(see Smoking, Tobacco)

Circumcision
Circumcision of the newborn (ED), 231
Current trends in routine newborn male circumcision in New York State, 243

Clinical Conferences (series)
Deep vein thrombosis in pregnancy, 309

Clinicopathologic Conferences (series)
Churg-Strauss syndrome, 190
Congophilic angiopathy, 64
Thoracic lymphadenopathy and disseminated *Pneumocystis carinii* in AIDS, 146

Cocaine
Cocaine (LE), 39
Crack abuse and asthma: A fatal combination (CR), 511
Crack cocaine: Ripples of a rock in a pond (ED), 487
Prevalence of marijuana (cannabis) use and dependence in cocaine dependence, 491
Public health consequences of crack cocaine, 493
Syphilis among parturients at an inner city hospital: Association with cocaine use and implications for congenital syphilis rates, 488

Colon
Carcinoma of the colon associated with systemic sclerosis (CR), 73

Commentaries
(see Editorial Commentaries)

Community health worker program
Community health worker program (LEN), 519

Computers
Computers aid in record keeping and can lower insurance costs (NF), 225

Congenital syphilis
  Rising incidence of congenital syphilis: Back to the future (ED), 485
  Syphilis among parturients at an inner city hospital: Association with cocaine use and implications for congenital syphilis rates, 488
Congophilic angiopathy
  Congophilic angiopathy (CPC), 64
Contact lenses
  Critical appraisal of contact lens disinfection, 12
Crack
  (see **Cocaine, Drug** abuse, **Substance** abuse)
Creativity
  Avicenna, problem solving, and creativity during sleep (LE), 378
Cross-cultural research
  Utility of cross-cultural health research (ED), 439
Cross-dressing
  Cross-dressing successfully treated with fluoxetine (LE), 566
Cyclosporin
  Low-dose cyclosporin reduces joint inflammation in rheumatoid arthritis (NF), 481
Cytomegalovirus
  AIDS and cytomegalovirus-related appendicitis (NF), 581
Deep venous thrombosis
  Deep vein thrombosis in pregnancy (CC), 309
  Deep venous thrombosis in patients with AIDS, 159
Diabetes
  Changing concepts of the pathogenesis of diabetic nephropathy (EP), 185
  Fibrous disease of the breast in juvenile diabetes, 356, (LE), 617
Diagnostic testing
  Diagnosing Alzheimer's disease with a biochemical assay (NF), 432
  New prenatal test yields results in one day (NF), 480
  Warning: Rapid strep A tests may not be reliable (NF), 582
Diphtheria
  Diphtheria and medical therapy in late 19th century New York City (HIST), 256
Disabilities
  Prevention of disabilities (LEN), 87
Disseminated *Pneumocystis carinii*
  Disseminated *Pneumocystis carinii* infection in the acquired immunodeficiency syndrome, 155
  Dissemination of *Pneumocystis* (ED), 121
  Thoracic lymphadenopathy and disseminated *Pneumocystis carinii* in AIDS (CPC), 146
Drug abuse
  (see also Cocaine, Substance abuse)
  Changes in AIDS risk behavior among intravenous drug abusers in New York City, 123
  Children of substance abusers in New York State, 179
  Cocaine (LE), 39
  Crack cocaine: Ripples of a rock in a pond (ED), 487
  Multiple addictions: Co-synchronous use of alcohol and drugs, 596
  Prevalence of marijuana (cannabis) use and dependence in cocaine dependence, 491
  Public health consequences of crack cocaine, 493
  Substance abuse during pregnancy (LE), 336
  Syphilis among parturients at an inner city hospital: Association with cocaine use and implications for congenital syphilis rates, 488
Drug nomenclature
  Trade and generic names of drugs (LE), 517
Drug testing
  Controversy grows over parallel track for experimental AIDS drug (NF), 169
Drug toxicity
  Use of free phenytoin levels in averting phenytoin toxicity (LE), 469
Dysmenorrhea
  Primary female syndromes: An update, 295
Editorial Commentaries
  Acquired immunodeficiency syndrome: The agenda for the 1990s, 115
  Adenocarcinoma of the cervix, 535
  Behavioral risks: Mother knows best, 346
  Bile duct carcinoma, 53
  Circumcision of the newborn, 231
  Crack cocaine: Ripples of a rock in a pond, 487
  Dissemination of *Pneumocystis*, 121
  Expanding the focus of human immunodeficiency virus prevention in the 1990s, 116
  Fusarium and the immunocompromised host: Liaisons dangeureuses, 586
  Granulomatous mastitis, 287
  Health and health care challenges for New York City in the 1990s, 51
  Hypersensitivity pneumonitis, 232
  Increasing role of primary care in the management of HIV-infected patients, 119
  Life, liberty, and the pursuit of happiness, 393
  Mainstreaming the minority and assumptions of rightful place, 283
  Mycobacterium tuberculosis: A disease of growing importance in urban population groups, 585
  New cover for the *New York State Journal of Medicine*, 345
  New York State Journal of Medicine enters its 90th year, 1
  Organ donation, 394
  Ovarian small cell carcinoma, 171
  Present and future importance of mortality statistics, 440
  Reevaluation of physician recredentialing, 531
  Rising incidence of congenital syphilis: Back to the future, 485
  Rural obstetric care and the family physician, 533
  Screening for breast cancer: Are physicians doing enough?, 285
  Small cell carcinoma of the ovary: A therapeutic challenge, 172
  Topical low-dose thrombolytic therapy, 2
  Uninvited guests: Cardiovascular foreign bodies, 347
  Utility of cross-cultural health research, 439
  Women, families, and academic medicine, 284
Emergency medical services
  Aeromedical emergency trauma services and mortality reduction in rural areas, 358
Empire State Medical, Scientific and Educational Foundation
  PRO contract (LE), 271
Endometrial carcinoma
  Endometrial carcinoma in five patients with breast cancer on tamoxifen therapy (CR), 207
Endometriosis
  Primary female syndromes: An update, 295
Eosinophilia-myalgia syndrome
  Eosinophilia-myalgia syndrome and L-tryptophan (CR), 512
  Eosinophilia myalgia syndrome (LEN), 380
Epilepsy
  NIH consensus paper on surgery for epilepsy (NF), 526
  Nintendo-induced epilepsy (NF), 387
Errata, 78, 518, 584, 618
Esophagus
  Hypercalcemia and carcinoma of the esophagus (NF), 387
  Leiomyosarcoma of the esophagus (CR), 371
Essay Reviews
  Country doctor, 472
  Practicing clinical preventive medicine, 79
  Unnatural causes: America's three leading killer diseases, 568
Essays in Pediatrics (series)
  Central nervous system malignancies in children, 601
  Changing concepts of the pathogenesis of diabetic nephropathy, 185
Estrogen
  Recall of about ten million generic estrogen tablets (NF), 226
Etidronate
  New drug to treat osteoporosis increases bone mass and reduces fractures (NF), 480
Family physicians
  Primary care obstetrics in rural Western New York: A multi-center case review, 537
  Rural obstetric care and the family physician (ED), 533
Fluconazole
  Two new infection-fighting drugs come to market (NF), 280
Fluoxetine
  Cross-dressing successfully treated with fluoxetine (LE), 566
Food and Drug Administration
  Food and Drug Administration halves dosage for AZT (NF), 168
Foot
  Open supramalleolar amputation of the infected foot: An underutilized, two-stage procedure (LE), 77
Foreign bodies
  Hypodermic needle embolization to the heart (CR), 368
  Uninvited guests: Cardiovascular foreign bodies (ED), 347
Fungal infections
  Disseminated infection caused by *Fusarium solani* in a patient with aplastic anemia (CR), 609
  Fusarium and the immunocompromised host: Liaisons dangeureuses (ED), 586

Two new infection-fighting drugs come to market (NF), 280

Fusarium solani
Disseminated infection caused by *Fusarium solani* in a patient with aplastic anemia (CR), 609
Fusarium and the immunocompromised host: Liaisons dangeureuses (ED), 586

Gallstone appendicitis
Gallstone appendicitis (LE), 77

Gardner-Diamond syndrome
Gardner-Diamond syndrome (CR), 373

Gary, Nancy E., MD
Interview with Nancy E. Gary, MD, a medical school dean, 321

Gas warfare
Gas warfare: Military-medical responsiveness of the Allies in the Great War, 1914–1918 (HIST), 499

Generic drugs
FDA supports generic drug use (NF), 387
Recall of about ten million generic estrogen tablets (NF), 226
Trade and generic names of drugs (LE), 517

Gene therapy
Balloon angioplasty finds application in gene therapy (NF), 582
Gene therapy ushers in an era of hope (NF), 581

Geriatrics
New Merck manual addresses geriatrics (NF), 481

Goiter
Superior vena cava syndrome and right pleural effusion due to giant goiter (CR), 467

Growth hormone
Growth hormone therapy may reverse some age-related bodily changes (NF), 481

Hallucinations
Visual hallucinations as grief reaction without the Charles Bonnet syndrome (LE), 216

Harvard Report
Harvard Report highlights (LEN), 338

Hazardous waste
History of infectious waste legislation and regulation and their impact on hospitals in New York City, 400

Health care
Allocation of scarce medical resources (committee report), 552
Current hospital crisis in New York City and policy options for resolving it, 247
Health and health care challenges for New York City in the 1990s (ED), 51, (LE), 565

Heart disease
Baseline survey for the Healthy Heart Program (LEN), 164

Heart transplants
He had a change of heart, 253

Hemoptysis
Use of a Swan-Ganz catheter and streptokinase in the management of massive hemoptysis (CR), 33, (LE), 270

Heparin
Low-dose subcutaneous heparin therapy (LE), 39

Hepatitis C
Blood donors will now be tested for hepatitis C (NF), 432

Hernia
Paraesophageal hernia (LE), 214

Hickman catheter
Superior vena cava syndrome associated with a Hickman catheter (CR), 208

Hispanics
Cancer incidence among NYC Hispanics 1982–1985 (LEN), 44
Mortality in the Hispanic population of Suffolk County, New York, 442
Present and future importance of mortality statistics (ED), 440
Utility of cross-cultural health research (ED), 439

Histoplasmosis
Disseminated histoplasmosis in AIDS: Clinicopathologic features in seven patients from a non-endemic area, 234

History of medicine
History of medicine archives in New York State, 23

History of Medicine (series)
Diphtheria and medical therapy in late 19th century New York City, 256
Gas warfare: Military-medical responsiveness of the Allies in the Great War, 1914–1918, 499
Henry Keller, MD, of New York and the Alyn Hospital in Jerusalem, 201
History of the New York Society of Tropical Medicine, 450
History of the Ray Brook State Tuberculosis Hospital, 406

Home health care
Residential and home health care for people with AIDS and HIV-related illnesses (LEN), 43

Homelessness
Drug resistant tuberculosis among the homeless in New York City, 351
Health problems of the homeless (NF), 226

Hormones
Breast cancer risk related to long term hormone replacement therapy (NF), 630
Recall of about ten million generic estrogen tablets (NF), 480

Hospitals
Current hospital crisis in New York City and policy options for resolving it, 247
Eliminating the requirement for medical libraries in hospitals (LE), 76
History of infectious waste legislation and regulation and their impact on hospitals in New York City, 400
Need for medical libraries in hospitals (LE), 420
Prevalence and characteristics of violent patients in a general hospital, 591

Human immunodeficiency virus
Erosive arthritis, atypical psoriasis, and *Pneumocystis carinii* pneumonia in an HIV-positive man, 157
Expanding the focus of human immunodeficiency virus prevention in the 1990s (ED), 116
HIV infected hospital patients in New York State: The development of longitudinal information from a hospital discharge data system, 238
Increasing role of primary care in the management of HIV-infected patients (ED), 119
Large pericardial effusion in a patient with human immunodeficiency virus type 1 (CR), 612
Lessons from New York City on HIV/AIDS in adolescents, 143
Multifocal human papillomavirus infections of the genital tract in HIV seropositive women (LE), 162
Perinatally acquired HIV infection: AIDS' youngest victims (NF), 169
Position paper of the Associated Medical Schools of New York on HIV testing and access to treatment, 496
Residential and home health care for people with AIDS and HIV-related illnesses (LEN), 43
Seroprevalence survey for human immunodeficiency virus antibody in mentally retarded adults, 139
Thoracic lymphadenopathy and disseminated *Pneumocystis carinii* in AIDS (CPC), 146

Human papillomavirus
Multifocal human papillomavirus infections of the genital tract in HIV seropositive women (LE), 162

Hydergine
Hydergine shown ineffective in Alzheimer's disease (NF), 526

Hypercalcemia
Hypercalcemia and carcinoma of the esophagus (NF), 387
Para-endocrine hypercalcemia and ovarian small cell carcinoma (CR), 206

Hypersensitivity pneumonitis
Hypersensitivity pneumonitis (ED), 232
Hypersensitivity pneumonitis associated with the use of ultrasonic humidifiers (CR), 263

Hypertension
Study reveals relationship between placental size, birth weight, and hypertension in adult life (NF), 527

Hyponatremia
Isolated adrenocorticotrophic hormone deficiency presenting as chronic hyponatremia (CR), 210

Hypothyroidism
Prevalence of subclinical hypothyroidism in adults with low-normal blood thyroxine levels, 541

Ibuprofen
Risk of acute ibuprofen overdose remains low (NF), 432

Immunization
Changes in state immunization requirements (LEN), 338
WHO embarks on vaccination campaign (NF), 581

Immunocompromise
Disseminated infection caused by *Fusarium solani* in a patient with aplastic anemia (CR), 609
Fusarium and the immunocompromised host: Liaisons dangeureuses (ED), 586

Impotence
Impotence: A single symptom with different causes (LE), 420

Infected aortitis
Infected aortitis masquerading as bronchogenic carcinoma (CR), 415

Infectious waste
History of infectious waste legislation and regulation and their impact on hospitals in New York City, 400

Infusaport
Tumor metastasis to an infusaport site (LE), 216

Injuries
- Bicycle injuries (LEN), 426
- Injury mortality in New York State (LEN), 382

Intestinal perforation
- Intestinal perforation/fistulization complicating temporal arteritis (LE), 270

Ivermectin
- Ivermectin shown effective against river blindness (NF), 629

Kaposi's sarcoma
- Upper airway obstruction due to Kaposi's sarcoma in the acquired immunodeficiency syndrome (CR), 613

Keller, Henry, MD
- Henry Keller, MD, of New York and the Alyn Hospital in Jerusalem (HIST), 201

Lead poisoning
- Lead poisoning during home renovation (LE), 215

Leads from *Epidemiology Notes* (series)
- AIDS morbidity and mortality, 572
- Baseline survey for the Healthy Heart Program, 164
- Benzodiazepines: Additional effects of the triplicate program, 273
- Benzodiazepines: Prescribing declines under triplicate program, 218
- Bicycle injuries, 426
- Birth outcomes in New York State, 619
- Birth outcomes in New York State (part II), 42
- Breast cancer detection and counseling program, 85
- Cancer incidence among NYC Hispanics 1982–1985, 44
- Cervical cancer in New York State, 521
- Changes in state immunization requirements, 338
- Community health worker program, 519
- Eosinophilia myalgia syndrome, 380
- Harvard Report highlights, 338
- Injury mortality in New York State, 382
- Mammography attitudes and awareness, 339
- Pesticide poisoning: A reportable disease in New York State, 621
- Poison control, 474
- Prevention of disabilities, 87
- Residential and home health care for people with AIDS and HIV-related illnesses, 43
- Safe use of capillary blood sampling devices, 574
- Screening for congenital syphilis begins, 166
- Skin cancer prevention, 520
- Time trends in breast cancer incidence, 473
- Toy safety, 166
- *Vibrio vulnificus* in raw oysters, 87
- Vital statistics of New York State, 85

Leeches
- Medical leeches making a comeback (NF), 630

Left atrial ball thrombus
- Systemic embolization from left atrial ball thrombus: The case for early surgical intervention (CR), 74

Leiomyosarcoma
- Leiomyosarcoma of the esophagus (CR), 371

Letters to the Editor
- AIDS legislation needed, 163
- Alcohol and public health: Implications for New York State, 41
- An alma mater by any other name is an alma mater, 215
- Animal experimentation for medical research, 214
- Authors' academic affiliation, 377
- Avicenna, problem solving, and creativity during sleep, 378
- Capgras' syndrome, 470
- Cervical tuberculosis, 270
- Claims-made policies and physician employment contracts, 378
- Cocaine, 39
- Combining parenthood and practice, 616
- Cross-dressing successfully treated with fluoxetine, 566
- Defining brain death, 517
- Eliminating the requirement for medical libraries in hospitals, 76
- Fetal heart rate changes during general anesthesia, 272
- Fibrous disease of the breast in juvenile diabetes, 617
- Gallstone appendicitis, 77
- Gender and sex, 566
- Health and health care challenges for New York City in the 1990s, 565
- Impotence: A single symptom with different causes, 420
- Influence of malpractice fears on the practice of medicine, 271
- Intestinal perforation/fistulization complicating temporal arteritis, 270
- Introduction to a current bibliography on women in medicine, 335
- Lead poisoning during home renovation, 215
- Low-dose subcutaneous heparin therapy, 39
- Make way for tomorrow, 469
- Melville's "Bartleby the Scrivener", 422
- Multifocal human papillomavirus infections of the genital tract in HIV seropositive women, 162
- Need for medical libraries in hospitals, 420
- Neglected resource, A, 517
- New health code regulations concerning postgraduate medical education in New York State, 39
- New *Journal* cover, 615
- New York State Department of Health's Part 405 regulations, 565
- Open supramalleolar amputation of the infected foot: An underutilized, two-stage procedure, 77
- Paraesophageal hernia, 214
- Physician as lobbyist, 424
- Physician recredentialing, 76
- Physicians needed to work with AIDS patients, 163
- Premenstrual syndrome, 615
- PRO contract, 271
- Resolution of cavitary *Pneumocystis carinii* pneumonia, 162
- Responses to "Parenthood and practice", 334
- Reversible hypoxic cerebral edema in severe hemolytic crisis, 518
- Student participation in mixed gender sports competition, 76
- Substance abuse during pregnancy, 336
- Trade and generic names of drugs, 517
- Tumor metastasis to an infusaport site, 216
- Turbulent years, The, 379
- UNY*Care, 377
- Use of a Swan-Ganz catheter and streptokinase in the management of massive hemoptysis, 270
- Use of free phenytoin levels in averting phenytoin toxicity, 469
- Visual hallucinations as grief reaction without the Charles Bonnet syndrome, 216

Leukemia
- Leukemia in men may be related to smoking (NF), 388

Libraries
- Eliminating the requirement for medical libraries in hospitals (LE), 76

Licensure requirements
- Overview of the activities of the New York State Board for Medicine, 545

Lobbying
- Physician as lobbyist (LE), 424

Lofgren's syndrome
- Lofgren's syndrome (acute sarcoidosis) sine erythema nodosum mimicking acute rheumatoid arthritis (CR), 463

Lung cancer
- Carcinoma of the lung presenting with spontaneous pneumothorax (CR), 611
- Lung cancer rates decreasing for young people in US (NF), 46

Lyme disease
- Diagnosing Lyme disease in seronegative patients (NF), 280
- Lyme disease has become a "diagnosis of exclusion" (NF), 432
- Report of a specific and sensitive test for Lyme disease: Detection of a single organism is possible (NF), 225

Malpractice
- Influence of malpractice fears on the practice of medicine (LE), 271

Mammography
- Breast cancer screening: Changing physician practices and specialty variation, 288
- Is lightscanning a viable alternative to mammography for detecting breast cancer? (NF), 341
- Mammography attitudes and awareness (LEN), 339
- Screening for breast cancer: Are physicians doing enough? (ED), 285

Marijuana
- Prevalence of marijuana (cannabis) use and dependence in cocaine dependence, 491

Mastitis
- Granulomatous mastitis (ED), 287

Medicaid
- Number of AIDS patients on Medicaid is increasing (NF), 581

Medical archives
- History of medicine archives in New York State, 23

Medical education
- Interview with Nancy E. Gary, MD, a medical school dean, 321
- New health code regulations concerning postgraduate medical education in New York State (LE), 39
- Trends in medical school enrollment (NF), 526
- Women, families, and academic medicine (ED), 284

Medical liability insurance
- Claims-made policies and physician employment contracts (LE), 378

Medical libraries
- Eliminating the requirement for medical libraries in hospitals (LE), 76
- Need for medical libraries in hospitals (LE), 420

Medical research
- Animal experimentation for medical research (LE), 214
- Mortality in the Hispanic population of Suffolk County, New York, 442
- Present and future importance of mortality statistics (ED), 440
- Utility of cross-cultural health research (ED), 439

Medical schools
- Trends in medical school enrollment (NF), 526

Medical Society of the State of New York
- Allocation of scarce medical resources (committee report), 552
- Neglected resource, A (LE), 517

Medical Society of the State of New York, House of Delegates
- Board of Trustees 1989–1990, 98
- Convention Committees, 104
- Councilors 1989–1990, 98
- House Committee on Bylaws, 106
- 1990 House of Delegates Members, 100
- Nominating Committee, 105
- Officers 1989–1990, 97
- Reference Committees, 104
- Schedule of sessions, 95

Medical waste
- History of infectious waste legislation and regulation and their impact on hospitals in New York City, 400

Medicare
- Simulated effects of the planned Medicare Fee Schedule (NF), 527

Medicine
- Allocation of scarce medical resources (committee report), 552
- Combining parenthood and practice (LE), 616
- Influence of malpractice fears on the practice of medicine (LE), 271
- Interview with Nancy E. Gary, MD, a medical school dean, 321
- Introduction to a current bibliography on women in medicine (LE), 335
- Mainstreaming the minority and assumptions of rightful place (ED), 283
- Make way for tomorrow (LE), 469
- Neglected resource, A (LE), 517
- Parenthood and practice, 326
- Perspectives on women physicians in New York State: Past, present, and future, 317
- Physician as lobbyist (LE), 424
- Responses to "Parenthood and practice" (LE), 334
- Senior women physicians: The question of retirement, 292
- Turbulent years, The (LE), 379
- Women, families, and academic medicine (ED), 284
- Women in medicine since Flexner, 302

Melville
- Melville's "Bartleby the Scrivener" (LE), 422
- Melville's "Bartleby the Scrivener": A case study, 17

Mentally retarded
- Seroprevalence survey for human immunodeficiency virus antibody in mentally retarded adults, 139

Metacarpophalangeal arthropathy
- Manual labor metacarpophalangeal arthropathy in a baker (CR), 268

Metaproterenol
- Metaproterenol inhaler taken off market (NF), 46

Methylprednisolone
- Treatment of acute spinal cord injury with methylprednisolone shows promise (NF), 387

Migraines
- Side effects of beta blockers in patients with migraines (NF), 388

Miscellaneous
- An alma mater by any other name is an alma mater (LE), 215
- Authors' academic affiliation (LE), 377
- Combining parenthood and practice (LE), 616
- Gender and sex (LE), 566
- He had a change of heart, 253
- Life, liberty, and the pursuit of happiness (ED), 393
- Maintaining serenity in chronic illness, 366
- Make way for tomorrow (LE), 469
- Medical student's dilemma, 154
- Melville's "Bartleby the Scrivener": A case study, 17
- Parenthood and practice, 326
- Passover matzoh and chocolate Easter eggs, 196
- Responses to "Parenthood and practice" (LE), 334
- Turbulent years, The (LE), 379

Mortality statistics
- AIDS morbidity and mortality (LEN), 572
- Mortality in the Hispanic population of Suffolk County, New York, 442
- Present and future importance of mortality statistics (ED), 440

Motor vehicle accidents
- Behavioral risks: Mother knows best (ED), 346
- Death takes a ride: Alcohol-associated single vehicle fatalities revisited, 349

Mycobacterium tuberculosis
- *Mycobacterium tuberculosis:* A disease of growing importance in urban population groups (ED), 585

Needlestick injuries
- New "sharps" disposal system reduces waste (NF), 526
- Safe use of capillary blood sampling devices (LEN), 574

Neurofibromatosis-2
- Neurocutaneous manifestations of neurofibromatosis-2 (CR), 513

New York City
- Cancer incidence among NYC Hispanics 1982–1985 (LEN), 44
- Changes in AIDS risk behavior among intravenous drug abusers in New York City, 123
- Current hospital crisis in New York City and policy options for resolving it, 247
- Diphtheria and medical therapy in late 19th century New York City (HIST), 256
- Drug resistant tuberculosis among the homeless in New York City, 351
- Health and health care challenges for New York City in the 1990s (ED), 51, (LE), 565
- History of infectious waste legislation and regulation and their impact on hospitals in New York City, 400
- Lessons from New York City on HIV/AIDS in adolescents, 143

New York Society of Tropical Medicine
- History of the New York Society of Tropical Medicine (HIST), 450

New York State
- AIDS behind bars: Epidemiology of New York State prison inmate cases, 1980–1988, 133
- Alçohol and public health: Implications for New York State (LE), 41
- Assessing the damage from cigarette smoking in New York State, 56
- Assessment of AIDS knowledge in selected New York State sexually transmitted disease clinics, 126
- Baseline survey for the Healthy Heart Program (LEN), 164
- Birth outcomes in New York State (LEN), 619
- Birth outcomes in New York State (part II) (LEN), 42
- Cervical cancer in New York State (LEN), 521
- Changes in state immunization requirements (LEN), 338
- Children of substance abusers in New York State, 179
- Community health worker program (LEN), 519
- Current trends in routine newborn male circumcision in New York State, 243
- History of medicine archives in New York State, 23
- HIV infected hospital patients in New York State: The development of longitudinal information from a hospital discharge data system, 238
- Injury mortality in New York State (LEN), 382
- New health code regulations concerning postgraduate medical education in New York State (LE), 39
- Perspectives on women physicians in New York State: Past, present, and future, 317
- Pesticide poisoning: A reportable disease in New York State (LEN), 621
- Primary care obstetrics in rural Western New York: A multi-center case review, 537
- Public health benefit of increasing tobacco taxes in New York State, 174
- UNY•Care (LE), 377
- Vital statistics of New York State (LEN), 85

New York State Board for Medicine
- Overview of the activities of the New York State Board for Medicine, 545

New York State Department of Health
- New York State Department of Health's Part 405 regulations (LE), 565

New York State Journal of Medicine
- New cover for the *New York State Journal of Medicine* (ED), 345
- New *Journal* cover (LE), 615
- *New York State Journal of Medicine* enters its 90th year (ED), 1
- Our referees, 83

New York State Triplicate Prescription Program
Benzodiazepines: Additional effects of the triplicate program (LEN), 273
Nintendo
Nintendo-induced epilepsy (NF), 387
Nutrition
Premenstrual syndrome and diet (NF), 341
Obituaries
Altschul, Eric G., 227
Amodio, Frank John, 91
Arbuse, David Irving, 482
Armistead, George C., 482
Augenfeld, Robert, 227
Autuori, Giovanni, 227
Badanes, Maynard B., 389
Ballantyne, Lowyd, Jr., 91
Barker, Leslie Paxton, 482
Batcheller, Delmer E., 227
Bates, Edgar Hodgins, 434
Beck, Isabel, 528
Bellows, Emma Louise, 389
Bennett, Ivan L., 528
Bergmann, Annelies, 482
Bergman, Philip Sandler, 528
Berl, Alfred, 434
Biempica, Luis, 583
Biezunski, Arthur, 227
Biren, Charles Maxwell, 434
Biris, Lawrence John, 389
Bleicher, Max, 389
Block, Irving Rosen, 91
Blume, Hyman, 434
Blumenthal, Mortimer J., 583
Blum, Lester, 91
Bodkin, Laurence Griffin, 583
Bologna, Angelo Robert, 227
Borkow, Ben Albert, 434
Bovino, Dominic Anthony, 91
Breed, Eben, 389
Brill, Gerald, 227
Brill, Henry, 482
Brodey, Adrian, 434
Brown, DeWitt Clinton, 434
Brown, Jesse Murray, 631
Burke, Matthew W., 389
Burness, Leonard, 227
Busino, William A., 434
Butsch, Winfield L., 91
Caddy, Glenna Garratt, 389
Cagan, Seymour Harvey, 227
Callin, Lynn R., 437, 482
Carrington, Ulric Irving, 528
Chalef, Rita Mendelson, 434
Chester, Ralph V., 482
Chiaramonte, Joseph T., 227
Chwatsky, David N., 227
Codik, Nicholas Robert, 227
Coffey, Edward Lyman, 482
Cohen, Murray, 91
Coletti, Jeff Joseph, 227
Coombs, Francis Place, 227
Coon, William McIntosh, 227
Coriale, Arthur S., 583
Cristo, Carmen, 389
Crouse, Irving I., 528
Cunningham, John E., 91
Dalven, Joseph, 389
Davison, Selvan, 583
De Angelis, Anthony M., 631
de Lalla, Emanuel, 227
Del Bello, Louis S., 528
de Marchis, Eugenio, 228
de Pass, Skottowe W., 228
De Respinis, Benedict J., 528
De Rostaing, Caesar, 583
Derrico, Charles John, 389
Deutsch, Alfred, 482
Dickinson, Selden Jerome, 228
Diefenbach, William C. L., III, 482
DiGiacoma, Michael, 91
Dineen, Peter, 91
Dlugash, Joel, 228
Dollin, Martin, 91
Drexler, Bernard, 91
Dugan, William D., 434
Duschnitz, Leo, 528
Duszynski, Diana Olga, 228
Dybich, Myron John, 91
Dygert, Joseph E., 228
Edwards, Walter Davis, 91
Ehrlich, Walter F., 228
Einhorn, William Lawrence, 434
Eissler, Ruth Selke, 91
Engler, William, 583
Ephraim, Alfred John, 528
Eppers, Edward Henry, 91
Faga, Aldo Rocco, 92
Fairbank, Matthew Elmer, 228
Falk, Joseph George, 389
Feldman, Abraham Isidor, 92
Feldman, Benjamin, 434
Fenstermacher, Walter A., 434
Filippo, Rosario P., 94
Finkelstein, James Howard, 228
Fischer, Arnold, 389
Fitch, Wilmer Price, 583
Fleck, Henry, 434
Foster, Charles D., 228
Friedland, Martin, 92
Friedman, Marcel, 228
Frisk, Raymond E., 528
Fuchs, Seymour, 482
Fuerst, Harold Theodore, 434
Galdston, Iago, 228
Garvin, William John, 228
Gatti, Peter Joseph, 434
Gattner, Julius, 389
Gelber, Louis Jack, 435
Geller, Alan Charles, 631
George, William B., 631
Gerchick, Elias H., 435
Giannattasio, Rudolph C., 92
Gochuico, Ramon Garcia, 92
Goldensohn, Sidney S., 228
Golding, George Theodore, 435
Gold, Lazar L., 435
Goldmark, Carl, 228
Goldstein, Lester B., 92
Goliger, Jack Jason, 92
Gordon, Arthur, 229
Gordon, Frederick, 389
Grady, John Francis, 229
Grand, Milton J. H., 229
Greenacre, Phyllis, 92
Greenbaum, Esther, 229
Greenberg, Morris E., 435
Green, David, 482
Green, James Louis, 482
Grobstein, Herman Jack, 92
Gumport, Stephen L., 92
Hamel, John Henry, 528
Harris, Earle Harold, 631
Harrison, Maxwell, 435
Hartley, Arthur Harold, 92
Hartmann, Hans A., 583
Hasan, Zubaida, 435
Hayos, Leslie L., 92
Healy, Robert J., 435
Hecht, Paul, 229
Heldmann, Walter T., 229, 391
Heller, Marie H., 631
Helpern, Herman Goodman, 435
Helwith, Edward E., 92
Herold, Lewis Jacob, 229
Herz, Karl, 631
Heslin, John Edward, 435
Hochman, Noemi G., 583
Hoffmann, Karl F. G., 389
Huber, Franklyn Alvin, 483
Hunter, Francis Robert, 435
Hyman, Joseph, 435
Izzo, Anthony J., 229
Jacobsen, Arnold, 92
Jacobsen, A. Wilmot, 229
Jacobs, Thomas P., 92
Jacoby, Emma I., 389
Jennings, Kenneth G., 528
Kahane, Abraham, 229
Kane, Edwin P., 389
Kase, Stuart, 435
Katz, Alexander A., 229
Katz, J. Harry, 92
Kaywin, Louis, 390
Kenzie, Joseph F., 229
Kerr, Andrew D., 92
Khilnani, Mansho Tolaram, 229
Kirby, Kathryn, 93
Kleinhandler, William W., 93
Klibanski, Aron, 483
Kogel, Marcus D., 93
Kopins, Howard J., 93
Koplon, Burton Stanley, 528
Kotsilimbas, Dimitrios G., 390
Kreis, David John, Jr., 93
Kresky, Beatrice, 93
Kroll, Rolf, 229
Kulka, Ernest W., 435
Ku, Yuan Kuang, 528
Landsberg, J. Walter, 93
La Russo, Anthony, 390
Leavitt, Alexander, 390
Lehv, Saul P., 583
Leighton, Benjamin, 583
Levine, Benjamin Bernard, 583
Lichtenstein, Heinz, 390
Lichtenstein, Stefanie, 631
Lieberman, Samuel L., 631
Lippman, Abbott, 435
Loftus, James Bernard, 390
Logaridis, Teologos, 483
Lustig, Saul, 390
Lutrin, Moisey, 483
Maltinsky, Maurice M., 390
Maniscalco, Anthony E., 229
Markham, Mary R. Headlee, 483
Marshall, Philip, 229
Mascia, Armond V., 435
Matthews, Edward J., 528
Mautone, Louis Joseph, 631
McCauley, John C., 390
Mc Cuaig, Duncan R., 583
Menter, Philip, 229
Merin, Jessie L. Gillespie, 93
Messina, Philip Vincent, 93
Miller, Helen Lovena, 528
Miller, Israel, 230
Mishuris, Bernard E., 631
Mitty, Virginia Connolly, 93
Morelli, Henry Ernest, 390
Moskowitz, Eugene, 631
Mott, Walter Charles, 93
Muehlbauer, Michael A., 436
Munteanu, John Paul, 230
Murphy, James William, 390
Nagler, Aron, 390
Nathanson, Joseph Norman, 230

Negrete, Francisco, 528
New, Bertrand Latimer, 483
Norris, Benjamin F., 631
Nugent, Paul Fordham, 93
Oberlander, Oscar W., Jr., 631
O'Brien, James J., 93
Occhino, Nicholas R., 529
Peters, Lloyd Calvin, 583
Pierce, Arte I., 583
Planer, Charles Kirby, 93
Pleshette, Norman P., 584
Rack, Leonard, 230
Radna, Rudolf, 93
Rakov, Harold L., 230
Rappaport, Hyman, 436
Rattner, Irving N., 390
Ravasz, Thomas James, 436
Raymond, William Henry, 93
Richter, R. Virginia F., 390
Ritzmann, Albert Jay, 584
Roberts, Norbert Joseph, 631
Robertson, Douglas H., 436
Robinson, Edward J., 390
Rose, George, 584
Rosenfeld, Sidney, 230
Rubin, Mitchell I., 436
Rubinstein, Benjamin B., 390
Rubinstein, Robert Israel, 436
Rubler, Shirley, 483
Rusk, Howard A., 93
Saffer, Sidney Hugh, 230
Samet, Sydney, 93
Sanford, Donald Barton, 390
Saperstein, Abraham N., 94
Sarot, Irving Arthur, 436
Sawyer, Philip, 631
Saxe, Earl, 436
Scarantino, Patrick P., 584
Schall, Edward, 632
Scharf, Nathan, 584
Scheuer, Lewis Alfred, 483
Schmeiser, Stephen J., Jr., 436
Schnabel, Frederick G., 391
Schwabe, Edward Ludwig, 483
Schwartzman, Joseph, 632
Schwyzer, Arnold Gustav, 391
Scimeca, Anthony Adolf, 230
Scribner, Lewis F., Jr., 436
Segal, Harry L., 483
Seley, Samson A., 94
Serio, Joseph Charles, 483
Shaver, Carrol Joslin, 391
Shulman, Simon, 632
Shusterov, Vladimir L., 230
Siegel, Irving, 391
Silberg, Benjamin, 584
Snodgrass, John James, 584
Snow, Shirley R., 230
Sobel, Ben, 529
Som, Max Leon, 436
Sonsire, James Michael, 436
Spiegelman, Harry, 94
Spitzer, Joseph Maurice, 584
Spotkov, David Haskell, 391
Steigbigel, Samuel, 584
Steinharter, Rudolf H., 230
Stein, Simon, 230
St Mary, John Taylor, 632
Straitiff, Max Charles, 436
Strongin, Harry, 94
Symonds, Francis C., 584
Szekely, Zsolt, 584
Tagliamonti, Joseph Alan, 584
Tanney, Allen Donald, 437
Taterka, Harvey Burton, 584
Terner, Felix, 391
Thaler, Max, 94
Thaler, Nathan, 584
Toll, William George, 94
Trattler, Arnold Joseph, 391
Trattler, Samuel Carlyle, 529
Traube, Sylvia Gould, 94
Trigg, Harold L., 632
Tuby, Robert, 94
Turell, Robert, 483
Turner, Nathan, 230
Van Fenema, Greta Herta, 230
Veprovsky, Edward C., 391
Vinicor, Henry, 437
Vogel, Harold T., 483
Volpe, Michael F., 529
Wainapel, David, 94
Wallis, Sue B., 94
Wallsh, Rita G., 437
Watson, Robin Caird, 632
Weidman, Abraham Isak, 391
Weinberger, Hugo, 391
Weiss, Fred Morris, 529
Wilson, Robert G., 437
Winer, Nahum J., 437
Wolf, Morris J., 437
Wood, Rodney Thorp, 230
Wright, Myron, 529
Yamin, Alfonso, 437
Yohalem, Stephen Bennett, 483
York, George Eayres, 437
Zaris, David, 632
Zimmerman, Morris, 632

Obituary notices
  An alma mater by any other name is an alma mater (LE), 215
Obstetrics
  Primary care obstetrics in rural Western New York: A multi-center case review, 537
  Rural obstetric care and the family physician (ED), 533
Onchocerciasis
  Ivermectin shown effective against river blindness (NF), 629
Oral contraceptives
  Antibiotics and other drugs may inhibit effectiveness of oral contraceptives (NF), 341
Organ transplantation
  Organ donation (ED), 394
  Pattern of organ donation in a large urban center, 396
Osteoporosis
  Attempt to predict bone mass in perimenopausal women using only clinical data (NF), 342
  New drug to treat osteoporosis increases bone mass and reduces fractures (NF), 480
  Osteoporosis: Does sodium fluoride strengthen bone? (NF), 342
  Osteoporosis: New therapies may add bone mass (NF), 342
Ovarian cancer
  Ovarian small cell carcinoma (ED), 171
  Para-endocrine hypercalcemia and ovarian small cell carcinoma (CR), 206
  Small cell carcinoma of the ovary: A therapeutic challenge (ED), 172
Oysters
  Vibrio vulnificus in raw oysters (LEN), 87
Pancreatectomy
  Total pancreatectomy with celiac artery occlusion (CR), 416

Paraesophageal hernia
  Paraesophageal hernia (LE), 214
Part 405 regulations
  New York State Department of Health's Part 405 regulations (LE), 565
Pericardial effusion
  Large pericardial effusion in a patient with human immunodeficiency virus type 1 (CR), 612
Pesticide poisoning
  Pesticide poisoning: A reportable disease in New York State (LEN), 621
Phenytoin
  Use of free phenytoin levels in averting phenytoin toxicity (LE), 469
Physician employment contracts
  Claims-made policies and physician employment contracts (LE), 378
Physician recredentialing
  Physician recredentialing (LE), 76
  Reevaluation of physician recredentialing (ED), 531
Pneumocystis carinii
  Disseminated *Pneumocystis carinii* infection in the acquired immunodeficiency syndrome, 155
  Dissemination of *Pneumocystis* (ED), 121
  Erosive arthritis, atypical psoriasis, and *Pneumocystis carinii* pneumonia in an HIV-positive man, 157
  Resolution of cavitary *Pneumocystis carinii* pneumonia (LE), 162
  Thoracic lymphadenopathy and disseminated *Pneumocystis carinii* in AIDS (CPC), 146
Pneumonia
  Erosive arthritis, atypical psoriasis, and *Pneumocystis carinii* pneumonia in an HIV-positive man, 157
  Resolution of cavitary *Pneumocystis carinii* pneumonia (LE), 162
Pneumonitis
  Hypersensitivity pneumonitis (ED), 232
  Hypersensitivity pneumonitis associated with the use of ultrasonic humidifiers (CR), 263
Pneumothorax
  Carcinoma of the lung presenting with spontaneous pneumothorax (CR), 611
Poisoning
  Pesticide poisoning: A reportable disease in New York State (LEN), 621
  Poison control (LEN), 474
Pott's disease
  Pott's disease in urban populations: A report of five cases and a review of the literature, 588
Pregnancy
  Birth outcomes in New York State (LEN), 619
  Community health worker program (LEN), 519
  Deep vein thrombosis in pregnancy (CC), 309
  Effects of prenatal alcohol exposure on child development (NF), 388
  Fetal heart rate changes during general anesthesia (LE), 272
  New prenatal test yields results in one day (NF), 480
  Substance abuse during pregnancy (LE), 336
  Unexpected, unattended deliveries (CR), 330
  Use of multivitamin/folic acid supplements during first weeks of pregnancy can help prevent neural tube defects (NF), 225

Premenstrual syndrome
- Premenstrual syndrome (LE), 615
- Premenstrual syndrome and diet (NF), 341
- Primary female syndromes: An update, 295

Preventive medicine
- Practicing clinical preventive medicine (ER), 79
- Prevention of disabilities (LEN), 87

Prison inmates
- AIDS behind bars: Epidemiology of New York State prison inmate cases, 1980–1988, 133

Professional review organizations
- PRO contract (LE), 271

Psoriasis
- Erosive arthritis, atypical psoriasis, and *Pneumocystis carinii* pneumonia in an HIV-positive man, 157

Public health
- *Mycobacterium tuberculosis:* A disease of growing importance in urban population groups (ED), 585
- Public health consequences of crack cocaine, 493

Pulmonary embolism
- Local low-dose infusion of streptokinase for massive pulmonary embolism when systemic thrombolysis is contraindicated (CR), 32

Radial artery cannulation
- Volar proximal skin necrosis after radial artery cannulation (CR), 375

Rationing
- Allocation of scarce medical resources (committee report), 552

Recredentialing
(see Physician recredentialing)

Renal hemorrhage
- Spontaneous renal hemorrhage secondary to choriocarcinoma (CR), 418

Retirement
- Senior women physicians: The question of retirement, 292

Rheumatoid arthritis
- Lofgren's syndrome (acute sarcoidosis) sine erythema nodosum mimicking acute rheumatoid arthritis (CR), 463
- Low-dose cyclosporin reduces joint inflammation in rheumatoid arthritis (NF), 481

River blindness
- Ivermectin shown effective against river blindness (NF), 629

Rural health care
- Aeromedical emergency trauma services and mortality reduction in rural areas, 358
- Alaskan community health aide scheme: A successful rural health program, 61
- Primary care obstetrics in rural Western New York: A multi-center case review, 537
- Rural obstetric care and the family physician (ED), 533

Ryan White Comprehensive AIDS Resources Emergency Act
- Federal funds allocated for clinical care of AIDS patients (NF), 526

Salmonella arthritis
- Salmonella arthritis (CR), 266

Sarcoidosis
- Lofgren's syndrome (acute sarcoidosis) sine erythema nodosum mimicking acute rheumatoid arthritis (CR), 463
- Sarcoidosis of the breast coexisting with mammary carcinoma (CR), 331

Sexually transmitted diseases
- Assessment of AIDS knowledge in selected New York State sexually transmitted disease clinics, 126
- Rising incidence of congenital syphilis: Back to the future (ED), 485

"Sharps" disposal
- New "sharps" disposal system reduces waste (NF), 526
- Safe use of capillary blood sampling devices (LEN), 574

Signet ring cell carcinoma
- Primary signet ring cell carcinoma of the uterine cervix (CR), 559

Skin cancer
- Skin cancer prevention (LEN), 520

Small bowel
- Intestinal obstruction secondary to bezoars (CR), 561
- Small bowel injuries following blunt abdominal trauma: Early recognition and management, 446

Small cell carcinoma
- Ovarian small cell carcinoma (ED), 171
- Para-endocrine hypercalcemia and ovarian small cell carcinoma (CR), 206
- Small cell carcinoma of the ovary: A therapeutic challenge (ED), 172

Smoking
- Assessing the damage from cigarette smoking in New York State, 56
- Cigarette smoking has a reversible, adverse effect on olfactory function (NF), 281
- Intervention to reduce the sale of cigarettes to minors, 54
- Leukemia in men may be related to smoking (NF), 388
- Magazines that do not accept tobacco advertising, 414
- Public health benefit of increasing tobacco taxes in New York State, 174

South Carolina
- Extent of underreporting of AIDS documented in South Carolina (NF), 168

Spinal cord injury
- Treatment of acute spinal cord injury with methylprednisolone shows promise (NF), 387

Spinal tuberculosis
- Pott's disease in urban populations: A report of five cases and a review of the literature, 588

Spinal tumors
- Metastatic cancer of the spinal intradural extramedullary space (CR), 562

Sports medicine
- Student participation in mixed gender sports competition (LE), 76

Spring-loaded lancets
- Safe use of capillary blood sampling devices (LEN), 574

Streptococcal pharyngitis
- Warning: Rapid strep A tests may not be reliable (NF), 582

Streptokinase
- Local low-dose infusion of streptokinase for massive pulmonary embolism when systemic thrombolysis is contraindicated (CR), 32
- Use of a Swan-Ganz catheter and streptokinase in the management of massive hemoptysis (CR), 33, (LE), 270

Substance abuse
- Multiple addictions: Co-synchronous use of alcohol and drugs, 596
- Substance abuse during pregnancy (LE), 336

Suffolk County
- Mortality in the Hispanic population of Suffolk County, New York, 442

Suicide
- Risk factors for teenage suicide (NF), 46

Superior vena cava syndrome
- Superior vena cava syndrome and right pleural effusion due to giant goiter (CR), 467
- Superior vena cava syndrome associated with a Hickman catheter (CR), 208

Surgery
- Too many surgeons for too few operations? (NF), 47

Sutures
- For every suture, a silver lining (NF), 226

Syphilis
- Rising incidence of congenital syphilis: Back to the future (ED), 485
- Screening for congenital syphilis begins (LEN), 166
- Syphilis among parturients at an inner city hospital: Association with cocaine use and implications for congenital syphilis rates, 488

Tamoxifen
- Endometrial carcinoma in five patients with breast cancer on tamoxifen therapy (CR), 207

Temporal arteritis
- Intestinal perforation/fistulization complicating temporal arteritis (LE), 270

Thoracic lymphadenopathy
- Thoracic lymphadenopathy and disseminated *Pneumocystis carinii* in AIDS (CPC), 146

Thromboembolic disease
- Local low-dose infusion of streptokinase for massive pulmonary embolism when systemic thrombolysis is contraindicated (CR), 32
- Low-dose subcutaneous heparin therapy (LE), 39
- Topical low-dose thrombolytic therapy (ED), 2

Thyroid disease
- Prevalence of subclinical hypothyroidism in adults with low-normal blood thyroxine levels, 541

Tobacco
(see also Smoking)
- Magazines that do not accept tobacco advertising, 414
- Public health benefit of increasing tobacco taxes in New York State, 174

Toxic shock syndrome
- Incidence of toxic shock syndrome drops dramatically over ten-year period (NF), 480

Trabecular carcinoma
- Trabecular (neuroendocrine) carcinoma of the skin: Report of four cases and review of the literature (CR), 35

Transplantation
- Organ donation (ED), 394
- Pattern of organ donation in a large urban center, 396

Trauma
- Small bowel injuries following blunt abdominal trauma: Early recognition and management, 446

Trauma services
- Aeromedical emergency trauma services and mortality reduction in rural areas, 358

Tropical medicine
History of the New York Society of Tropical Medicine (HIST), 450
L-tryptophan
Eosinophilia-myalgia syndrome and L-tryptophan (CR), 512
Eosinophilia myalgia syndrome (LEN), 380
Tuberculosis
Cervical tuberculosis (LE), 270
Drug resistant tuberculosis among the homeless in New York City, 351
History of the Ray Brook State Tuberculosis Hospital (HIST), 406
Mycobacterium tuberculosis: A disease of growing importance in urban population groups (ED), 585
Pott's disease in urban populations: A report of five cases and a review of the literature, 588
Ultrasonic humidifiers
Hypersensitivity pneumonitis associated with the use of ultrasonic humidifiers (CR), 263
UNY•Care
UNY•Care (LE), 377
Vaccination
(see Immunization)
Vibrio vulnificus
Vibrio vulnificus in raw oysters (LEN), 87
Video games
Nintendo-induced epilepsy (NF), 387
Violence
Prevalence and characteristics of violent patients in a general hospital, 591
Visual hallucinations
Visual hallucinations as grief reaction without the Charles Bonnet syndrome (LE), 216
Vitamin supplements
Use of multivitamin/folic acid supplements during first weeks of pregnancy can help prevent neural tube defects (NF), 225
Warthin's tumor
Extraparotid Warthin's tumor: An unexpected finding in three female patients (CR), 465

Westchester County
History of the formation of the Committee on Women of the Westchester County Medical Society, 324

Williams, Jane V., MD
Jane V. Williams, MD (1924–1989): An appreciation, 327

Women physicians
Combining parenthood and practice (LE), 616
History of the formation of the Committee on Women of the Westchester County Medical Society, 324
Interview with Nancy E. Gary, MD, a medical school dean, 321
Introduction to a current bibliography on women in medicine (LE), 335
Mainstreaming the minority and assumptions of rightful place (ED), 283
Parenthood and practice, 326
Perspectives on women physicians in New York State: Past, present, and future, 317
Responses to "Parenthood and practice" (LE), 334
Senior women physicians: The question of retirement, 292
Women, families, and academic medicine (ED), 284
Women in medicine since Flexner, 302

Women's health
Adenocarcinoma of the cervix (ED), 535
AIDS is affecting women at increased rates (NF), 480
Antibiotics and other drugs may inhibit effectiveness of oral contraceptives (NF), 341
Attempt to predict bone mass in perimenopausal women using only clinical data (NF), 342
Breast cancer detection and counseling program (LEN), 85
Breast cancer screening: Changing physician practices and specialty variation, 288
Cervical cancer in New York State (LEN), 521
Granulomatous mastitis (ED), 287
Incidence of toxic shock syndrome drops dramatically over ten-year period (NF), 480
Is lightscanning a viable alternative to mammography for detecting breast cancer? (NF), 341
Mammography attitudes and awareness (LEN), 339
Multifocal human papillomavirus infections of the genital tract in HIV seropositive women (LE), 162
Premenstrual syndrome (LE), 615
Premenstrual syndrome and diet (NF), 341
Primary female syndromes: An update, 295
Primary signet ring cell carcinoma of the uterine cervix (CR), 559
Screening for breast cancer: Are physicians doing enough? (ED), 285
Substance abuse during pregnancy (LE), 336
Time trends in breast cancer incidence (LEN), 473
Unexpected, unattended deliveries (CR), 330
World Health Organization
WHO embarks on vaccination campaign (NF), 581
Zidovudine
Food and Drug Administration halves dosage for AZT (NF), 168

AUTHOR INDEX

Abney, Ella, 335
Akerman, Michael, 467
Allam, Azmy, 234
Alper, Philip R., 524
Alunday, Arthur, 73
Anaissie, Elias J., 586
Anandarao, Nanarao, 371
Andelora, Gary P., 424
Andersen, Kathleen S., 23
Ankobiah, William A., 234
Azer, George S., 562

Bailie, Michael D., 185
Balaji, Malur R., 77
Barranco, Elizabeth A., 163
Barzel, Uriel S., 541
Batchelor, Allison J., 292
Battan, Ruggero, 351
Bauer, Stanley, 336
Beck, Frederick, 406
Belkin, Beth M., 596
Bennett, Allen J., 552
Bennett, Nancy M., 284
Berger, Mitchel, 601
Bernstein, Anne Elayne, 616
Bhat, Kiran, 467
Bhat, Subrahmanya, 467
Bianco, Emidio A., 224
Bigas, Miguel A. Rodriguez , 465
Bloomfield, Randall D., 379, 469, 517
Bluestone, Harvey, 591
Bluestone, Naomi R., 283, 327, 525
Blum, Steve, 243
Bokkenheuser, Victor D., 4
Bouknight, LaClaire Green, 493
Brandstetter, Robert D., 33, 270
Brecher, David B., 429
Brickner, Philip W., 476
Broaddus, Raymond K., 133
Brotman, Andrew W., 523
Brownlee, William, 74
Bryski, Alan, 476
Bunkin, Irving A., 253, 517
Burg, Mary Ann, 288
Burke, Lillian, 420
Burt, Richard K., 277
Butt, Khalid M., 396

Candaras, Michael M., 513
Carrasco, Manuel, 234
Carroll, Patricia B., 210
Cassell, Eric J., 552
Castillo, Nieva B., 465
Caubel, Patrick, 162
Chabon, Ada B., 207
Chang, Hwa-gan H., 126
Chaudhry, M. Rashid, 272
Chechani, Vijay, 234
Chen, Eachou, 349
Chintalapally, Shobha, 488
Chitkara, Rajinder K., 162
Chu, Florence C., 429
Chumas, John C., 559
Cioroiu, Michael, 561
Citron, Marc, 216
Cocores, James A., 491
Cohen, Jon R., 159
Cohen, Paul, 467
Cole, Solon R., 263
Coleman, Edward J., 446
Coli, Lorraine, 415
Conetta, Rick, 162
Cummings, K. Michael, 54, 174
Curran, Anita S., 129

David, William J., 518
Davis, J. Mostyn, 477
Decker, Michael D., 346
Deckoff, Stephen L., 176
Degnan, Edward, 206
DeGraff, Arthur C., Jr., 263
DeHovitz, Jack A., 119, 276, 622
Delaney, Vera, 396
Delke, Isaac, 90, 488
Deren, Sherry, 179
Deshpande, Vilas, 611
Dietz, Patrick A., 446
Diferdinando, George T., Jr., 126
Dmytrenko, George M., 221
Doscher, William, 176
Douglas, R. Gordon, Jr., 157, 266
Drucker, Mitchell, 207
Dytoc, Joseph N. T., 69

Eden, Alvin N., 478
Einhorn, Arnold, 612
El-Ani, Arif S., 609
Ellinas, Panayiotis A., 588
Estefan, Mokhtar Y., 272
Evans, James T., 615

Faisal, Mohammed A., 4
Falvo, Cathey E., 349
Farnsworth, Peter B., 531, 552
Faustin, Daniel, 272
Feldman, Joseph, 488
Fernandez, Henry A., 545
Fine, Edward J., 513
Fiscella, Kevin A., 623
Fisher, Laura L., 266
Fitzgibbons, Patrick L., 287
Flam, Robin, 129
Flatto, David, 129
Fleischer, Renee A., 335
Florentin, Rudolfo, 206
Flynn, John T., 393
Foulques, Henri, 162
Fox, Daniel M., 23
Foy, Donald F., 377
Frank, Blanche, 179
Freilich, Daniel A., 358
Friedman, Matthew S., 176
Friedman, Steven G., 176

Gaines, Benjamin, 336
Galloway, Jacqueline, 366
Garcia, Roger L., 155
Gary, Nancy E., 377
Gelfand, Maxwell L., 373
Gemson, Donald H., 285
Gerson, Charles D., 523
Gerstenblitt, Dan, 469
Geyer, J. Russell, 601
Gold, Mark S., 491, 596
Golden, Richard L., 512
Goldstein, Marvin, 76
Goldstein, Robert D., 375
Gordon, Mark Forrest, 469
Gordon, Michael J. V., 375
Grabau, John C., 133, 238
Groff, Donald N., 163
Gross, Lillian, 321
Gumbs, Milton A., 331
Gump, Frank E., 356, 617
Gutierrez, Louis, 272

Haire-Joshu, Debra, 578
Hajdu, Ivan, 415
Haller, John S., Jr., 499
Halpern, Abraham L., 552
Han, Yangsook, 133, 238
Hanrahan, John P., 133
Hanson, Richard M., 384
Haraldson, Sixten S. R., 61
Harbert, Kenneth R., 579, 623
Harper, Rita G., 330
Harris, Randall E., 123
Harvey, N. A., 215
Havlik, Richard J., 440
Hazinski, Thomas, 88, 89
Hebert, James R., 123
Hein, Karen, 143
Hena, Muhammad A., 270
Henry, Daniel A., 347
Henry, John B., 552
Hertz, Alan B., 223, 576
Hirschman, Richard J., 207
Hoffman, Nancy Yanes, 617
Hoffman, Robert S., 39
Holden, David M., 537
Holt, Peter R., 4
Holtzman, Rosanne, 76
Hong, Joon H., 396
Hopkins, Michael P., 535
Hyland, Millard J., 349

Imperato, Pascal James, 1, 51, 115, 196, 215, 221, 345, 377, 430, 450, 517, 566, 625
Ismail, Mohammed, 74

Jacob, Jessica R., 334, 616
Jacobs, Bernard, 8
Jaffe, Bernard M., 214
Jorgensen, Virginia T., 566

Kabakow, Bernard, 207
Kahng, Kim U., 214
Kain-Hyde, Sue, 133
Kaiser, Valerie, 334, 616
Kamholz, Stephan L., 234, 467
Kanick, Virginia, 552
Kark, Pieter R., 552
Karp, Nolan, 416
Katlama, Christine, 162
Katz, Bernard J., 271
Kaufman, Gerald I., 238
Kaufman, Lee D., 463
Kaufman, Stephen, 214
Kaukonen, Larisa, 39
Kerner, Bruce A., 176
Kerr, Harry Davidson, 208
Khatib, Reza, 562
Klahr, Aryeh L., 491
Kolisch, Paul deR, 566
Kotler, Donald P., 4
Krajewski, Kenneth J., 487
Krishnasastry, K. V., 176
Krongrad, Ehud, 385
Kwan, Tak, 612

Lackner, Rudy, 159
Lamb, David R., 222
Lamparello, Patrick L., 416
Landolt, Allison B., 552
Lane, Dorothy S., 288
Langrod, John, 123
LaRaja, Raymond D., 561
Lawrence, Charles E., 133
Lee, Hyung Kon, 591
Lee, Ira, 74
Lehman, L. B., 88
Li, John K., 35
Liese, Bruce S., 17, 422
Lindenbaum, Connie R., 384
Lister, Eric D., 576
Lobl, Henry, 386
Lockridge, Robert, 336
Loeb, Laurence, 552
Loewen, Gregory, 415
Logan-Young, Wende W., 617
Lowenfels, Albert B., 349
Lowinson, Joyce, 123

Maguire, Barbara H., 133
Makin, Myer, 201
Malabonga, Vic M., 613
Malfetano, John H., 206
Manjoney, Deborah L., 467
Mann, William J., 418, 559
Margolin, N. Lionel, 477
Margolis, Irving B., 77
Marino, Catherine, 73, 268
Markowitz, Martin, 271
Marshall, James, 54
Marx, Gertie F., 517
Mascola, Laurene, 485
Mathew, Anna, 207
McClure, Lucretia, 76
McDermott, Janette, 356, 617
McDonald, Elizabeth, 73, 268
McHenry, Lee, 210
Mensah, George A., 157
Merkel, Frederick K., 394
Metcalf, Tom, 231
Mikl, Jaromir, 133
Miller, Michael Joseph, 541
Miller, Norman S., 491, 596
Mincer, Flora K., 35
Minkoff, Howard, 488
Mitra, Raghu N., 525
Moline, Margaret L., 615
Moll, Ute M., 559
Monahan, Thomas J., 545
Monroe, James, 406
Morse, Dale L., 56, 126, 133
Moy, Donald R., 378
Munoz, Eric, 216
Murphy, Dennis, 126

Murphy, Robert X., Jr., 35

Nacht, Robert, 612
Nakao, Michael A., 32, 270
Nanda, Deepak, 488
Narasimhan, Parthas, 518
Narasimhan, Sudha, 518
Nickerson, Katherine G., 284
Novick, Toni G., 324
Numann, Patricia J., 552

O'Connor, John F., 420
O'Dell, Michael L., 17, 422
Ona, Fernando V., 69, 552

Pablos-Mendez, Ariel, 351
Pace, Benjamin W., 77
Pan, Chao, 541
Paradowski, Linda J., 415
Parente, John T., 336
Parker, Frederick, 74
Pasternack, Jonathan B., 377
Patel, Rameshbhai P., 611
Patel, Shailesh R., 371
Patsner, Bruce, 559
Patton-Greenidge, Loretta, 224
Pellegrino, Robin M., 424
Perkel, Robert L., 472
Pillari, George, 159
Pilon, Vernon A., 121
Pincus, Susan H., 139
Polednak, Anthony P., 442
Polos, Peter G., 511
Powell, Steven, 234
Pryce, Edith, 561

Quiroga, Virginia A. Metaxas, 256

Rafferty, Margaret, 223
Rajaratnam, Shanthini, 429
Ramos-Zuniga, Rodrigo, 351
Rampolla, Michael, 116
Rango, Nicholas A., 116
Ranson, John H. C., 416
Rao, A. Naveen, 511
Ravalli, Stefano, 155
Raviglione, Mario C., 351
Razak, Mohamed S., 465
Reddy, Gaddam, 626
Reddy, Ramachandra C., 562
Reeves, Paul E., 76
Reiner, Steven C., 39
Rinaldi, Michael G., 586
Risemberg, Herman M., 552
Ritterband, Arnold B., 565
Rogatz, Peter, 76, 517, 552, 615
Rosenberry, Edward H., 422
Rosenthal, Thomas C., 537
Rosner, Fred, 214, 524, 552, 588
Ross, Harvey M., 279
Rossi, Ricardo L., 53
Rubin, Lewis J., 2
Ruder, Avima M., 129

Sacks, Robert, 577
Sako, Kumao, 465
Salem, Raja, 562
Salerno, Monica J., 420
Santilli, Veronica C., 317
Santogade, Peter J., 4
Saunders, Marguerite T., 41
Scanlon, Adelaide T., 326, 616
Scherzer, Herbert H., 263
Schlessel, Jerrold S., 330
Schmeidler, James, 179
Schmidt, Eugene M., 238
Schneck, Jerome M., 216, 378, 422, 470
Schneiderman, Henry, 270
Schoenbaum, Ellie E., 139
Scholes, John V., 4
Sciandra, Russell, 54, 174
Scully, Robert E., 172
Seaton, Elizabeth, 330
Sechzer, Philip H., 552
Sedlis, Melissa M., 215
Severino, Sally K., 615
Shah, Ajay K., 331
Shannon, Michael, 89
Sharnoff, J. George, 39
Sheehy, John P., 616
Shein, Ruvan, 155
Sheka, Kedambady P., 611
Shiue, Shyi-Tang, 263
Shultz, James M., 56
Sicular, Arthur, 214
Siegel, Jane, 77
Silver, Archie A., 576
Singh, Inderjit, 611
Skretny, Michelle T., 54
Slocum, Peter, 39
Slonim, Roberta, 624
Smego, Raymond A., Jr., 163
Smith, Perry F., 56
Smith, Peter R., 613
Smucker, Douglas R., 533
Solomon, Barry A., 154
Solomon, Lydia, 331
Song, Jing, 467
Sordillo, Peter P., 552
Soria, Emilio D., 513
Spiegel, Allen D., 79, 358, 428, 568
Spinazzola, Regina, 330
Stankus, Richard P., 232
Starr, Michael B., 12
Steele, Kevin D., 222, 278
Stein, Kenneth M., 157
Steinbach, Barbara G., 368
Steiner, R. Prasaad, 580
Stern, Marc F., 368
Stillman, Richard M., 276
Stolfi, Julius E., 277, 278, 386, 479, 626
Strauch, Berish, 35
Sumrani, Nabil, 396
Svoboda, Jerry J., 77
Sweeney, Kenneth, 491

Tarlau, Milton, 422
Thomas, Claudia, 477, 622
Thorbjarnarson, Bjorn, 385
Thorgeirsson, Snorri S., 277
Thorpe, Kenneth E., 247
Travin, Sheldon, 591
Trevino, Fernando M., 439
Troncone, Michael T., 400
Truman, Benedict I., 133

Ulbright, Thomas M., 171
Usai, Amedeo, 561
Ushkow, Martin C., 76

Vaidya, Kamini, 234
Verbalis, Joseph G., 210
Vincent, Raf A., 155

Wadhera, Premlata, 518
Walsh, Mary Roth, 302
Walther, Frederick A., 511
Wang, Robert C., 276
Washecka, Robert M., 418
Webber, Mayris, 139
Weinberg, Jeffrey, 429
Weir, Matthew R., 585
Wenig, Peter, 159
Whitehead, Fred A., 17, 422
Wilion, Felicia, 185
Wilkes, Michael S., 243
Winter, Simon T., 201
Wiznitzer, Max, 430
Wolpaw, Jonathan R., 552
Woodward, Willow, 537
Wright, Kenneth W., 406
Wrisley, David, 74
Wröblewski, Felix, 76
Wynder, Ernst L., 123

Yankauskas, Ellen, 295, 518
Young, Richard A., 625
Young, Robert H., 172

Zang, Edith, 123
Zelnick, Ronald, 216
Zorgniotti, Adrian W., 420, 565
Zuna, Rosemarie, 418

ACQUIRED IMMUNODEFICIENCY SYNDROME

Current Issues and Scientific Studies

Edited by Pascal James Imperato, MD

With a foreword by
George J. Lawrence, Jr., MD, Chairman
AIDS Task Force
Medical Society of the State of New York

The epidemic of acquired immunodeficiency syndrome has presented Americans with a number of social, medical, political, and economic issues that are now the focus of much public discussion and, in some instances, heated debate. The chapters in this volume reflect a range of social and philosophical challenges posed by the epidemic and provide some stimulating insights from authors who have given these issues serious thought. Topics covered include the epidemiology of AIDS in the northeastern United States, an overview of the problem of AIDS in Africa, health education intervention strategies, the impact of the epidemic on the health-care system, issues related to HIV antibody testing, and common and unusual clinical complications encountered in the disease. This book consists of important papers on AIDS published in the *New York State Journal of Medicine* in 1987 and 1988.

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Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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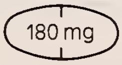

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References: 1. 1988 Joint National Committee: The 1988 report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. *Arch Intern Med* 1988;148:1023-1038. **2.** *Physicians' Desk Reference®*, ed 44. Oradell, NJ, Medical Economics Company Inc, 1990.

BRIEF SUMMARY

Contraindications: Severe LV dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block (if no pacemaker is present), atrial flutter/fibrillation with an accessory bypass tract (eg, WPW or LGL syndromes), hypersensitivity to verapamil.
Warnings: Verapamil should be avoided in patients with severe LV dysfunction (eg, ejection fraction < 30%) or moderate to severe symptoms of cardiac failure and in patients with any degree of ventricular dysfunction if they are receiving a beta-blocker. Control milder heart failure with optimum digitalization and/or diuretics before Calan SR is used. Verapamil may occasionally produce hypotension. Elevations of liver enzymes have been reported. Several cases have been demonstrated to be produced by verapamil. Periodic monitoring of liver function in patients on verapamil is prudent. Some patients with paroxysmal and/or chronic atrial flutter/fibrillation and an accessory AV pathway (eg, WPW or LGL syndromes) have developed an increased antegrade conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving I.V. verapamil (or digitalis). Because of this risk, oral verapamil is contraindicated in such patients. AV block may occur (2nd- and 3rd-degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with verapamil.
Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol clearance may occur with combined use. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digitoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration. Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels or increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.
Adverse Reactions: Constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF, pulmonary edema (1.8%), fatigue (1.7%), dyspnea (1.4%), bradycardia: HR < 50/min (1.4%), AV block: total 1°,2°,3° (1.2%), 2° and 3° (0.8%), rash (1.2%), flushing (0.6%), elevated liver enzymes. The following reactions, reported in 1.0% or less of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitations, purpura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis or bruising, cerebrovascular accident, confusion, equilibrium disorders, insomnia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, arthralgia and rash, exanthema, hair loss, hyperkeratosis, macules, sweating, urticaria, Stevens-Johnson syndrome, erythema multiforme, blurred vision, gynecomastia, increased urination, spotty menstruation, impotence.

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Constipation, which is easily managed in most patients, is the most commonly reported side effect of Calan SR.

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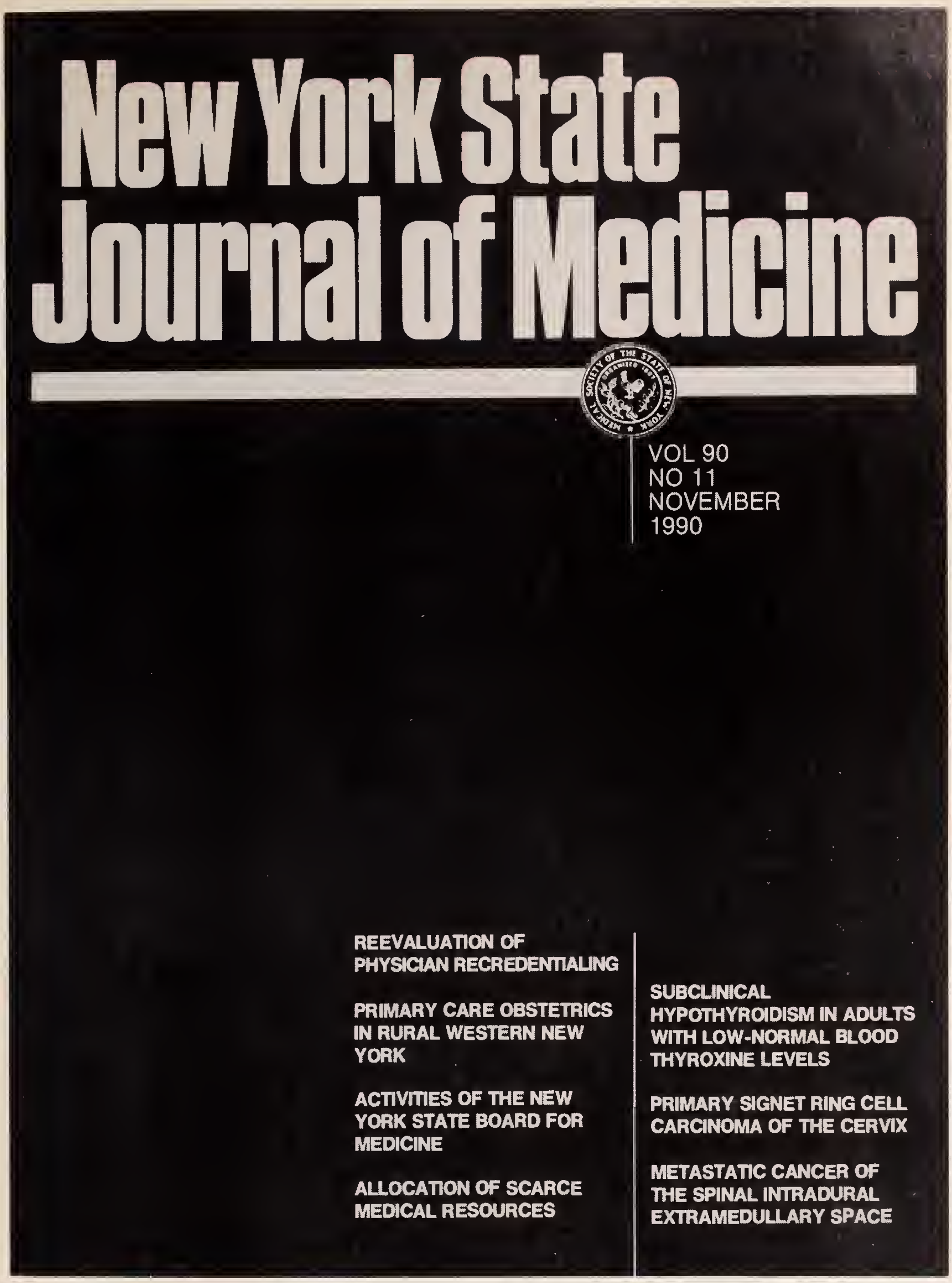

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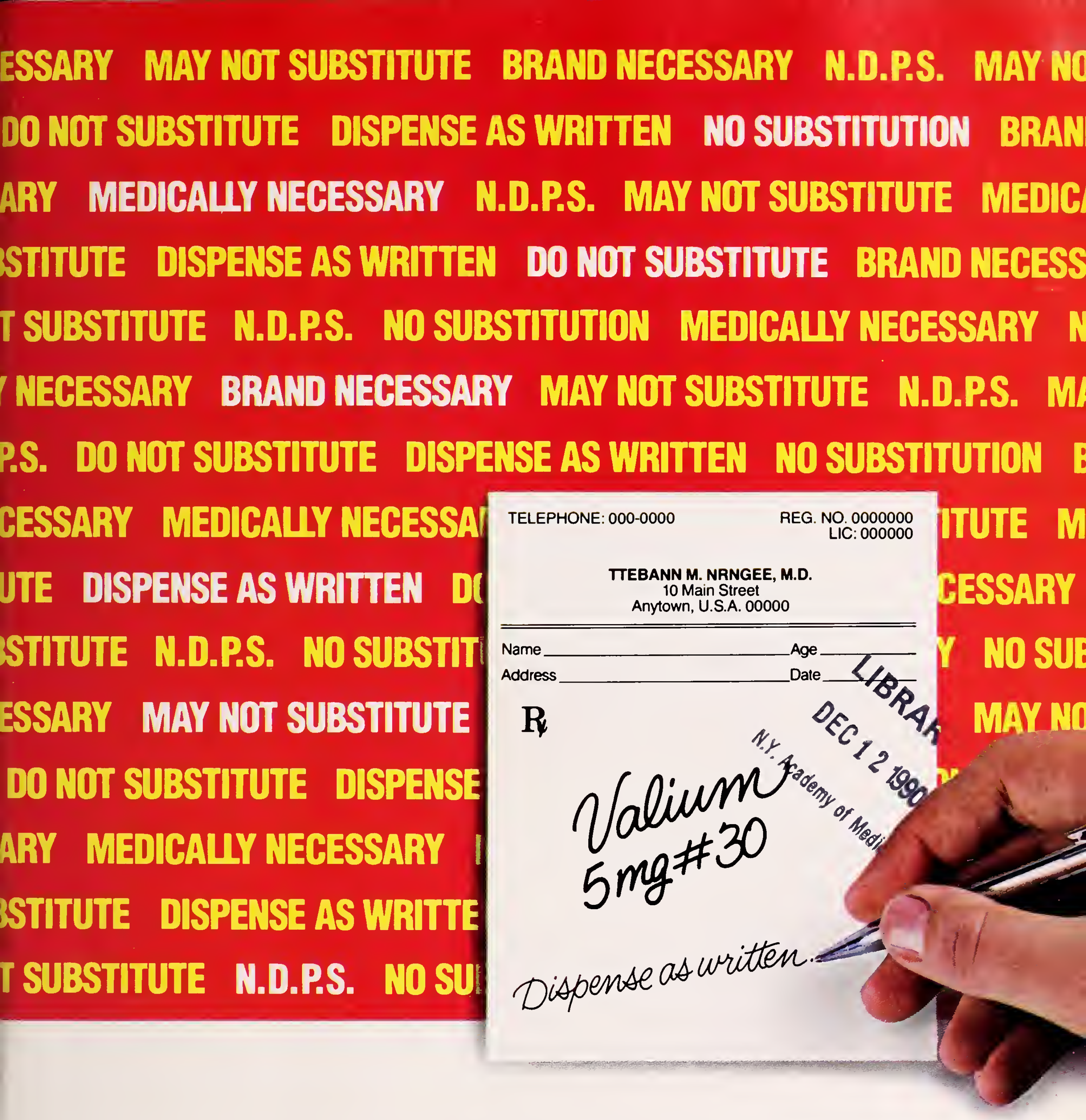
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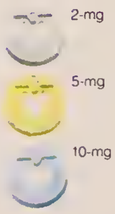

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